



Pain management and Anaesthesiologists

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Pain is generally divided into acute pain, primarily due to nociception, the neuronal response only to traumatic or noxious stimuli and chronic pain due to nociception, psychological and behavioral factors. Pain can also be classified according to pathophysiology (e.g. nociceptive or neuropathic pain), etiology (e.g. postoperative pain or cancer pain), or the affected area (e.g. headache or low back pain). Such classifications are useful in the selection of treatment modalities and drug therapy. We also need to know a thorough knowledge of anatomy and physiology of pain (nociception), pharmacodynamics and pharmacokinetics of analgesics.

The term "pain management" is usually applied to the entire discipline of anaesthesiology, but its modern usage is restricted to management of pain outside the operating room. However, there are patients who require short- and long-term pain management both in and out of the hospital. The goal of pain management is to treat all kinds of pain and to minimize pain rather than eliminate it.

We all knew that there is acute shortage of qualified anaesthesiologists in and outside India. So also, the number of anaesthesiologists choosing to practise pain therapy is very limited. The practice of pain management is not just limited to anaesthesiologists but includes other practitioners such as physicians (internists, general surgeons, neurologists, oncologists, psychiatrists and radiotherapists) and non-physicians (psychologists, chiropractors, acupuncturists and hypnotists). Currently, there

are no established standards for the types of disciplines that must be included, and this is another reason why treatment offerings will vary from clinic to clinic. To qualify as a pain management specialist in the eyes of the American Board of Medical Specialties, a health care provider should be an MD with board certification in at least one of the following specialties: Anaesthesiology, Physical rehabilitation, Psychiatry and neurology.

The most effective approach is multidisciplinary, in which the patient is evaluated by one physician who conducts the initial examination and formulates and treatment plan, and the services and resources of other specialists are readily available and also evidence-based approach¹. Some widely used techniques of pain management are :

I. Non-invasive non-drug pain management e.g. exercise includes hydrotherapy, flexion exercises and aerobic routines ; manual techniques like manipulation of affected areas by means of chiropractic adjustments, osteopathy and massage therapy ; behavioral modification such as cognitive - behavioral approaches, hypnosis, relaxation, biofeedback and imagery cutaneous stimulation; acupuncture ; electrotherapy like transcutaneous electrical nerve stimulation (TENS).

II. Non-invasive pharmacologic pain management e.g., analgesics include acetaminophen, Nonsteroidal anti-inflammatory agents (NSAIDs) include

aspirin, ibuprofen, naproxen, and the new COX-2 inhibitors ; muscle relaxants to treat muscle spasms due to pain and protective mechanisms; opioids medications (oral/transdermal/parental) for acute or post-operative pain (most appropriate); antidepressants, neuroleptics and anti-convulsants, systemic local anaesthetics and alpha1-adrenergic agonists to treat neuropathic ("nerve") pain. Corticosteroids are also used for their anti-inflammatory and possibly analgesic actions. Botulinum, toxin injection are helpful for the treatment of painful conditions associated with skeletal muscle.

III. Invasive pain management e.g. Injections of steroids or anesthetic or opioids to nerve, joint, epidural or intrathecal space ; prolotherapy-injection of solution to stimulate blood circulation and ligament repair at affected site; surgically implanted electrotherapy devices like implantable spinal cord stimulators (SCS) and implantable peripheral nerve stimulators ; implantable opioid infusion pumps-surgically implanted pumps that deliver opioid agents directly to affected nerve and radiofrequency radioablation-deadening of painful nerve via heat produced by a specialized device.

The anaesthesiologists can act in time his technical expertise and skill in the pain management. His/her technical knowledge in regional analgesia is also another added advantage in the pain therapy. The administration of local anaesthetics-opioid

mixtures neuraxially (particularly epidural) is an excellent technique for managing postoperative pain following abdominal, pelvic, thoracic, or orthopedics procedures on the lower extremities. In spite of introduction of newer opioids analgesics, serious side-effects of epidural or intradural opioids like delayed respiratory depression is still possible and dose-dependent.

Anaesthesiologists trained in pain management are in a unique position to coordinate multidisciplinary pain management centers because of broad training in dealing with a wide diversity of patients from surgical, obstetric, pediatric, and medical subspecialties, as well as expertise in clinical pharmacology and applied neuroanatomy including the use of peripheral and central nerve blocks. Today, anaesthesiologists has become a competent pain therapist in such centers. In one study, 80% of the acute pain management programme was headed by anaesthesiologists and 94% as a member of the acute pain management team.² Pain clinics is also effective both for nerve block treatments³ and for psychological based therapies^{4,5}.

Lastly, I am convinced that no other specialty provides anaesthesiologists's clinical experienced in pain alleviation so much though it is multidisciplinary approach. So, anaesthesiologists must act now and begin to work in pain management including pain clinics globally.

References

1. National health and Medical Research council. Acute pain management: scientific evidence. Canberra, Australia: National health and Medical Research council;1995.
2. Warfield CA, Kahn CH. Acute pain management prognosis in US hospitals and experiences and attitudes among US adults. *Anesthesiology* 1995; 83:1090-4.
3. Davies HTO, Crombie IK, Brown JH, Martin C. Diminishing returns or appropriate treatment strategy? - an analysis of short - term outcomes after pain clinic treatment. *Pain* 1997;70: 203-8.
4. Flor H, Fydrich T, Turk DC. Efficacy of multidisciplinary pain treatment centres: a meta-analytic review. *Pain* 1992;49:221-30.
5. Kingery WS. A critical review of controlled clinical trials of peripheral neuropathic pain and complex regional pain syndromes. *Pain* 1997; 73:123-39.



Knowledge and attitude on obesity among high school students

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Abstract

Objective: To assess the knowledge and attitude of obesity among high school students and to find out any difference in knowledge on obesity between the sexes. **Methods:** A school based cross sectional study was conducted among a sample of 376 students. Two urban and one rural private school were selected purposively for the study. Data were collected using a predesigned, semi-structured questionnaire and it was presented in descriptive statistics. For analysis purpose, chi-square test was used. **Results:** Out of 376 students, 200 and 176 were males and females respectively. 36.7% of them had heard about the term obesity. Among those who had heard of obesity, around 42% did not consider it as a disease. 61.6% could list a few factors causing obesity while 44% had knowledge about complications of obesity. For 75% of them, books were the main sources of information. Their views about obese people were mostly on the positive side. No significant differences regarding knowledge about obesity were noted between sexes. **Conclusion:** Majority of the participants had poor knowledge on obesity. Educating the students on obesity, preferably through school curriculum could be considered.

Key words: Obesity, knowledge, attitude, high school students.

Introduction

Obesity is becoming a public health problem. The increasing prevalence of overweight and obesity in adults and children demonstrate a steadily growing epidemic both in developed and developing countries. This rising rate of obesity is associated with obesity related co-morbidities including cardiovascular disease, hypertension, some cancers, joint diseases, type 2 diabetes etc.¹ It has been estimated that an average of 20-40% adults and 10-20% of children and adolescents are affected with obesity.²

Obese individuals suffer from social stigmatization, discrimination and low-self esteem. Longevity is also reduced in obesity. The health consequences of obesity can be avoided if preventive measures are taken up early. There is no concrete data on the knowledge of obesity among the adolescents in Manipur. So, the present study was undertaken with the objective of assessing the knowledge and attitude of obesity among high school students and to find out any difference in knowledge on obesity between the sexes.

Material and methods

This school based cross-sectional study was undertaken during September to October 2005. One school from rural and two schools from urban areas were selected purposively for the present study. A sample of 376 students studying in class VIII, IX and X standards was selected.

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A prior appointment was taken from the principals of the respective schools and data were collected on a pretested, semi-structured, self-administered questionnaire put to the respondents in the class and students were given forty-five minutes time to fill in the questionnaire. Extra time was also given to the students who could not finish within the stipulated time to make the questionnaire completed. The questionnaire consisted of socio-demographic information, knowledge about different aspects of obesity, sources of information and their attitude towards obese persons. Those students who were not willing to participate and absent on the day of visit were excluded from the study. Statistical analysis was carried out using descriptive statistics and Chi-square test. A probability value of <0.05 was considered significant. Prior approval of the institute's research and ethical committee was obtained.

Results

Three hundred and seventy six students could be enrolled in the present study. Out of these students 138 (36.7%) responded that they have heard of the term obesity and their main source of information were books (75%). The mean age was 14.6 years with a range of 12 to 18 years. Boys (53.2%) outnumbered girls,

Table 1. Background characteristics of the study subjects

Characteristics	Number	Percentage
Sex		
Boys	200	53.2
Girls	176	46.8
Class		
VIII	116	30.9
IX	144	38.2
X	116	30.9
Religion		
Hindu	265	70.5
Christian	090	23.9
Muslim	021	05.6
Dietary habits		
Vegetarian	180	47.9
Non-veg	196	52.1
Parents' Education		
Father		
Illiterate	005	01.3
= Matric	113	30.4
>Matric	253	68.3
Mother		
Illiterate	017	04.5
= Matric	158	42.0
>Matric	201	53.5
Students' built		
Normal	288	76.7
Thin	053	14.2
Fat	035	09.1

and majority belonged to Hindu community (70.5%). The majority of the parents were literate. Nearly 76.7% of the students felt that they have normal built while 9.1% considered themselves fat (table 1). Those respondents who have heard of the term obesity were asked further questions pertaining to knowledge and attitude on obesity.

Students' knowledge about obesity is shown in table 2. Eighty-five students (61.6%) claimed that they knew some factors causing obesity. Overeating (52.5%) was the commonest factor listed by them followed by lack of physical exercise/activity (20.9%). Less than half of them (44.9%) responded that they knew some complications of obesity. However only two third of the students could list hypertension and heart diseases as one of the complications. 57.8% of the students considered obesity as a disease. The majority (79%) of the respondents did not want themselves being obese. However, 115 of the students (83.4%) responded that even if their friends were obese it would not affect their relationship and 75.3% of them held a positive

Table 2. Knowledge of the students on obesity

Questions on	Number	Percentage
Do you know some factors causing obesity?		
Yes	85	61.6
No	53	38.4
If yes, list them*		
Overeating	83	52.5
Lack of physical activity	33	20.9
Since birth	13	08.2
Bad company	10	06.3
Others	19	12.1
Do you know some diseases caused by obesity?		
Yes	62	44.9
No	76	55.1
If yes, list them*		
Hypertension	31	37.8
Heart diseases	21	25.6
Diabetes mellitus	12	14.7
Cancer	01	01.2
Others (Malaria, TB, Gastritis)	17	20.7
Do you consider obesity as a disease?		
Yes	78	57.8
No	60	42.2
Can you name some means for preventing obesity?*		
Taking regular exercise or Physical activity.	67	69
Reduce fatty food or over eating	06	10
Increase intake of green leafy vegetables	44.1	45.4
Clean environment	03.9	06.6

*numbers calculated from the total number of responses

attitude regarding obese people. Knowledge about obesity between the sexes was not statistically significant (table 3).

Table 3. Knowledge on obesity by sex

Some aspects of knowledge on obesity	Sex		X ²	p-value
	Male(%)	Female(%)		
Heard of obesity	68(49.3)	70(50.7)	0.11	0.29
Knew some factors causing obesity	39(49.4)	46(50.6)	1.99	0.16
Consider it as a disease	43(55.1)	35(44.9)	0.07	0.79
Knew some complications	35(56.5)	27(43.5)	0.18	0.67

Discussion

The worldwide increase in the prevalence of obesity and major health problems associated with it has drawn an interest in research on it. Modest weight loss (5% to 10% of the total body weight) through lifestyle intervention approaches has been found to have a beneficial effect on co-morbid conditions. Research studies have found that interventions that combine a low-calorie diet, increased physical activity and behavior therapy are most effective for weight loss and maintenance.¹ Some sedentary behavior like watching television, playing videos and computer games continuously for more than 3 hours were significantly associated with obesity.³ In this study, majority of the students 238 (63.2%) had not heard of the term obesity. Many who had heard of it could not list out factors and diseases associated with it. A few of them wrongly felt that bad companies were one of the factors causing obesity. As obesity is not a part of the school curriculum, students are expected to know less about it. Studies in this aspect of obesity among school students are limited. However in a study, half of the dental hygiene students reported that there was no obesity education prior to professional education.⁴ In one study, a substantial number

of patients attending a hospital at Karachi understood the meaning of obesity and considered it as a major health problem.⁵ More than half of the students in our study were

not aware about the complications of obesity. Nyaruhucha and colleagues⁶ in their study revealed similar findings. We found that seventy-eight (57.8%)

students considered obesity as a disease. In a study by Bocquire and colleagues⁷, 90.2% of the general practitioners regarded obesity as a disease requiring long-term management. Attitude towards obese persons was favorable from most of the students. Some researchers also reported similar findings.⁴ However, in another study 30% of the respondents had a negative attitude towards overweight and obese persons.⁷ In our study, no significant difference was found regarding the knowledge of different aspects of obesity between boys and girls students indicating that they have the same knowledge exposure. A further study with a representative sample should be considered to obtain more detail information.

Conclusion

Results of the current study indicate majority of the adolescent high school students had a poor knowledge about obesity. Nevertheless, they had a positive attitude towards obese people. Their main sources of information were books and doctors. There is a need to sensitize the adolescents to obesity as this population group is just right for introducing primary prevention interventions.

References

1. Lak Hampal KR. Treatment and prevention of obesity. *Indian Journal of Nutrition and Dietetics*. 1978; 15: 18-92.
2. Park K. Obesity. In : Park's text book of preventive and social medicine, 17th edition Jabalpur : M/s Banarsidas Bhanot; 2002 .p. 293-300.
3. Kantachuvessiri A, Sirivichayakul C, Kaewkungwal J, Tungtrongchitr R, Lotrakul M. Factors associated with obesity among workers in a metropolitan waterworks authority. *Southeast Asian J Trop Med Public Health*. 2005; 36(4): 1057-65.
4. Magliocca KR, Jaberio MF, Alto DL, Magliocca JF. Knowledge, beliefs and attitude of dental and dental hygiene students towards obesity. *J Dent Educ*. 2005; 69(12): 1332-9.
5. Qidwai W, Azam SI. Knowledge, attitude and practice regarding obesity among patients at Aga Khan University Hospital, Karachi. *J Ayub Med Coll Abbottabad*. 2004; 16(3): 32-4.
6. Nyaruhucha CN, Achen JH, Msuya JM, Shayo NB, Kuwa KB. Prevalence and awareness of obesity among people of different age groups in educational institutions in Morongo, Tanzania, East Afr Med J. 2003; 80(2): 68-72.
7. Bocquier A, Paraponaris A, Gourheux JC, Lussault PY, Basdevant A, Verger P. Obesity management knowledge, attitudes and practices of general practitioners in southeastern France; results of a telephone survey.(Article in French; English Translation-Pubmed). *Presse Med*. 2005; 34(11): 769-75.



Pattern reversal visual evoked potentials in cases of optic neuropathies of various causes

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Abstract

Objective: To evaluate the transient Pattern reversal visual evoked potentials (t-PRVEP) changes in patients with optic neuropathies (optic atrophy/optic neuritis) of different causes in a cross sectional study in the Neuro-physiology laboratory, Department of Physiology, MGIMS, Sewagram, Maharashtra, India. **Methods:** 50 clinically diagnosed cases (4-75years) of optic neuropathies including both unilateral and bilateral involvements and those with established causes of optic neuropathies were evaluated for t-PRVEP changes. Abnormalities were defined based on normative values obtained from PRVEP recordings in 146 normal subjects (292eyes) within the age range of 1-75 years. Data analysis of the latency, amplitude and duration of the main positive wave P100 were done by regression studies and ANOVA by using ORIGIN Software. **Results:** Significant changes in P100 latency, amplitude and duration were obtained in all the affected eyes. Out of the 100 eyes examined, 88 affected eyes showed definite PRVEP changes (amplitude reduction + latency prolongation & widening duration). 23(63.89%) out of 36 eyes which showed absent VEP or P100 latency prolongation beyond 130ms + amplitude reductions were cases of clinically diagnosed

optic atrophy while 50 eyes (96.15%) of 54 eyes with P100 latency between 100ms and 130ms were clinically diagnosed cases of optic neuritis or those with known causes of optic neuropathies. 12 eyes showed no significant changes. **Conclusion:** The study confirms that PRVEP evaluation is a very sensitive diagnostic tool for optic neuropathies. It also suggests that the extent/degree of latency prolongation may be a good indicator for the severity of functional disruption of the optic nerve. This may prove to be one of the good prognostic indicators in these cases to enable early intervention and management to influence the treatment outcome.

Key words: PRVEPs, optic neuropathies

Introduction

Pattern reversal visual evoked potentials (PRVEPs) abnormalities in cases of optic neuritis and neurological diseases like multiple sclerosis are well established¹⁻⁴. Their usefulness in neurological and neuro-ophthalmic evaluation of clinical and sub-clinical lesions of the visual pathways is beyond doubt. It presents a non invasive, low-cost and easy method to determine sub-clinical lesions of the optic nerve, making it a very important tool in the diagnosis of demyelinating diseases besides other uses.

Celesia et al⁵ in 1986 have attempted to develop a method to differentiate demyelination from axonal damage to the optic nerve by simultaneous recording of pattern

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Electro retinography (PERGs) and visual evoked potentials in 35 patients of clinical multiple sclerosis. They demonstrated that the concomitant use of P-ERG and PRVEPs results in a better classification of the type and severity of dysfunction affecting the optic nerve. However, no report correlating the PRVEPs abnormalities in optic neuropathies with their severity is found in any literature till date.

In the present study, we recorded PRVEPs in 100 eyes of 50 patients with the clinical diagnosis of optic neuropathy ranging from optic atrophy to optic neuropathies of different known causative factors like retrobulbar neuritis, multiple sclerosis, unilateral trauma to one eye, glaucoma, diabetes mellitus, hypertensive retinopathy, Leber's neuropathies, etc. We studied the various PRVEP changes with an attempt to correlate them with the severity of optic neuropathy so as to determine the discriminating value of the test in cases of optic neuropathies resulting from various causes.

Material and methods

50 patients (18 females, 32 males) within the age range of 4-78 years with the clinical diagnosis of optic atrophy or optic neuropathies of different causes including unilateral involvement were recruited from those referred for Neuro-physiological evaluations to the Neurophysiology laboratory of the Department of Physiology, MGIMS, Sewagram, India from various Departments like Ophthalmology, Medicine, and Pediatrics of Kasturba Hospital and MGIMS, Sewagram. All patients have undergone a complete neuro-ophthalmic examination including fundoscopy favouring optic neuropathies in at least one eye before being referred to the lab. Both eyes of each subject were included in the study making the total eyes studied to 100.

recording PRVEPs from 146 normal subjects (108 males +38 females, 292 eyes) in the age range of 1-75 years before the study was carried out. Prior ethical committee approval and informed consent of the subjects were taken before the study.

The recording was done monocularly on an indigenous Evoked Potential Recorder (RMS EMG.EP MARK II, Chandigarh, India) generating a full field black and white checkerboard pattern (check size 8x8) with a central red fixation point. The pattern reverses at a rate of 1.71 Hz with a sweep duration of 300ms. The low cut filters were set at 2 Hz and high cut at 100 Hz. The sensitivity was maintained at 2 μ V and 200 epochs were averaged and recorded twice for reproducibility.

All recordings were made in an air-conditioned, sound-proof, quiet and dark room with the subject seated comfortably at 1 meter distance from the monitor screen. Standard EEG disc electrodes were used and as per recommendations of the IFCN⁶ and ISCEV⁷, the montages were done in accordance to the 10-20 International system of electrode placement^{8,9} with the reference electrode 12cm above the nasion (Fz), the active at 2cm above the inion (Oz) and the ground at the vertex (Cz). The electrode impedance was kept below 5k Ω . The luminance (59candelas/sqm.) and contrast level (80%) were kept constant for all the recordings. Full concentration and alertness of each of the subjects were maintained all throughout the procedure.

Results

Normative values for 292 eyes in the age range of 1-75 years are shown in table 1. Upper limits of normality were established at 99% tolerance limits. A response is taken as

A baseline value was established in the laboratory prior to the study by **Table 1. Normative PRVEP parameters (n=292): Mean latency (ms) & amplitude (μ V) with 99% tolerance limits in brackets**

Age group (in yrs), n=292	N70 latency	P100 Latency	N155 Latency	P100 amplitude (N70-P100)	P100 duration (N70-N155)
1-15(n=60)	66.05(80.15)	97.84(111.64)	146.8(179.8)	12.88	80.8(108)
16-25(n=60)	69.14(79.94)	99.06(110.76)	153.3(191.7)	6.06	79(120)
26-60(n=120)	65.22(80.07)	96.08(110.17)	137.8(166)	5.68	74(101)
>60(n=52)	64.79(83.39)	99.59(113.69)	140.26(163.1)	3.8	75(106)

abnormal if PRVEP is absent or the parameter (latency/amplitude) or its inter-ocular difference exceeds the mean value +2.5 SD of the normal population. The observations in the subjects with optic neuropathies are shown in table 2 and 3.

Table 2. Classification of eyes based on PRVEP changes (n=100)

Group	PRVEP changes	No. of eyes
1	Absent VEP	3
2	P100 latency ≥ 130 ms	33
3	P100 latency between $\geq 110 < 130$ ms	21
4	P100 latency $\geq 100 < 110$ ms	21
5	Only amplitude reduction	10
6	Normal latency & amplitude	12

Table 3. Grouping of affected eyes with PRVEPs abnormalities based on latency & amplitude changes (n=88)

Group	P100 latency	Optic atrophy	Optic Neuritis	Others
A	Absent VEP	3	0	0
B	≥ 130 ms	20	6	7
C	$\geq 110 < 130$ ms+ low amplitude	1	6	14
D	$\geq 100 < 110$ ms+ low amplitude	1	9	11
E	Only Amplitude Reduction	0	2	8
Total		25	23	40

For convenience and effective analysis, each patient is grouped into 3 main classes based on their clinical diagnosis:

1. Optic Atrophy: consisting of clinically diagnosed cases of Primary (5 bilateral+1 unilateral) and secondary optic atrophies (13).
2. Optic neuritis group: those with clinically diagnosed cases of multiple sclerosis (3), retrobulbar neuritis (3), neuroretinitis(1), papillitis (3).

3. Other known causes of optic neuropathy: trauma- unilateral (4), toxic neuropathies (ATT induced neuropathies-2), metabolic neuropathies (diabetes mellitus-1), glaucomatous optic neuropathies (3), hypertensive retinopathies (3), macular degeneration (1), vitreous degeneration (1), Retinitis pigmentosa (2), Leber's neuropathy (1), chronic papilloedema(2), CRAO(1).

12 eyes were found to be normal (3-ATT induced ON, 3- the unaffected eyes of unilateral eye trauma patients; 1 each of optic neuritis, multiple sclerosis, retrobulbar neuritis, papillitis, papilloedema, glaucoma-1). Out of these, 10 eyes represent the unaffected eye of patients with unilateral involvement showing normal fundoscopy and 2 eyes are those of a patient suspected of ATT induced optic neuropathy. Therefore the number of eyes showing PRVEP abnormalities (prolongation of P100 latencies + amplitude reduction or reduction of amplitude alone-10) was found to be 88.

These 88 eyes are then grouped (table 3) into 5 classes according to the P100 latency obtained in the study. If the P100 latency falls within normal limits, then amplitude reduction of more than 2 μ V from the normative mean for the age group or inter ocular difference of more than 2 μ V is taken as abnormal.

The PRVEP recordings in bilateral optic atrophy, unilateral optic atrophy and in bilateral optic neuritis are shown in fig 1. A comparison between the main PRVEP parameter P100-its latency, duration and amplitude in normal subjects and in patients are shown in fig 2.

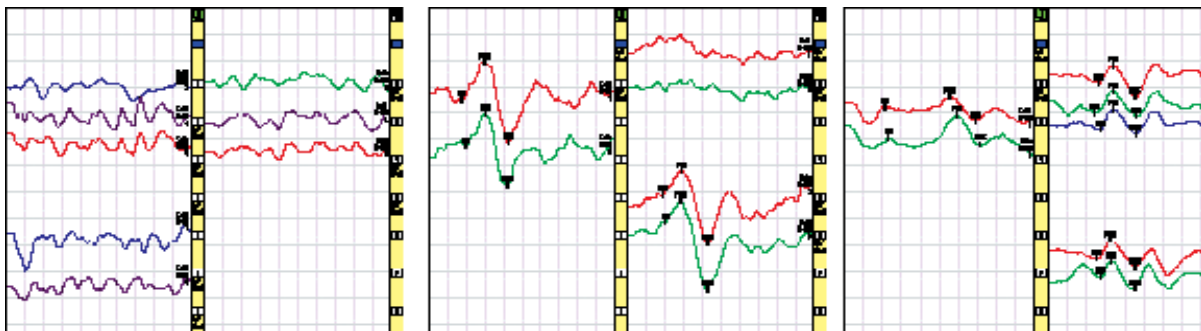


Fig 1. PRVEPs in cases of bilateral optic atrophy (left), unilateral optic atrophy of right eye with hypertensive retinopathy (center) and bilateral optic neuritis (right). Left eye recording are on left side of the graph, right eye on right side and both eyes, lower tracings.

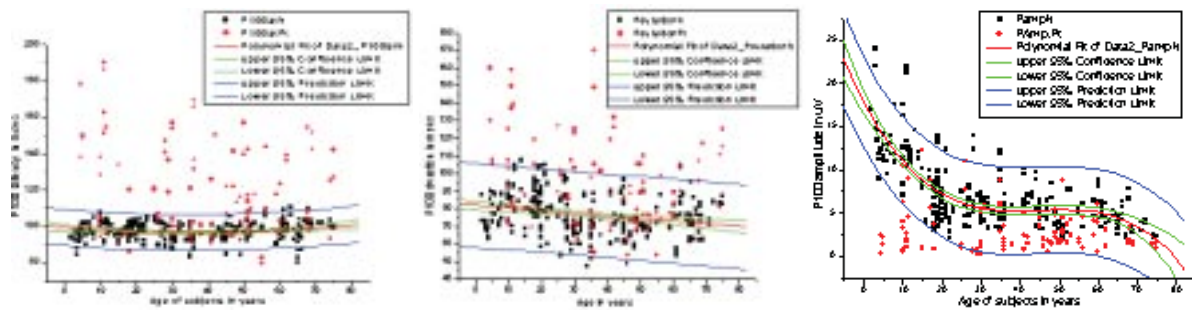


Fig 2. PRVEP parameters in normal subjects and patients with optic neuropathies (Left: P100 latency, Middle: P100 duration and Right: P100 amplitude).

Discussion

The importance of PRVEPs in the diagnosis of optic neuritis has long been established.¹⁻⁴ It is especially of great value in diagnosing nervous system disorders like Multiple sclerosis (MS) even in its subclinical stage before the manifestation of any obvious clinical symptoms which presents only after considerable damage has already been done to the CNS. The most common cause of optic neuritis is MS (70%) which may be one of its very initial presentations. Patients presenting with optic neuritis without any other signs of MS and even normal MRI ultimately may develop MS within 5 years (16%) and around 50% of patients with optic neuritis showing demyelinating lesions on MRI with a high risk of developing clinical MS within 5-10 years¹⁰. This also means that 30% of optic neuritis is due to other causes. Early diagnosis and intervention of any disease is always the best recognized method to minimize the morbidity and mortality associated with it. Hence the importance of detecting early optic neuritis does not need to be further emphasized upon.

As observed by some other workers¹¹, Visual acuity and optic disc appearance especially in young children have been demonstrated to be not reliable for assessing the visual dysfunction but PRVEPs especially serial recordings may be most reliably used for detecting progressive visual dysfunction due to optic nerve involvement.

Celesia et al⁵ in 1986 simultaneously recorded pattern Electroretinography (PERGs) and visual evoked potentials in 35 patients of clinical multiple sclerosis with an attempt to differentiate demyelination from axonal damage to the optic nerve. Korean Medical database¹² in 1992 reported simultaneously recorded

PRVEP, flash VEP and flash ERG on 215 patients and 374 eyes with visual disturbances and observation of PRVEP abnormalities with delayed P1 latency in 76.9% of patients with Retrobulbar neuritis, in 90.5% of Optic Atrophy, 90% of traumatic ischaemic lesions and 50% of retinopathy. Normal latency with reduced amplitude in 45.5% of amblyopia patients was also observed. Their study also observed the usefulness of simultaneous recording of VEP and ERG in assessment of optic atrophy and diabetic retinopathy PRVEP in multiple sclerosis, flash VEP in amblyopia and only flash ERG in pigmentary degeneration of retina.

In the present study, out of the 100 eyes studied, 88 affected eyes showed definite PRVEP changes with amplitude reduction + latency prolongation or widening of duration. 23(63.89%) out of 36 eyes which show either absent PRVEPs or P100 latency beyond 130 ms are accounted for by those cases of optic atrophy while only 6(16.67%) and 7(19.44%) are due to optic neuritis and other causes respectively.

If we consider those eyes with P100 latency above 140ms, then 16 out of 27 eyes are cases of optic atrophy (that is 59.26%) while only 4 (14.82%) are due to optic neuritis (mostly retrobulbar neuritis-2, isolated optic neuritis-2) and 7 (25.93%) are due to other causes (bilateral macular degeneration-2, trauma-1, hypertensive retinopathy-1, CRAO-1 diabetic retinopathy-1, Leber's neuropathy-1).

There were 17 eyes with P100 latency above 150ms, out of which 11(64.71%) are due to optic atrophy and 3(17.65%) each are accounted by the optic neuritis (retrobulbar neuritis-2, isolated optic neuritis-1) and other causes (bilateral macular degeneration-2, trauma-1).

Thus, 11(47.83%) out of 23 eyes with optic

atrophy have latency prolongation beyond 150ms, 5 eyes (21.72%) have latencies between 140-150ms and 7 eyes (30.44%) between 130-140ms.

The 13 eyes which showed P100 latencies beyond 130ms in the optic neuritis group and due to other causes are probably those in severe degree of optic neuropathy or misdiagnosed cases of severe optic atrophy. Considering those eyes with P100 latency beyond 100ms and up to 130ms as cases with mild neuropathies, it is obvious that 50(96.15%) out of 52 eyes are due to optic neuritis and other causes suggesting that these are probably those with milder to moderate degree of neuropathies and appropriate management at this stage may halt the disease process resulting in a better treatment outcome.

However, those eyes with prolongation of P100 latency above 130 ms carry a comparatively poorer prognosis and unless a prompt,

appropriate management is instituted, they may progress to irreversible loss of vision due to complete atrophy of the optic nerve. Therefore, PRVEP abnormalities may help to segregate patients for better management so that vision in patients presenting with optic neuropathies may be salvaged and permanent visual loss be avoided in time.

Conclusion

The study suggests that the extent/degree of latency prolongation may be a good indicator for the severity of functional disruption of the optic nerve. This may prove to be one of the good prognostic indicators in these cases in absence of advanced investigations like PERGs and magnetic resonance imaging techniques to enable early intervention and management to improve the prognosis. Therefore, PRVEPs should always be evaluated for the diagnostic and even prognostic aspects while dealing with any case presenting with optic neuropathies.

References

1. Celesia GG, Daly RF. A new electrophysiological test for diagnosis of optic nerve lesions. *Neurology*. 1977; 27:637-41.
2. Halliday AM, McDonald WI, Mushin J. Visual evoked potentials in patients with demyelinating disease. In: *Visual evoked potentials in man: New developments*. Desmedt JE(Ed). Oxford, England: Clarendon press; 1977:p.438-49.
3. Celesia GG. Visual evoked responses. In: *Evoked potential testing: Clinical applications*. Owen JH, Davis H (Eds). New York: Grune & Stratton; 1985.p.1-54.
4. Neima D, Regan D. Pattern visual evoked potentials and spatial vision in retrobulbar neuritis and multiple sclerosis. *Arch Neurol*. 1984;41:198-201.
5. Celesia GG, Kaufman D, Cone SB. Simultaneous recording of pattern electro-retinography and visual evoked potentials in multiple sclerosis. *Arch Neurol*. 1986; 43:1247-52.
6. Celesia GG, Wollner IB, Chatrian GE, Harding G F A, Sokol S, Spekreijse H. Recommended standards for electroretinograms and visual evoked potentials. An IFCN committee report. *Electroencephal clin Neurophysiol*. 1993; 87:421-36.
7. Odom JV, Bach M, Barber C, Brigell M, Marmor M F, Tormene A P, et al. Visual evoked potentials standard (2004). *Documenta Ophthalmologica*. 2004;108:115-23.
8. American Encephalographic Society. Guideline thirteen: Guidelines for standard electrode position nomenclature. *J. clin. Neurophysiol*. 1994; 11:111-5.
9. Jasper HH. Report of the committee on methods of clinical examination in electroencephalography. *Electroencephal clin Neurophysiol*. 1958; 10:370-5.
10. Kanski J J (Ed). *Neuro-ophthalmology*. In: *Clinical ophthalmology*, 5th edition, Elsevier: Butterworth Heinemann, International publication; 2003:p.601-2.
11. Liasis A, Nischal K K, Walters B, Thompson D, Hardy S, Towell A, et al. Monitoring visual function in children with syndromic craniosynostosis: a comparison of 3 methods. *Arch Ophthalmol*. 2006; 124:1119-26.
12. Korean medical database. Clinical significance of visual evoked potentials and flash electroretinograms in neurological and ophthalmological diseases. *MedRIC*. 1992; 31(1):77-84.



A study of neonatal jaundice (0-14 days)

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Abstract

Objective : To find out the various etiological factors of neonatal jaundice, its correlation of clinical jaundice with total serum bilirubin level for the institution of appropriate therapy and to follow up those complicated cases for neuro-developmental sequelae. **Methods :** 300 new born babies both term and preterm upto 14 day of ages with jaundice, admitted in Pediatric ward, Regional Institute of Medical Sciences(RIMS) Hospital were randomly selected for the study. Clinical assessment of neonatal jaundice was carried out before the collection of first blood samples. Estimation of total serum bilirubin and direct serum bilirubin levels were done using the Modified Jendrassik and Grof's method. Subsequent monitoring of serum bilirubin levels were done daily. The results obtained were analysed and accordingly specific management was given to reduce serum bilirubin to safe levels. Exchange transfusion was planned for those cases where serum bilirubin were >20 mg/dL (term) and >15 mg/dL (pre-term). **Results :** 222 (74%) cases of neonatal jaundice (0 – 14 days) were term while 78 (26%) were preterm neonates. 171 (57%) cases belongs to physiological jaundice while the remaining 129 (43%) cases constituted pathological jaundice.

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Pre-maturity was the commonest cause of pathological jaundice. In 20 (15.5%) cases the underlying causes cannot be determined Kernicterus was observed in 3 cases. There are 3 (1%) deaths out of 300 neonates. **Conclusion :** Physiological, jaundice needs close monitoring of serum bilirubin levels while pathological jaundice needs to be investigated and appropriately managed in order to reduce morbidity and mortality. Though phototherapy was the conventional therapy, exchange transfusion should be intervened timely.

Key words : Jaundice, phototherapy, exchange transfusion, preterm, term.

Introduction

Neonatal jaundice is the most common condition requiring medical attention in newborn babies, affecting about 60% of term and 80% of preterm neonates. It is also a common cause of readmission to hospital after early discharge of newborn babies.¹ Neonatal hyperbilirubinemia, in most cases, is a benign condition. Nonetheless, untreated severe unconjugated hyperbilirubinemia is potentially neurotoxic, has high mortality rate and surviving infants developed neuro-developmental sequelae. Phototherapy is now the standard form of treatment for infants with neonatal hyperbilirubinemia. which is non-invasive, effective, inexpensive and easy to use.^{2,3} When phototherapy fails to control the hyperbilirubinemia, exchange transfusion should be considered. This study was therefore undertaken to study various aspects of neonatal jaundice among newborn babies,

0-14 days of age, admitted in the department of Pediatrics, RIMS Hospital.

Material and methods

A total of 300 newborn babies, both term and preterm, up to 14 days of age, with clinical jaundice, admitted in Pediatric ward, RIMS Hospital between 1st July, 2005 and 31st July, 2007 were randomly selected and included in the study.

Inclusion criteria : Newborns babies with jaundice upto 14 days of age. This included term and per-term babies.

Exclusion criteria : Newborns with major malformations, cholestatic jaundice and post-term neonates were excluded from the study. A detailed history of both mother and baby and meticulous physical examination of the baby were recorded in a pre-tested proforma. The babies were divided into term and preterm as per standard definitions. Clinical assessment of neonatal jaundice was carried out at the time of collection of first blood sample. The level of clinical jaundice was recorded according to very widely used clinical criteria (National Neonatology Forum, 2000).³ First blood samples were subjected to bilirubin estimation within an hour of collection before the child had been subjected to phototherapy. Estimation of total serum bilirubin and direct serum bilirubin levels were done using the Modified Jendrassik and Grof's method. Subsequent monitoring of serum bilirubin levels were done by serial measurements once a day in the mornings between 8:00 am and 9:00 am. Physiological jaundice was diagnosed only after excluding pathological jaundice. Criteria considered for the diagnosis of pathological jaundice included: clinical jaundice detected before 24 hours of age; increase in serum bilirubin of more than 5 mg/dl/24 hrs.; serum bilirubin more than 15 mg/dl; clinical jaundice persisting after 14 days; and direct bilirubin more than 2 mg/dl at any time.³ Conventional phototherapy units were used to provide both conventional (lights kept at a distance of 45cm above body surface) as well as intensive phototherapy (lights kept at a distance of 20cm above body surface) to the jaundiced newborns with significant

hyperbilirubinemia (total serum bilirubin levels of more than 15mg/dL) and to those babies with lower levels of serum bilirubin in the presence of one or more risk factors such as prematurity, sepsis etc. Irradiance was monitored regularly with the help of a radiometer and maintained at a minimum of 4-8 microwatt/cm²/nm. All clinical data, investigation reports, diagnosis and management outcome were recorded and assessed periodically during hospital stay. All neurological deficits in those who recovered from kernicterus were assessed and recorded at the time of discharge for follow up.

Results

A total of 222 term and 78 pre-term babies were studied. Pathological and physiological jaundice constitute 43% (129) and 57% (171) respectively. Table 1 showed the overall etiology of pathological jaundice. There were 56.6% (73) male and 43.4% (56) female babies in pathological jaundice with a male : female ratio of 1.3 : 1 (table 2). Forty four (34.2%) were born to mothers aged 25 years or less while 85 (65.8%) were born to mothers aged more than 25 years.

Table 1. Distribution of etiology of pathological jaundice among neonates (0-14 days).

Etiology	No. of cases	%
Prematurity	41	31.8
Sepsis	26	20.2
ABO Incompatibility	16	12.4
Breast Milk Jaundice	14	10.8
Cephalhematoma	9	6.9
Rh. Isoimmunization	2	1.6
Infant of Diabetic Mother	1	0.8
Undetermined	20	15.5
Total	129	100

Table 2. Distribution of associated risk factors for pathological jaundice (0-14 days).

Risk factors	No. of cases	%	
Sex:	Male	73	56.6
	Female	56	43.4
Maternal Age(Years):	≤ 25	44	34.2
	> 25	85	65.8
Oxytocin:	Yes	78	60.5
	No	51	39.5
Vitamin K:	Yes	73	56.6
	No	56	43.4
Breast Feeding:	Yes	74	57.4
	No	55	42.6
Jaundice in Sibling:	Yes	13	28.3
	No	33	71.7

Table 3 showed that of the 73 term neonates with total serum bilirubin (TSB) >15 mg/dL, 2(2.7%) infants developed kernicterus. Both these infants had TSB levels of >20mg/dL at the time of diagnosis of kernicterus. Of the 39 preterm neonates with TSB >15mg/dL, 1(2.6%) infant developed kernicterus whose TSB level at the time of diagnosis of kernicterus was 18.9mg/dL.

Table 3. Distribution of cases of kernicterus among cases with peak TSB >15 mg/dL.

TSB Peak(mg/dL)	Term		Preterm	
	No. of cases	No. of cases with kernicterus	No. of cases	No of cases with kernicterus
>15-20	55	0	34	1
>20-24	13	1	3	0
>24	5	1	2	0
Total	73	2 (2.7%)	39	1 (2.6%)

Discussion

Jaundice is the commonest abnormal physical finding during the first few weeks of life and clinically detectable on face when serum total bilirubin level is > 5mg/dL compared to > 2 mg/dL in adult.⁴ In our study, prematurity was the commonest cause of pathological jaundice. Our findings were similar to other authors.⁵⁻⁷

Narang et al⁸ have reported that idiopathic hyperbilirubinemia ranged between 8.8% to 57.6%. In our study, the cause remained undetermined in 15.5% (20) of all cases of pathological jaundice.

In the study, sepsis occupies the second cause of pathological jaundice which was a little higher when compared to other workers.^{5,7} Male sex is a known risk factor for pathological jaundice. In our study, there was significant preponderance of male sex which had been documented by other authors.⁹⁻¹¹

In our study oxytocin was used on mothers of 78 out of 129 babies with pathological jaundice. Connor BH and Seaton PG¹² in their prospective study of 1977 babies reported that there were a significant association between the use of oxytocin and the incidence of jaundice.

Vitamin K injection was administered at birth

to 73 babies out of 129 babies with pathological jaundice. There were reports of hyperbilirubinemia after administration of vitamin K in newborns. According to Stoll BJ and Kleigman RM¹³, vitamin K administered to the infant is a risk factor for neonatal hyperbilirubinemia.

As many as 74 out of 129 babies with pathological jaundice were receiving full breast feeds. According to Stoll BJ and Kleigman RM¹³, breast feeding is a risk factor for neonatal hyperbilirubinemia.

Phototherapy was provided to 151 jaundiced babies. Almost all babies were successfully managed with no major side effects. Geiger AM et al¹⁴ did a case-control study for rehospitalisation for jaundice with phototherapy in newborns. None of the rehospitalised infants died. The authors suggested that attention to risks associated with jaundice might reduce the undesirable outcome.

Out of the 300 neonates studied, there were 3 (1%) deaths. One case was a premature, small for gestational age, male newborn (35 wks., 1530 gms.) with Rh. incompatibility who developed kernicterus (18.9 mg/dL). Exchange transfusion was planned but patient party cannot arrange O (-ve) bloods. The other two deaths were attributed to causes other than neonatal jaundice. In a prospective study on 102 neonates with jaundice due to ABO incompatibility by Lucas GN¹⁵, phototherapy was given when the indirect serum bilirubin level exceeds 9 mg/dl. Exchange transfusion was done in 39 cases. However, 8 babies had to undergo multiple exchanges transfusion. There were report of 2(1.9%) deaths.

Kernicterus was observed in three cases (2 term and 1 preterm neonate) constituting 2.7% (3 out of 112 cases). All cases were attributed to hemolytic jaundice [2 cases of Rh. incompatibility (1 term and 1 preterm) and 1 case of ABO incompatibility (term)]. Both term babies with kernicterus recorded high peak bilirubin levels of 23.4 mg/dL and 24.6 mg/dL

at the time of diagnosis of kernicterus while the preterm baby (35wks., 1530 gms.) developed kernicterus at a lower level of 18.9 mg/dL.

Out of the 112 neonates with total serum bilirubin (TSB) levels of more than 15 mg/dL, 80 were successfully followed up for 6 months, 43 for 12 months while only 20 completed 18 months of follow up. 3 babies who recovered from kernicterus, one was followed up for 4 months while the others were followed up for 6 months. Neurological sequelae were noted in both term babies who recovered from kernicterus in the form of generalized hypertonia, dysconjugate eye movements and delayed developmental milestones while one preterm developed

athetoid cerebral palsy. However, longer periods of follow-up are needed to find out the full long-term effects of bilirubin-induced toxicity to the neonatal brain.

Conclusion

The study has revealed that the physiological jaundice is the commonest cause of neonatal jaundice between 0 – 14 days of age while pathological jaundice needs to be investigated, closely monitored and appropriately managed in order to reduce mortality and morbidity. Phototherapy is the standard form of treatment and when it fails to control the hyperbilirubinemia, exchange transfusion should be taken up immediately so that the life of the neonate can be saved without neuro-developmental sequelae.

References

1. Akobeng AK. Neonatal jaundice. *Clinical Evidence* 2004; 11: 1-2.
2. Tan KL. Neonatal jaundice: update on phototherapy management. *Ann Acad Med Singapore* 1993; 22 (2) : 225-8.
3. National neonatology forum. Neonatal jaundice, operationalisation of essential newborn care at district level, national neonatology forum, government of India ;2000.p.52 -8.
4. IAP – NNF Guidelines on Level-II Neonatal Care: Jaundice in newborn; 2006.p.189 – 209.
5. Bahl L, Sharma R, Sharma J. Etiology of neonatal jaundice at Shimla. *Indian Pediatrics* 1994; 31 (10) : 1275-8.
6. Basu K, Das PK, Bhattacharya R, Bhowmik PK. A new look on neonatal jaundice. *Journal of Indian Medical Association* 2002; 100 (9): 556-74.
7. Guaran RL, Drew JH, Watkins AM. Jaundice. *Clinical practice in 88,000 liveborn infants. Aust NZ Journal of Obstetrics and Gynaecology* 1992; 32 (3): 186-92.
8. Narang A, Gathwala G, Kumar P. Neonatal jaundice. An analysis of 551 cases. *Indian Pediatrics* 1997; 34:429-32.
9. Biddulph J, Woodfield DG. Survey of neonatal jaundice in Port Moresby PNG *Med J* 1974; 17(4): 364 – 72.
10. Fok TF, Lau SP, Hui CW. Neonatal jaundice : its prevalence in Chinese babies and associating factors. *Aust Paediatr J* 1986; 22 (3) : 215 – 9.
11. Bracci R, Buonocore G, Garosi G, Bruchi S, Berni S. Epidemiologic study of neonatal jaundice. A survey of contributing factors. *Acta Paediatr Scand Suppl* 1989; 360 : 87 – 92.
12. Connor BH, Seaton PG. Birth weight and use of oxytocin and analgesic agents in labour in relation to neonatal jaundice. *Med J Aust* 1982; 2 (10) : 466– 9.
13. Stoll BJ, Kleigman RM. Jaundice and hyperbilirubinemia in the newborn, *Nelson Text Book of Pediatrics*, Behrman RE, Kleigman RM and Jenson HB, 17th Edn. Philadelphia: Saunders Indian Reprint ; 2004.p.592 – 6.
14. Geiger AM, Petiti DB, Yao JF. Rehospitalisation for neonatal jaundice : risk factors and outcomes, *Paediatr. Perinat. Epidemiol* 2001;15(4):352-8.
15. Lucas GN. Neonatal jaundice due to ABO incompatibility in Shri Lanka. *Indian J Pediatr* 1996;63(3):381-4.



Attenuation of cardiovascular response to extubation by lignocaine, esmolol and propofol

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Abstract

Objective: To compare effectiveness of lignocaine, esmolol and propofol in attenuating the haemodynamic response to extubation.

Methods: A longitudinal study was conducted in the Anaesthesiology Department, Regional Institute of Medical Sciences, Imphal. Ninety adult patients of ASA I of both sexes for elective surgery under general anaesthesia were distributed into three groups (n=30) viz. Group I - 2% Injection lignocaine 1.5mg kg⁻¹ intravenous 2-3 minutes prior to extubation, Group II- Injection esmolol 0.5mg kg⁻¹ intravenous 2-3 minute prior to extubation and Group III - 1% Injection propofol 0.5mg kg⁻¹ intravenous 2-3 minutes prior to extubation. Heart rate (HR), mean arterial pressure (MAP) and ECG changes were measured just before the study drug injection, before extubation, after extubation at 1 minute, 2 minutes, 3 minutes, 5 minutes and 10 minutes in all the three groups. **Results:** Intravenous esmolol (0.5mg/kg⁻¹) 2-3 minutes before extubation effectively attenuated the tachycardiac response and is found to be more effective than propofol (0.5mg/kg⁻¹) or lignocaine (1.5mg/kg⁻¹). However all the three drugs are effective in attenuating the heart rate. Propofol at the dose of 0.5mg/kg⁻¹ given intravenously 2-3 minutes before extubation is found to be more effective in attenuating blood pressure

than esmolol and lignocaine given at the dose of 0.5mg/kg⁻¹ and 1.5mg/kg⁻¹ respectively.

Conclusion: Esmolol is relatively effective in attenuating the HR right from pre extubation period. Propofol gives better results in decreasing blood pressure right from pre-extubation period.

Key words: Haemodynamic response, extubation, lignocaine, esmolol, propofol.

Introduction

Tracheal extubation is known to cause significant increase in heart rate and arterial blood pressure. Though these rises are transitory and of little consequences in healthy young individuals, it causes deleterious effect in hypertensive and patients with coronary artery disease. The hypertensive episodes following tracheal extubation are associated with an increased incidence of cerebral hemorrhage, myocardial ischemia and pulmonary edema. At the same time, tracheal extubation may also lead to unwanted airway reflexes resulting in coughing, laryngospasm and bronchospasm.¹

Intravenous lignocaine hydrochloride prevents the cardiovascular response to extubation by causing direct cardiac depression and by peripheral vasodilatation.² In addition, it attenuates both mechanically and chemically induced airway reflexes and because of its anti-arrhythmic properties it is of special benefit to patients with underlying cardiovascular disease.³

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The effects of pharmacological agents acting directly on the cardiovascular system have been investigated. The cardioselective beta-adrenoreceptor antagonist esmolol hydrochloride, because of its rapid onset and short duration of action, makes it an ideal agent to prevent acute increase in heart rate and arterial blood pressure occurring at extubation.⁴ The two most prevalent theories of the cause of emergence hypertension after any surgical procedure include increased blood levels of catecholamine and increased sympathetic stimulation.⁵ The efficiency of esmolol, in the treatment of immediate postoperative hypertension, is consistent with these theories.⁶ Esmolol also blunts the cerebral blood flow velocity increase during emergence from anaesthesia in neurosurgical patients.⁷ Because of its rapid onset of action and short duration, propofol is considered as an ideal drug for attenuating the cardio-vascular response to tracheal extubation and this may be attributed to its vasodilating effect and negative inotropic properties.⁸ Moreover, the ability of propofol to obtund laryngeal reflexes accounts for smooth emergence from general anaesthesia. The present study is designed to compare the effects of intravenous lignocaine, intravenous esmolol and intravenous propofol for the attenuation of cardio-vascular response to extubation in normotensive patients in the quest for a better agent or method.

Material and methods

A longitudinal randomized double blind study was conducted in the department of Anaesthesiology, Regional Institute of Medical Sciences, Imphal to study the effectiveness of lignocaine, esmolol and propofol in attenuating the haemodynamic response to extubation and to compare their relative effectiveness. Ninety adult patients of ASA grade I of both sexes scheduled for various elective surgical procedures under general anaesthesia were randomly distributed into three groups (n=30) as under, group I - 2% injection lignocaine hydrochloride 1.5mg kg⁻¹

(intravenous) i.v 2-3 minutes prior to extubation, group II- injection esmolol hydrochloride 0.5mg kg⁻¹ i.v 2-3 minutes prior to extubation and group III-1% injection propofol 0.5mg kg⁻¹ i.v 2-3 minutes prior to extubation. Patients with history of hypertension, coronary artery disease, cerebro-vascular disease, bronchospasm and possibility of difficult intubation were excluded from this study. Heart rate (HR), mean arterial pressure (MAP) and ECG changes were measured just before the injection of the study drug, before extubation, after extubation at 1 minute, 2 minutes, 3 minutes, 5 minutes and 10 minutes in all three groups, and the data were analysed statistically.

Results

It is observed from table 1 that there was a uniform sex distribution in the three groups with 11 males and 19 females each. Again the age distribution in different study groups were comparable having mean age of 34.96± 12.24 years in group I, 36.73±11.51 years in group II and 30.76±10.79 years in group III. Mean body weight of the patients in the three groups were 54.36±7.69kg, 54.90±9.60kg and 51.10±8.34kg respectively.

Table 1. Sex, age and weight wise distribution of patients according to groups (Mean ± S.D)

Patient characteristics	Group I (n=30)	Group II (n=30)	Group III (n=30)
Sex (M: F)	11:19	11:19	11:19
Age (years)	34.96±12.24	36.73±11.51	30.76±10.79
Weight (Kg)	54.36±7.69	54.90±9.60	51.10±8.34

As shown in table 2A and 2B, the heart rate decreased significantly from the pre-injection value right from 1 minute after extubation till the 10th minute in all the three groups. There was some visible increase in heart rate at pre-extubation period in lignocaine group which is statistically insignificant.

At pre extubation, MAP slightly raised above preinjection values in lignocaine group as shown in table 3A. However the rise was statistically insignificant. In contrast to this, there was fall in MAP at this stage compared to pre-injection value in both esmolol and propofol group. After extubation at 1 minute,

Table 2A. Results of paired "t" test within groups for heart rate at different time interval

Group	Time	Mean \pm S.D.	df	t-value	p-value
I	Pre injection	109.70 \pm 16.961			
	Pre extubation	111.97 \pm 18.115	29	1.003	0.324
	After extubation at 1 min	104.10 \pm 17.94	29	2.587	0.015
	At 2 minute	99.80 \pm 16.60	29	4.563	<0.001
	At 3 minute	96.23 \pm 16.38	29	6.482	<0.001
	At 5 minute	91.93 \pm 15.47	29	8.871	<0.001
	At 10 minute	81.23 \pm 10.95	29	9.897	<0.001
II	Pre injection	109.67 \pm 16.59			
	Pre extubation	101.60 \pm 12.76	29	3.033	0.005
	After extubation at 1 min.	97.20 \pm 14.52	29	4.372	<0.001
	At 2 minute	95.13 \pm 13.96	29	4.409	<0.001
	At 3 minute	93.17 \pm 14.26	29	6.386	<0.001
	At 5 minute	90.90 \pm 12.53	29	7.684	<0.001
	At 10 minute	83.30 \pm 11.05	29	10.230	<0.001
III	Pre injection	108.23 \pm 19.75			
	Pre extubation	103.90 \pm 15.43	29	1.454	0.157
	After extubation at 1 min.	96.93 \pm 14.86	29	3.883	0.001
	At 2 minute	93.23 \pm 14.59	29	5.299	<0.001
	At 3 minute	89.87 \pm 15.07	29	7.063	<0.001
	At 5 minute	88.40 \pm 14.74	29	8.748	<0.001
	At 10 minute	78.13 \pm 13.11	29	11.264	<0.001

Table 2B. Results of independent "t" test between groups for heart rate at different time intervals

Group	Time	Mean \pm S.D.	Df.	t-value	p-value
I VS II	Pre injection	109.70 \pm 16.96	58	0.008	0.994
	Pre extubation	109.67 \pm 16.59	58	2.562	0.013
		111.97 \pm 18.11			
	After extubation at 1 min.	104.10 \pm 17.94	58	1.637	0.107
		97.20 \pm 14.52			
	At 2 minute	99.80 \pm 16.60	58	1.178	0.243
		95.13 \pm 13.96			
	At 3 minute	96.23 \pm 16.38	58	0.773	0.443
		93.17 \pm 14.26			
	At 5 minute	91.93 \pm 15.47	58	0.284	0.777
		90.90 \pm 12.53			
	At 10 minute	81.23 \pm 10.95	58	0.727	0.470
		83.30 \pm 11.05			
	I VS III	Pre injection	109.70 \pm 16.96	58	0.309
Pre extubation		108.23 \pm 19.75	58	1.856	0.068
		111.79 \pm 18.11			
After extubation at 1 min.		103.90 \pm 15.43	58	1.685	0.097
		104.10 \pm 17.94			
At 2 minute		96.93 \pm 14.86	58	1.627	0.109
		99.80 \pm 16.60			
At 3 minute		93.23 \pm 14.59	58	1.566	0.123
		96.23 \pm 16.38			
At 5 minute		89.87 \pm 15.07	58	0.906	0.369
		91.93 \pm 15.47			
At 10 minute		88.40 \pm 14.74	58	0.994	0.325
		81.23 \pm 10.95			
II VS III		Pre injection	109.67 \pm 16.59	58	0.304
	Pre extubation	108.23 \pm 19.75	58	0.629	0.532
		101.60 \pm 12.76			
	After extubation at 1 min.	103.90 \pm 15.43	58	0.070	0.944
		97.20 \pm 14.52			
	At 2 minute	96.93 \pm 14.86	58	0.515	0.608
		95.13 \pm 13.96			
	At 3 minute	93.23 \pm 14.59	58	0.871	0.387
		93.17 \pm 14.26			
	At 5 minute	89.87 \pm 15.07	58	0.708	0.482
		89.87 \pm 15.07			
	At 10 minute	90.90 \pm 12.53	58	1.650	0.104
		88.40 \pm 14.74			
		83.30 \pm 11.05			
	78.13 \pm 13.11				

MAP was found decreased in all the 3 groups. The decrease in MAP was highly significant in lignocaine group, very highly significant in esmolol group and highly significant in propofol group. After extubation from 2 minute onward, MAP decreased significantly in all the three groups. Preinjection values of MAP were comparable in all three groups which is statistically insignificant ($p>0.05$). From table 3B, it is observed that there was no variation in mean values at each stage of time between lignocaine (group I) and esmolol (group II). On the contrary, there were significant variations of means between lignocaine (group I) and propofol (group III) at 2nd, 3rd and 10th minutes after extubation. Significant difference was observed at 2nd and 3rd min. while comparing between esmolol (group II) and propofol (group III).

No ECG changes were observed in any of the patients of the three groups. Extubation score: The extubation score was 0 in 27 cases of Group I and in 29 cases of Group II and III as shown in table 4.

Table 3A. Results of paired at "t"-test within groups for mean arterial pressure (MAP) at different time-intervals

Group	Time	Mean \pm S.D.	df	t-value	p-value
I	Pre injection	118.27 \pm 14.76			
	Pre extubation	120.17 \pm 17.75	29	0.736	0.468
	After extubation at 1 min.	110.93 \pm 12.86	29	3.286	0.003
	At 2 minute	106.47 \pm 12.53	29	5.228	<0.001
	At 3 minute	104.03 \pm 12.24	29	6.063	<0.001
	At 5 minute	101.93 \pm 13.22	29	7.439	<0.001
	At 10 minute	92.93 \pm 8.71	29	10.607	<0.001
II	Pre injection	117.93 \pm 13.54			
	Pre extubation	115.77 \pm 14.62	29	0.956	0.347
	After extubation at 1 min.	109.77 \pm 13.95	29	3.522	0.001
	At 2 minute	106.63 \pm 13.63	29	4.998	<0.001
	At 3 minute	103.80 \pm 12.92	29	6.111	<0.001
	At 5 minute	100.80 \pm 12.33	29	8.262	<0.001
	At 10 minute	91.37 \pm 10.32	29	11.769	<0.001
III	Pre injection	112.50 \pm 13.84			
	Pre extubation	109.17 \pm 14.04	29	1.123	0.271
	After extubation at 1 min.	105.00 \pm 10.26	29	2.481	0.019
	At 2 minute	99.97 \pm 9.81	29	4.427	<0.001
	At 3 minute	97.07 \pm 10.58	29	4.783	<0.001
	At 5 minute	96.93 \pm 10.36	29	5.227	<0.001
	At 10 minute	87.93 \pm 7.20	29	10.238	<0.001

Table 3B. Results of the independent "t"-test between groups for mean arterial pressure (MAP) at different time intervals.

Between groups	Time	Mean \pm S.D.	df	t-value	p-value
I VS II	Pre injection	118.27 \pm 14.76	58	0.091	0.928
		117.93 \pm 13.54			
	Pre extubation	120.17 \pm 17.75	58	1.047	0.299
		115.77 \pm 14.62			
	After extubation at 1 minute	110.93 \pm 12.86	58	0.337	0.738
		109.77 \pm 13.95			
	At 2 minute	106.47 \pm 12.53	58	0.049	0.961
		106.63 \pm 13.63			
	At 3 minute	104.03 \pm 12.24	58	0.072	0.943
		103.80 \pm 12.92			
I VS III	At 5 minute	101.93 \pm 13.22	58	0.343	0.733
		100.80 \pm 12.33			
	At 10 minute	92.93 \pm 8.71	58	0.635	0.528
		91.37 \pm 10.32			
	Pre injection	118.27 \pm 14.76	58	1.560	0.124
		112.50 \pm 13.84			
	Pre extubation	120.17 \pm 17.75	58	2.661	0.010
		109.17 \pm 14.04			
	After extubation at 1 minute	110.93 \pm 12.86	58	1.975	0.053
		105.00 \pm 10.26			
II VS III	At 2 minute	106.47 \pm 12.53	58	2.236	0.029
		99.97 \pm 9.81			
	At 3 minute	104.03 \pm 12.24	58	2.357	0.022
		97.07 \pm 10.58			
	At 5 minute	101.93 \pm 13.22	58	1.630	0.109
		96.93 \pm 10.36			
	At 10 minute	92.93 \pm 8.71	58	2.423	0.019
		87.93 \pm 7.20			
	Pre injection	117.93 \pm 13.54	58	1.536	0.130
		112.50 \pm 13.84			
Pre extubation	115.77 \pm 14.62	58	1.783	0.080	
	109.17 \pm 14.04				
After extubation at 1 minute	109.77 \pm 13.95	58	1.507	0.137	
	105.00 \pm 10.26				
At 2 minute	106.63 \pm 13.63	58	2.173	0.034	
	99.97 \pm 9.81				
At 3 minute	103.80 \pm 12.92	58	2.207	0.031	
	97.07 \pm 10.58				
At 5 minute	100.80 \pm 12.33	58	1.315	0.194	
	96.93 \pm 10.36				
At 10 minute	91.37 \pm 10.32	58	1.494	0.141	
	87.93 \pm 7.20				

Table 4. Extubation score in the three groups

Extubation score	Group I	Group II	Group III	Total
0	27	29	29	85
1	3	1	1	5
2	-	-	-	-
3	-	-	-	-

Discussion

Studies by Wohlner EC et al⁹ showed ineffectiveness of lignocaine in attenuating the haemodynamic response up to 10 minutes after extubation. Fujii Y et al¹⁰ also observed that lignocaine attenuated HR and MAP only 3 minutes onward after extubation. Contrary to the above reports Bidwai AV et al¹¹ concluded in their study that intravenous injection of lignocaine (1mg/kg) administered 2 minutes before tracheal extubation prevents coughing and rise in blood pressure and HR at or after extubation or in recovery room. Similar findings were also observed by Wallin G et al¹² and Mikawa K et al¹³. This is in concurrence with the findings in the present study where the effectiveness of lidocaine in attenuating the tachycardiac response to extubation was observed from 1 minute onwards up to 10 minutes.

Most of the studies using esmolol to attenuate haemodynamic response like tachycardia and hypertension following extubation indicate effectiveness of the drug for the purpose.^{4,14-16} Similarly in the present study, the heart rate reduced significantly right from pre extubation period up to 10 minute postextubation in the esmolol group which signifies that esmolol is more effective in reducing rise in heart rate during endotracheal extubation.

Very few studies have been undertaken to evaluate effects of propofol on cardiovascular attenuation during extubation. Eshak Y et al⁸ administered propofol 0.5mg kg⁻¹ i.v two minutes before extubation resulted in attenuation of HR and blood pressure at extubation as well as one minute after extubation. In a study by Chhabra B et al¹⁶, propofol 0.5mg. kg⁻¹ intravenous given 2 minutes before extubation attenuated the hypertensive response more effectively than tachycardiac response. In our study, propofol reduced the heart rate significantly from 1 minute onwards after extubation signifying that

propofol is also very effective in reducing heart rate during tracheal extubation.

Conclusion

It is observed that intravenous esmolol at a dose of 0.5mg/kg⁻¹ effectively attenuated the tachycardiac response during extubation and is found to be more effective than propofol (0.5mg/kg⁻¹) or lidocaine (1.5mg/kg⁻¹). Whereas, for attenuation of blood pressure, propofol at the dose of 0.5mg/kg⁻¹ given intravenously 2-3 minutes before extubation is found to be more effective than esmolol and lidocaine given at the dose of 0.5mg/kg⁻¹ and 1.5mg/kg⁻¹ respectively.

References

1. Jee D, Park SY. Lidocaine sprayed down the endotracheal tube attenuates the airway – circulatory reflexes by local anaesthesia during emergence and extubation. *Anaesth. Analg.* 2003; 96: 293-7.
2. Himes RS, Difazio CH, Burney RG. Effects of lidocaine on the anaesthetic requirements for nitrous oxide and halothane, *Anesthesiology.* 1977; 47: 437-40.
3. Nishino T, Hiraga K, Sugimore K. Effects of i.v. lignocaine on airway reflexes elicited by irritation of the tracheal mucosa in humans anaesthetized with enflurane. *Br. J. Anaesth.* 1990; 64: 682-7.
4. Lim SH, Chin NM, Tai HY, Wong M, Lin TK. Prophylactic esmolol infusion for the control of cardiovascular responses to extubation after intracranial surgery. *Ann. Acad. Med. Singapore.* 2000; 29: 447-50.
5. Lowrie A, Johnston PL, Fell D, Robinson SL. Cardiovascular and plasma catecholamine response at tracheal extubation. *Br. J. Anaesth.* 1992; 68: 261-3.
6. Muzzi DA, Block S, Tosasso TJ. Labetolol and esmolol in the control of hypertension after intracranial surgery. *Anaesth. Analg.* 1990; 70: 68-71.
7. Grillo P, Bruder N, Auquier P, Pellissier D, Gouin F. Esmolol blunts the cerebral blood flow velocity increase during emergence from anaesthesia in neurosurgical patients. *Anaesth. Analg.* 2003; 91(4): 1145-9.
8. Eshak Y, Farci F, Khalid A, Bhatti TH. Small dose propofol attenuate cardiovascular response to tracheal extubation. *Anaesth. Analg.* 1998; 86: 537.
9. Wohlner EC, Usubiaga LJ, Jacoby RM, Hill GE. Cardiovascular effects of extubation. *Anesthesiology.* 1979; 51: S194.
10. Fujii Y, Saitoh Y, Takahashi S, Toyooka H. Combined diltiazem and lidocaine reduces cardiovascular responses to tracheal extubation and anesthesia emergence in hypertensive patients. *Can. J. Anaesth.* 1999; 46: 952-6.
11. Bidwai AV, Bidwai VA, Roger CR, Stanley TH. Blood pressure and pulse rate responses to endotracheal extubation with and without prior injection of lidocaine. *Anesthesiology.* 1979; 51: 171-3.
12. Wallin G, Cassuto J, Hongstrom S, Linden I, Faxen A, Rimbak G et al. Effects of lidocaine infusion on sympathetic response to abdominal surgery. *Anesthesia and Analgesia.* 1987; 66:1008-13.
13. Mikawa K, Nishina K, Takao Y. Attenuation of cardiovascular responses to tracheal extubation: Comparison of verapamil, lidocaine and verapamil-lidocaine combination. *Anaesth. Analg.* 1997; 85: 1005-10.
14. Dyson A, Isaac PA, Pennant JH, Giesecke AH, Lipton JM. Esmolol attenuates cardiovascular responses to extubation. *Anaesth. Analg.* 1990; 71: 675-9.
15. Kovac A, Masiogal A, Davis T. Comparison of Nicardipine versus Esmolol in attenuating haemodynamic responses to Anaesthesia emergence and extubation. *Anesth. Analg.* 1998; 86: S551.
16. Chhabra B, Malhotra N, Bhardwaj M, Goel GK. Haemodynamic response to extubation: Attenuation with propofol, lidocaine and esmolol. *J. Anaesth. Clin. Pharmacol.* 2003; 19 (3): 283-8.



A study on laparoscopic cholecystectomy in acute cholecystitis

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Abstract

Objective: Elective laparoscopic cholecystectomy (LC) has almost replaced the conventional open cholecystectomy (OC). The role of LC in the management of acute cholecystitis (AC) is far less established and debatable in terms of morbidity, postoperative complication, timing of surgery and conversion. The aim of this study was to evaluate the safety and feasibility of LC in AC.

Methods: This study was conducted in Surgical GI and MAS unit, Department of Surgery, Regional Institute of Medical Sciences(RIMS) Hospital, Imphal, over the period of 22 months. All patients underwent LC after diagnosis of AC was established by physical examination and by series of investigations. The results thus obtained were recorded systematically. **Results:** Average age of the patients of sample (n=50) under consideration was 42.38years. The duration of acute Symptoms was 36.32 ± 18.85 hours. The average operative time was 78.16 ± 32 minutes and timing of operation was 47.52 ± 18 hours. One patient was converted to OC due to uncontrolled bleeding. Overall postoperative complications were 18%. One patient developed bile leak postoperatively and was managed conservatively. The postoperative

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hospital stay was 59.52 hours and the total hospital stay of 96.40 ± 21.96 hours.

Conclusion: Laparoscopic cholecystectomy for acute cholecystitis which was contraindicated in the initial period of laparoscopic surgery is now a safe procedure. Laparoscopic cholecystectomy should be carried out as soon as the diagnosis of acute cholecystitis is established.

Key words: Acute cholecystitis, laparoscopic cholecystectomy, feasibility.

Introduction

Cholelithiasis has plagued the mankind for over 2000 years. The treatment remained relatively primitive and ineffective until Karl Langenbuch, a noted German Surgeon, performed the first successful cholecystectomy in 1882, which marked the beginning of the successful management of the disease. For the last 100 years open cholecystectomy remained the gold standard for the definitive management of symptomatic gallstone disease. Since the first reported LC by Francois Dubois in April 1988¹, the procedure had gained a foothold and has now become the gold standard approach for gallstone diseases in elective cases ².

AC was considered to be relatively contraindicated and many surgeons believe that the inflammation and edema present in such patients distort the biliary ductal and vascular anatomy, making laparoscopic treatment difficult and resulting in a significant higher complications rate. LC in AC though practiced

but has not been accepted as the 'Gold standard' treatment in AC due to controversy in terms of morbidity, post-operative complications, timing of surgery and rate of conversion. This study was undertaken to ascertain the safety and feasibility, if any, in overall management of the LC in AC.

Material and methods

This study was conducted in Surgical GI and MAS unit, Department of Surgery, RIMS hospital, Imphal over the period of twenty two months (September 2006 to July 2008) and included patients with clinical diagnosis of AC. Diagnosis of AC was made on the following criteria: history of right upper quadrant or epigastric pain and tenderness, fever $>38^{\circ}\text{C}$, leucocytosis $10,000/\text{mm}^3$, pericholecystic fluid collection, wall distension or thickening, positive USG Murphy's sign, with or without cholelithiasis by ultrasound and duration of acute attack for 4 days. The exclusion criteria were cholecystitis > 4 days, spreading peritonitis, patients with jaundice and pregnancy with acute cholecystitis and cirrhotic patients, coagulation disorders, shock on admission and carcinoma of GB, contraindications to general anesthesia.

The cause of acute cholecystitis was assessed preoperatively by ultrasound, intra-operatively by laparoscopic finding and post operatively from histopathological examination for final diagnosis.

After a detail and complete workup, patients were submitted for LC using four ports. Pneumo-peritoneum was created by infraumbilically placed closed veress needle technique and pressure maintained between 11-14 mmHg. Two 10 mm ports, (1 camera port in umbilicus and 1 working port in the epigastrium), two 5 mm ports (1 working port in right subcostal in the midclavicular line for surgeons left hand and 1 at the level of umbilicus in right anterior axillary line for retracting Gall Bladder) were inserted. After complete diagnostic survey of the intra-abdominal cavity, patient was positioned in reverse trendelenberg position approximately 20-30 degrees and the operating table was turned to the left. Dissection was initiated

directly adjacent to the gall bladder; any adhesion to the gall bladder was taken down. Cystic duct and cystic artery were identified and skeletonized before application of the clips separately. Gall bladder was dissected off from the gall bladder bed of the liver using harmonic scalpel and by blunt dissection. The liver bed was reinspected for adequate hemostasis or bile leak prior to the detachment of the gall bladder. Gall bladder was removed through the epigastric port. The peritoneal cavity was irrigated with normal saline and tube drain was placed through axillary port in the Morrison's pouch when hemorrhage or bile leak was suspected. Fascial closure was done for all the 10mm port site and skin closed with absorbable 000 sutures. Intravenous fluid and injectable analgesics (Diclofenac and tramadol) was continued till the 1st POD and further analgesics were given based on patient's perception of pain. A third generation cephalosporin was continued in complicated patients. Timing of operation, rate of conversion, postoperative complications, post-operative and total hospital stay were recorded.

Results

The present study was based on 50 cases of AC that underwent LC within 96 hours of onset of the symptoms. To achieve the objectives of the present study of LC in AC, the following parameters have been used - timing of operation (in minutes), rate and reason of conversion, postoperative complications and morbidity, and length of the hospital stay (in hours).

The average age of the patients of sample ($n=50$) under consideration was 42.38 ± 14.91 years (mean \pm SD). Females were more in number than the males (table 1). The mean duration of acute onset of symptoms was 36.32 hours with SD of 18.85 hours with almost 100% positive Murphy's sign. Thirty nine (78%) patients here reported with temperature between $37.1 - 39^{\circ}\text{C}$ (table 2).

Forty-seven (94%) patients had gallstone as the cause of acute cholecystitis. Positive USG Murphy's sign was seen in 42 (84%) cases, pericholecystic fluid collection was observed

Table 1. Socio-demographic profile of the patient

Parameters	Numbers (n=50)
Age(years)	42.38(±14.91)*
Sex	
Male	14 (28)!
Female	(72)
Religion	
Hindu	38 (76)
Christian	10 (20)
Muslim	2 (4)
Occupation	
House wife	22 (44)
Govt.servant	12 (24)
Business	8 (16)
Student	3 (6)
Unemployed	(10)

* Mean (± S.D). Figures within parentheses indicate percentage.

Table 2. Clinical Data and laboratory results on admission

Parameter	Numbers n =50
Duration of acute Symptoms*	36.32 ± 18.85 hours
Murphy's sign	
Yes.	50 (100)
No.	0
Abdominal mass (tenderness)	
Yes.	9 (18)
No.	41 (82)
Hb gm%	11.2 (9.6-13.9)
Temperature	
≤ 37 °C	11 (22)
37.1 – 39 °C	39 (78)
≥ 39 °C	0
WBC	
≤ 10,000/Cumm	24 (48)
10,000 – 20,000/mm ³	26 (52)
≥ 20,000/Cumm	00

Figures within parenthesis indicate percentage.

*Mean ± SD.

in 7(14%) patients and 24(48%) cases showed gall bladder wall thickening of 4 mm (table 3).

Table 3. Ultrasonographic findings on initial admission.

USG findings	Numbers (n=50)
Thickened gallbladder wall	24 (48)
Edematous gallbladder wall	16 (32)
Distended gallbladder	20 (40)
Presence of gallstone	47 (94)
USG Murphy's sign	42 (84)
Pericholecystic fluid collection	7 (14)
Sludge in gallbladder	11 (24)

Figures within parenthesis indicate percentage (%).

The duration of LC for AC was 78.16±32 minutes and on an average 90% of the patients who underwent early LC had acute inflammation of the gallbladder identified by congestion and redness of the GB wall (table 4).

Table 4. Intraoperative findings

Intra-operative Findings	Numbers (n=50)
Operative time (minutes)*	78.16±32
Inflamed gallbladder	45 (90)
Empyema GB	6 (12)
Tensely distended GB	13 (26)
Perforated GB	0 (0)
Severe adhesion	37 (74)
Intra-operative bleeding	8 (16)

Figures within parentheses indicate percentage.

* indicates mean ± SD.

On an average overall complication rate of this study was 18% and 2% developed bile leakage on second postoperative day (table 5) which was managed by USG guided percutaneous drainage. However, the patient recovered dramatically and was discharged after 6 days of hospital stay. One (2%) of the patients had to be converted to open cholecystectomy (table 6) due to uncontrolled bleeding associated with huge adhesion obscuring calot's triangle.

Table 5. Post-operative complications

Postoperative complications	Numbers (n=50).
Wound/port site infection	2 (4)
CBD injury	0 (0)
Bile leak	1 (2)
Chest infection	0 (0)
Paralytic ileus	1 (2)
Pyrexia	4 (8)
Pancreatitis	0 (0)
Cardiac complication	1 (2)
Total	9 (18)

Figures within parentheses indicate percentage.

The timing of operation, described by the time between the onset of acute symptoms to laparoscopic cholecystectomy was 47.52±18.27 hours (mean ±SD) (table 6). The mean duration of postoperative hospital stay observed was 59.52±25.06 hours (table 6).

Table 6. Post-operative outcome of LC in acute cholecystitis

Parameters	Numbers (n=50)
Timing of operation (hours)*	47.52±18.27
Postoperative analgesia (hours)*	37.44±17.41
Postoperative Hospital stay (hours)*	59.52±25.06
Total Hospital stay (hours)*	96.40±21.96
Conversion to OC!	01 (2)
Mortality rate!	00 (0)

Figures within parentheses indicate percentage.

*Mean (± S.D).

Two patients, one with ventricular fibrillation and another bile leak had postoperative stay of 144 hours (6 days). No mortality and CBD injury were reported in this study.

Discussion

Laparoscopic cholecystectomy for interval cholecystectomy has been already accepted as gold standard procedure. However, laparoscopic cholecystectomy for acute cholecystitis was contraindicated initially as it was associated with high rate of conversion and technical difficulty due to obscured anatomy. While laparoscopic cholecystectomy has gained a leading role in the treatment of acute cholecystitis, there is still some debate as to the optimal timing of operation. Many studies have varied reports of higher conversion rate when surgery was carried out 48 hours after admission or later as an elective procedure³ or after 4 days^{4,5}. Similarly, there is no difference in conversion rate when laparoscopic cholecystectomy is performed before and after 96 hours of admission.⁶ This study showed that timing of operation of 47.52 hours has got no any influence on the conversing rate and complications.

Conversion rates in acute cholecystitis resulting from the technical difficulty of identifying biliary anatomy and managing severe inflammatory adhesions around the acutely inflamed Gall bladder varies in different literatures. Many studies have showed conversion rate ranging from 14% to 38.6% for acute cholecystitis and 9.6% for elective laparoscopic cholecystectomy with decreasing conversion rate from the beginning to the end of the study.^{7,8} However, this study shows a conversion rate of 2% which is similar with other studies.^{9,10}

Two percent of the patient developed bile leak postoperatively which is similar to other studies.¹¹ However, 4% port site wound infection in this study is much less than the other studies.¹² Overall postoperative complications rate in patient undergoing LC for AC is 18% which is similar with other literature.^{13,14} No CBD injury, chest infection, pancreatitis, and mortality were reported in the study.

Many literatures and studies have reported prolonged hospital stay in patients undergoing laparoscopic cholecystectomy for acute cholecystitis due to high rate of conversion and postoperative complications.¹² In the present study we have found that the postoperative hospital stay was 2.4 days and total hospital stay 4.02 days which are similar as reported in other studies.¹⁵ Two patients had to stay for 8 days in hospital, one due to development of ventricular fibrillation during recovery from anesthesia and another bile due to leak on second postoperative days. Both the patients recovered with conservative management.

Conclusion

The study has revealed that laparoscopic cholecystectomy for acute cholecystitis is a safe procedure and Laparoscopic cholecystectomy should be carried out as soon as the diagnosis of acute cholecystitis is established. Early laparoscopic cholecystectomy yields both medical and socioeconomic benefits, in terms of a briefer hospital stay and accelerated recuperation period. Early laparoscopic cholecystectomy also offers definitive treatment during the same admission and avoids the problems associated with failed conservative management. Conversion to open surgery has become a marker of a truly difficult cholecystectomy.

References

1. Nagy AG, Patterson EJ. Laparoscopic surgery historical perspectives, surgical laparoscopy, Zucker KA editor. 2nd edition. USA: Lippincott Williams and Wilkins 2001.p.7.
2. Mason EM, Duncan DT. Laparoscopic cholecystectomy. Mastery of endoscopic and laparoscopic surgery, Eubanks WS, Swanstrom LL, Soper JN editors. USA:

- Lippincott Williams and Wilkins 2000.p.241.
3. Peng WK, Sheikh Z, Nixon SJ, Paterson B. Role of laparoscopic cholecystectomy in the early management of acute gall bladder disease. *Br JS* 2005; 92:586-91.
 4. Navez B, Mutter D, Russier Y, Vix M, Jamali F, Lipski D, et al. Safety of laparoscopic approach for acute cholecystitis. Retrospective study of 609 cases. *World Journal of Surgery* 2001; 25(10): 1352-6.
 5. Samuel E, Edmond S, Ernest N, Jack A, Ibrahim M. Laparoscopic cholecystectomy for acute cholecystitis: Prospective trial. *World J Surg* 1997; 21:540-5.
 6. Knight JS, Mercer SJ, Somers SS, Walters AM, Sadek SA, Toh SKC. Timing of urgent laparoscopic cholecystectomy does not influence conversion rate. *Br JS* 2004;91:601-4.
 7. Pessaux P, Tuech JJ, Rouge C, Duplessis R, Cervi C, Arnaud P. Laparoscopic cholecystectomy in acute cholecystitis: A prospective comparative study in patients with acute Vs chronic cholecystitis. *Surg Endosc*. 2000; 14:358-61.
 8. Soffer D, Blackbourne H, Schulman CI, Goldman M, Habib F, Benjamin R, et al. Is there an optional time for laparoscopic cholecystectomy in acute cholecystitis ? *Surg Endosc* 2007; 21:805-9.
 9. Wang YC, Yang HR, Chung PK, Jeng LB, Chen RJ. Urgent laparoscopic cholecystectomy in the management of acute cholecystitis: Timing does not influence conversion rate. *Surg Endosc* 2006; 20:806-8.
 10. George T, Dimitris Z, Paraskevi L, Theodoros T, George P, Constantine H. Timing of Laparoscopic cholecystectomy for acute cholecystitis: Prospective non randomized study. *World J Gastroenterol* 2006; 12(34):5528-31.
 11. Cox MR, Wilson TG, Luck AJ, Jeans PL, Padbury RTA, Toouli J. Laparoscopic cholecystectomy for acute Inflammation of the gallbladder. *Annals of Surgery* 1993; 218(5): 630-4.
 12. Lo CM, Liu CL, Fan ST, Lai EC, Wong J: Prospective randomized study of early versus delayed laparoscopic cholecystectomy for acute cholecystitis. *Annals of Surgery* 1998; 227 (4): 462-7.
 13. Kolla SB, Aggarwal S, Kumar A, Kumar R, Chumber S, Prasad R, et al. Early versus delayed laparoscopic cholecystectomy for acute cholecystitis. A prospective randomized trial. *Surg Endosc* 2004; 18(9) : 1323.
 14. Johansson M, Thune A, Stierstam M, Westman B, Lundell L. Randomized clinical trial of open versus Laparoscopic cholecystectomy for acute cholecystitis. *Br JS* 2005; 92: 44-9.
 15. Kum CM, Eypasch E, Lefering R, Paul A, Neugebauer E, Troidl H. Laparoscopic cholecystectomy for acute cholecystitis. Is it really safe? *World J. Surg* 1996;20:43-9.



Post-operative epidural analgesia – comparison of bupivacaine plus buprenorphine with bupivacaine alone

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Abstract

Objective: The study was conducted to investigate whether the combination of epidural bupivacaine and buprenorphine provides longer duration of post-operative analgesia than epidural bupivacaine alone. **Methods:** Sixty adult patients were divided into 2 groups of 30 patients each. 2 hours after spinal anaesthesia group A patients received 8ml of 0.25% bupivacaine along with 0.1 mg buprenorphine, and group B patients received 8 ml of 0.25% bupivacaine alone through prefixed epidural catheter. **Results:** Duration of post-operative analgesia with epidural combination of bupivacaine plus buprenorphine and epidural bupivacaine alone, were 12.23 ± 1.72 hours and 4 ± 0.69 hours respectively. **Conclusion:** Combination of epidural bupivacaine and 0.1 mg of buprenorphine provides longer duration of post-operative analgesia than epidural bupivacaine alone and is recommended in patients undergoing gynaecological and lower limb orthopaedic surgery.

Key words: Epidural analgesia, post-operative analgesia, bupivacaine, buprenorphine.

Introduction

Post-operative pain is probably the most

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severe form of pain suffered by human being. Combined spinal and epidural anaesthesia is a recent innovation of regional anaesthesia by which the advantages of subarachnoid and epidural anaesthesia are retained and combined.

Some workers claimed that addition of buprenorphine to local anaesthetic, did not improve the quality of post-operative epidural analgesia.¹ But many other investigators found that addition of buprenorphine to bupivacaine is much more superior to local anaesthetic alone, for post-operative analgesia.²⁻⁴ Hence, the present study is designed to compare the duration of the post-operative epidural analgesia in bupivacaine alone and in combination with buprenorphine.

Material and methods

The study was conducted in the Department of Anaesthesiology, Regional Institute of Medical Sciences (RIMS), Imphal. After obtaining institutional ethics committee approval and written informed consent, 60 adult patients of ASA grade I and II, scheduled to undergo gynaecological and lower limb orthopedic surgery under spinal anaesthesia, in the age group 20 to 60 years, were selected and divided into 2 equal groups of 30 patients each. Preanaesthetic evaluation was done for all patients. Visual Analog Scale (VAS) consisting of 100 mm line with 0 = no pain and 100 = worst possible pain were explained to all the patients. Each patient was premedicated with ranitidine 50 mg IV, 2 hours prior to surgery and preloaded with 500ml of Ringer's lactate solution.

Under strict aseptic and antiseptic precaution, epidural catheterization (perifix, 401 LOR, B. Braun) was performed with loss of resistance technique at L₂₋₃ interspace in the midline leaving 3-4 cm of catheter length in the epidural space. Intradural migration and intravascular placement of the epidural catheter were ruled out by negative aspiration and a test dose of 3ml of 2% lidocaine with adrenaline (1:2 00 000). Dural puncture at a different space, L₃₋₄ interspace was also performed with the midline approach using a 24G Quincke needle and intrathecal injection of 3ml of 0.5% bupivacaine heavy was done in all the patients.

Two hours after spinal anaesthesia –

(i) In group A each patient was injected 8 ml of 0.25% bupivacaine mixed with 0.1 mg of buprenorphine over 20 minutes through prefixed epidural catheter.

(ii) In group B each patient was injected 8 ml of 0.25% bupivacaine over 20 minutes through prefixed epidural catheter.

After epidural injection heart rate, MAP, respiratory rate and SPO₂ were recorded at 2 minutes intervals for 1st 10 minutes, then at 5 minutes intervals upto 30 minutes followed by at 6 hours intervals upto 24 hours post-operatively.

First complaint of pain demanding analgesia in hours after epidural injection, time to first pain medication in hours after epidural injection and visual analog scale (VAS in mm) scoring at first pain medication were recorded. Post-operative side effects, if any were recorded.

Results

Statistically there was no significant difference between the two groups in patient's characteristics, physical status (ASA) and distribution in relation to surgery (table 1).

Pre-anaesthetic haemodynamic and

Table 1. Demographic profile in group A and group B

Patients' characteristic	Group A (n=30) (Mean ± SD)	Group B (n=30) (Mean ± SD)
Age (yrs)	40.63 ± 8.89	42.70 ± 8.15
Sex (M:F)	6:24	5:25
Body height (cm)	157.26 ± 4.65	157.23 ± 5.41
Body weight (kg)	54.26 ± 7.60	54.26 ± 7.30

respiratory variables were comparable in the two groups with no significant statistical difference ($p > 0.05$) (table 2).

Table 2. Pre-anaesthetic haemodynamic and respiratory variables of patients in group A and group B.

Haemodynamic & respiratory variable	Group A (Mean ± SD)	Group B (Mean ± SD)	t-value	P
Heart rate (per minute)	77.86 ± 9.50	78.63 ± 9.75	0.32	P > 0.10
Blood pressure (MAP in mmHg)	95.76 ± 6.26	95.56 ± 5.67	0.13	P > 0.10
Respiratory rate (breaths per minute)	20.96 ± 2.94	21.26 ± 2.92	0.40	P > 0.10
SPO ₂ (%)	97.96 ± 1.35	98.50 ± 1.13	1.74	P > 0.05

There was significant rise of mean MAP and heart rate in group A patients between 6 hours and 24 hours with peak rise at 12 hours and in group B patients between 30 minutes and 12 hours with peak rise at 6 hours. No significant difference was observed between the 2 groups at other time intervals in relation to MAP and heart rate (fig1).

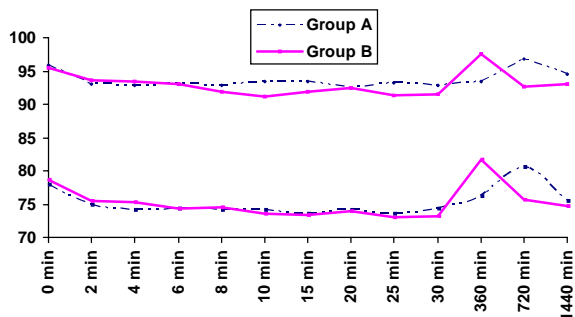


Fig 1. Mean changes in MAP (mmHg) and Heart Rate (per min) in group A and group B in the study period

There was no significant statistical difference between the 2 groups in any period of time in the study period in relation to mean respiratory rate ($p > 0.10$) and mean SPO₂ ($p > 0.05$).

The mean time of first complaint of pain at the operated site in group A and group B were 12.23 ± 1.78 hours and 4 ± 0.69 hours respectively after epidural injections, which is

highly significant ($p < 0.001$) (table 3).

Mean VAS scoring was 50 ± 7.42 mm in group A and 49 ± 6.07 mm in group B at first pain medication after epidural analgesia with no significant statistical difference ($p > 0.10$).

Table 3. Distribution of patients in group A and group B in relation to first complain of pain demanding analgesia against post-operative category of time.

Group	First complaint of pain demanding analgesia		Chi-square	P
	3-10 hr	10-16 hr		
A(n=30)	5	25	42.91	P<0.001
B(n=30)	30	0		

In group A, 3 patients had post-operative nausea and vomiting (PONV), 1 patient had prolonged post-operative sedation. In group B, 2 patients had PONV, 1 patient had urinary retention and 1 patient complained of burning micturition. No other side effect was found in any patient of either group. The difference between the 2 groups is statistically insignificant ($p > 0.50$) in relation to post-operative side effects.

Discussion

In the present study, duration of post-operative epidural analgesia in group A was much longer (12.23 ± 1.78 hours) than the duration of post-operative epidural analgesia in group B (4 ± 0.69 hours).

Gao F et al⁵ also found that the duration of analgesia was longer with caudal bupivacaine plus buprenorphine alone (mean 606 min) than with bupivacaine (mean 126 min) which is comparable to the present study.

Kamal RS and Khan FA³ found that caudal

post-operative analgesia with plain bupivacaine and buprenorphine combination was of longer duration (24 hours) than that of plain bupivacaine alone. However, the duration of analgesia in their study was much longer than the present study.

Loh SP⁶ found that the duration of post-operative analgesia by the combination of epidural buprenorphine and bupivacaine was ranged from 10 hrs to greater than 36 hrs, which is comparable to the present study.

However, the duration of analgesia in the combination group as reported by Loh SP⁶, Kamal RS and Khan FA³ were longer (10-36 hrs and 24 hrs respectively) than the present study (9-16 hrs). In the present study, we used 0.25% bupivacaine plus 0.1 mg buprenorphine in the combination group, which was lower than the dose used in the previous studies (bupivacaine 0.5% + buprenorphine 0.15 mg). This has resulted in shorter duration of analgesia in the combination group in comparison to the duration of analgesia provided by the combination group in our studies.

In the present study, rises in MAP and HR in group A at around 12 hrs and in group B at around 6 hrs after epidural analgesia, were due to return of pain sensation at the end of epidural analgesia and correspond to the mean duration of post-operative epidural analgesia in the respective groups.

Conclusion

Combination of epidural bupivacaine and buprenorphine provides longer duration of post-operative analgesia than epidural bupivacaine alone and is recommended in adult patients undergoing gynaecological and lower limb orthopaedic surgery.

References

1. Komatsu H, Matsumoto S, Nagasaki G, Hori M. Combination of buprenorphine and bupivacaine for post-operative pain relief using a portable 0.5ml. h⁻¹ type infuser with patient control module. Masui 1996; 45 (6): 735-40.
2. Hirabayashi Y, Fukuda H, Saitou K, Tamagawa K, Ikeno S, Furuya K, et al. Post-operative pain relief by continuous epidural infusion of bupivacaine and buprenorphine. Masui 1992; 41 (10): 1580-4.

3. Kamal RS, Khan FA. Caudal analgesia with buprenorphine for post-operative pain relief in children. *Paediatr. Anaesth.* 1995; 5 (2): 101-6.
4. Lehmann KA, Stern S, Breuker Kh. Obstetrical peridural anaesthesia with bupivacaine and buprenorphine. A randomized double-blind study in comparison with untreated controls. *Anaesthesist* 1992; 41 (7): 414-22.
5. Gao F, Waters B, Seager J, Dowling C, Vickers MD. Comparison of bupivacaine plus buprenorphine with bupivacaine alone by caudal blockade for post-operative pain relief after hip and knee arthroplasty. *Eur. J. Anaesthesiol.* 1995; 12 (5): 471-6.
6. Loh SP. Epidural buprenorphine and bupivacaine in major gynaecological operations. *Med. J. Malaysia* 1993; 48 (2): 207-10.

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Dr. L. Deban Singh,
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Atypical tubercular-meningoencephalitis - a case report

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A 30 yrs old female patient was admitted on 2/3/08 at Medicine Ward, Regional Institute of Medical Sciences(RIMS) Hospital Imphal with complaints of a) Dizziness off and on for 4 months, b) blurring of vision off and on for 3 months, c) depression for 3months, d) stiffness of the right upper and lower limbs for one and half months, e) weakness of right side of the body for one and half month, f) slurring of speech for one week, g) vomiting for 2 days, h) loss of consciousness for 1 day and i) urinary incontinence for 1day.

The patient had past history of headache lasting for one year in 2004. She had normal CT scan brain and headache improved with symptomatic management at that time. Since then there was no history of headache. There was no associated history of fever and tuberculosis. The menstrual history of the patient revealed that she had amenorrhea for 2 months. Personal history suggested that she was having normal non-vegetarian dietary habit. Treatment history of the patient expressed that she had been on anti-psychotic and anti-depressant treatment for one and half months. Haloperidol and metoclopramide injections were given intravenously one day before admission. At the time of examination the patient was afebrile,

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pulse rate was 78/min regular, blood pressure was 110/70mmHg and respiratory rate was 18/ min. On CNS examination the patient was found comatose, but responding to painful stimuli only. No signs of meningeal irritation (no neck stiffness, no Kernig's sign) were detected. Respiratory, cardiovascular and abdominal systemic examination findings were within normal limit.

The patient was first managed in the psychiatry ward, RIMS Hospital. She was transferred to Medicine ward of RIMS Hospital as the patient developed high fever. Next day, the patient was found to be drowsy, febrile, but meningeal signs were absent. One and half eye sign was present. Decorticate posture and right sided hypertonia were present clinically. The muscle powers of grade 3/5 (left) and grade 2/5(right) were present in both upper and lower limbs. Plantar response was extensor on right (Babinski's sign present) and mute on the left.

On investigation Hb- 9.6gm/dl, TLC-10,000/cumm, polymorph-78%, lymphocyte-20%, monocyte-1%, eosinophil-1%, ESR-45 mm/ 1st hr, urine RE, LFT and KFT reports were within normal limits. Blood sugar (random) was 172%. The fasting blood sugar and post-prandial blood sugar reports were normal. Retro virus status of the patient was negative. Clinically being suspicious of encephalitis, CSF examination was done with lumbar puncture which showed (i) protein: 203 mg%, (ii) sugar: 40 mg%, (iii) TLC 20/cumm (iv) DLC: Lymphocytes: 98%, Neutrophil 5%, (v) ADA:

16 U/L. Fundoscopy examination revealed normal study. On further investigation, MRI Brain showed flair hyperintense lesions involving right parieto-occipital lobe, left temporal lobe, left basal ganglia and other parts of brain stem suggestive of multiple infarction lesions. Small haemorrhagic spots were also seen in the ventral pontomedullary area and haemorrhagic infraction in the brain stem (fig.1,2 & 3). MRA and MRV screening reports were normal.



Fig1. MRI Brain T1 coronal view showing hyperintense lesion possibly bleeding.

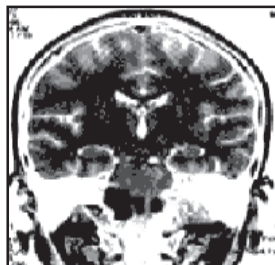


Fig 2: MRI Brain T2 coronal view showing hyperintense lesion possibly bleeding.



Fig 3. MRI Brain Saggital view showing feature of pre pontine hemorrhage.

The patient also had aspiration pneumonia and deep vein thrombosis. The aspiration pneumonia was managed with broadspectrum antibiotics and deep vein thrombosis was treated with enoxaparin injection and acenocoumarol 2 mg daily with elastic stockings. The patient was first treated empirically with intravenous Methyl prednisolone for 5 days as the patient presented with episodes of clinical features of multiple lesions in the brain and showed clinical improvement. After a few days the patient deteriorated clinically becoming drowsy and features of diffuse neurological deficit more obvious. Then antitubercular therapy (refampicin, isoniazide, ethambutol,

pyrazinamide and ofloxacin) with steroids was started empirically along with other supportive measures. Within 2 weeks the patient improved considerably and after 1 month the patient was discharged from the hospital satisfactorily.

Discussion

TB meningitis is associated with high morbidity and mortality. Infection is always secondary to haematogenous spread or in some cases local extension of tubercle bacilli from a focus of infection. A tuberculoma in the cerebral cortex spread by haematogenous route may enlarge and rupture with dissemination of Bacilli and development of meningitis. At least 75% of individuals have a tuberculous infection at least twelve months before admission for meningitis.¹ Neuro-tuberculosis is seen upto 10% of patients with systemic tuberculosis as a result of haematogenous spread from a primary focus, usually from the lung.^{2,3} Our patient is a case of tubercular meningoencephalitis presenting in an atypical picture. The first clinical presentation was similar to a case of psychiatric disorder with depression. Headache presenting with this particular case may mimic chronic daily headache or chronic migraine subtype of chronic daily headache.⁴⁻⁶ The MRI is a sensitive and non-invasive tool for diagnosis and localizing intra-medullary as well as brain tuberculomas. MRI will also delineate the extent of surrounding oedema, determine the stage of tuberculoma formation and central caseation (target sign)⁶ which is the evidence of tubercular meningitis involving brain stem. The third cranial nerve lesions can also be seen in tubucular meningitis.⁷

When the CSF result is not very suggestive of tubercular meningitis, polymerase chain reaction (for Mycobacterial protein) combined with contrast MRI can be used with a reasonable degree of certainty, without restarting to an invasive biopsy.⁸ MRA and MRV reports were normal in our patient. MRA and MRV studies which reveal vascular system may not reveal minor vasculitic lesions in certain cases for which DSA (digital subtraction angiography) is required to confirm the minor vasculitic lesions. DSA was

not done to confirm the vasculitis including TB arteritis in this patient since the facilities were not available in our centre.

Antitubercular therapy with anti-oedema measures is the mainstay of treatment of these patients.⁹ Even though unproven in patients with perilesional oedema short-term steroids may be helpful.¹⁰ Usually the conservative treatment should continue for one year, which can resolve the tuberculomas. In our patient, there was a transient period of hypertonia (bilateral) for few days as result of transient Parkinsonism (extrapyramidal lesion) and was treated with oral trihexy phenidyl hydrochloride and levodopa. Parkinsonism symptoms in tubercular meningitis can occur rarely.

The possibility of stiffness of right half of the body for one and half months due to the side effects of antipsychotic drugs treatment was ruled out by the corresponding diffuse haemorrhagic infarction lesions in the brain and

brainstem. In rare cases of meningoencephalitis, the clinical features of meningeal irritation may be absent and can only be revealed by CSF and MRI pictures. The clinical signs of bilateral pyramidal tract lesion present in both upper and lower limbs, the diffuse brain stem lesion revealed clinically by one and half eye sign (combined lesion of medial longitudinal fasciculus and parapontine reticular formation) and clinical features of basal ganglia lesions presented with transient parkinsonism (extrapyramidal lesion) features were all due to the diffuse tubercular arteritis occurring scatteringly in different parts of brain and brain stem.

In conclusion, this case emphasizes that tubercular meningoencephalitis can present in an atypical clinical picture simulating psychiatric disorder initially characterized by headache and depression followed by recurrent episodes of multiple diffuse neurological deficits mimic multiple sclerosis and afterwards presenting with clinical features of meningoencephalitis.

References

1. Souza RD, Franklin D, Simpson J, Kerr F. Atypical presentation of tubercular meningitis. *SMJ* 2002;47(1): 14-45.
2. Thacker MM, Puri AI. Concurrence intramedullar and intra-cranial tuberculomas. *J. Postgrad. Med* 2004;50:107-9.
3. Yen HL, Lee RJ, Lin JW, Chen HJ. Multiple tuberculomas in the brain and spinal cord: a care report spine 2003;28: E 499-502.
4. Silberstien S, Diener HC, Lipton R, Goadsby P, Dodick D, Busoone G et al. Epidemiology, risk factors and treatment of chronic migraine: a focus on topiramate headache. 2008 Jul; 48(7): 1087-95.
5. Apostol G, Cady RK, Lafoset GA, Robieson WZ, Abi-Saab WM, Saltarelli M. Divalproex extended release in adolescent migraine prophylaxis: results of a randomized, double-blind, placebo-controlled study. *Headache*. 2008 Jul; 48(7):1012-25.
6. Agarwal S, Magu S, Kamal K. Reversible white matter abnormalities in a patient with migraine. *Neurol India*. 2008 Apr-Jun; 56(2): 182-5.
7. Lin SK, Wu T, Wai YY. Intramedullary spinal tuberculomas during treatment of tuberculous meningitis. *Clin Neurol Neurosurg*. 1994; 96(1): 71-8.
8. Shen WC, Cheng TY, Lee SK, HOY J, Lee KR. Disseminated tuberculomas in spinal cord and brain demonstrated by MRI with Gadolinium – DTPA. *Neuroradiology* 1993;35:213-5.
9. Lamba PA, Bhalla JS, Mullick DN. Ocular manifestations of tubercular meningitis; a clinobiochemical study. *J. Pediatr Ophthalmol strabismus*. 1986 May-Jun; 23(3) : 123-5.
10. Kumar R, Jain R, Kaur A, Chhabra DK. Brainstem tuberculosis in children. *Br. J. Neurosurg* 2008;14:356-61.



Trisomy 18 Neonate with unusual phenotypic features– a case report

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A female preterm baby, small for gestational age (36 weeks gestation) was born by vaginal delivery to a 28 years old unbooked gravida 3 mother with Apgar Score of 5,7, and 8 at 1, 5 and 10 minutes respectively. There was no history of consanguinity in the parents. The eldest sibling 7 years old was healthy and the second pregnancy ended in a spontaneous abortion at 2 month 15 days of pregnancy. On physical examination at 24 hours of age, the weight was 1.5 kg, crown heel length was 40 cms, head circumference measured 29 cms and the calculated ponderal index was 2.3. The neonate had microcephaly with prominent occiput, micrognathia, extremely low set ears with posteriolly rotated pinna, short and webbed neck, short sternum with widely spaced nipples, flexed fingers with index finger overlapping the third and fifth finger overlapping the fourth and prominent heels (fig. 1 and 2). Clinical examinations of respiratory and cardiovascular system were normal. No organomegaly was found on abdominal examination. Neurological examination revealed poor suck and a weak cry.

Investigations revealed a normal hemogram. There was no radiological abnormality on chest radiography. Echocardiography demonstrated a ventricular septal defect.

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Fig 1. Photograph showing webbed neck, widely spaced nipples with short sternum & rocker bottom feet.



Fig 2. Photograph showing flexed fingers with index finger overlapping the third & the fifth finger overlapping the fourth.

Karyotyping (G-banding) revealed a female chromosomal pattern with Trisomy 18 → 47, XX + 18 (fig. 3). She was given parenteral fluids for the first 3 days and nasogastric feeds

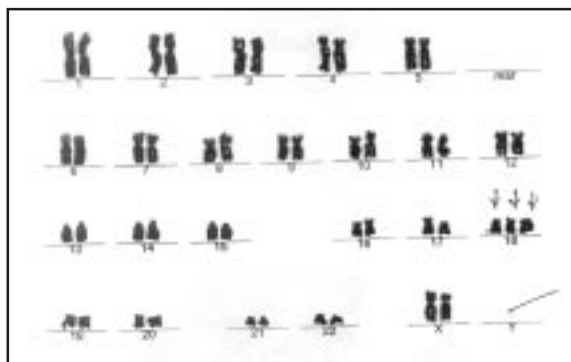


Fig.3 Chromosomal analysis of whole blood & Karyotyping(G-banding) revealed female chromosomal pattern – 47,XX, +18, with trisomy 18 in all the cells examined. This is consistent with Edward syndrome.

with expressed breast milk was introduced in small volume on the fourth day and gradually increased. She was administered prophylactic antibiotic as per our nursery protocol. On day 10, she developed fever (temp 102°F), tachyp-

noea, grunting and tachycardia. The baby was managed conservatively but did not show signs of clinical improvement and succumbed within one hour of onset of symptoms. Consent for post mortem examination was refused. After obtaining the cytogenetic report, genetic counselling was offered to the parents.

Discussion

Trisomy 18 (Edwards Syndrome) is the second most frequently encountered autosomal aneuploidy affecting about 1 in 6000 newborns.¹ The syndrome was described by Edwards and colleagues in 1960, on a female child with multiple congenital anomalies with an extrachromosome belonging to the E group on post mortem cytological examination.² A wide spectrum of clinical abnormalities have been described in Trisomy 18 of which, the characteristic clinical findings include low birth weight, microcephaly, prominent occiput, micrognathia, low set ears with malformed pinna, finger flexion deformity, rocker bottom feet and cardiovascular abnormalities.¹⁻³

Various structural and non structural abnormalities documented in Trisomy 18 is based on prenatal ultrasonography and post mortem findings on fetuses.³⁻⁷ In our case, the most striking feature in addition to the commonly described manifestations of Trisomy 18, a combination of webbed neck and widely spaced nipples. In spite of medline search, Trisomy 18 livebirths with the above combination has not been reported. On the other hand, nuchal translucency thickness measurement has been described as one of the most sensitive and important markers of Trisomy 18 on prenatal ultrasonography during second trimester of pregnancy. In the present case, there was considerable confusion in diagnosis because of many of the features which resembled the criteria of Turner syndrome including female sex, low birth

weight decreased length, low set ears, micrognathia, webbed neck and widely spaced nipples were present and the diagnosis was established only after karyotyping. In Trisomy 18, a maternal age related autosomal Trisomy, the median age of mothers' of Trisomy 18 is 32.8 years², although there are also a large number of age independent cases⁴. In the present report, the mother was 28 years old. Cytogenetics studies revealed that approximately 80% of patients were typical Trisomy for chromosome 18, mosaics constitute 10% and the rest are associated with double trisomies for another chromosome.⁴

Intrauterine mortality is very high in Trisomy 18 affected fetuses and about 2.5% of all Trisomy 18 conceptions survive to be liveborn. Of the livebirths, 90% of babies die by six months and 5% are alive at 1 year of age.³

Whenever ultrasonographic findings are consistent with Trisomy 18, prenatal karyotyping is to be undertaken and pregnancy termination considered in view of the very poor prognosis. The sensitivity of detecting fetal Trisomy 18 on prenatal sonography is variable⁴ although one study has reported a sensitivity of 100% in the hands of experienced sonologists⁵. A combination of prenatal maternal serum biochemistry markers and ultrasound scanning have been used to diagnose Trisomy 18 during first and second trimesters.⁴

As there is little information regarding the risk of recurrence, only genetic counselling is to be offered to those mothers with a livebirth Trisomy 18. The present case highlights that Trisomy 18 should also be included in the differential diagnosis of neonate with webbed neck and widely spaced nipples.

References

1. Descartes M, Carroll AJ. Cytogenetics. In : Behrman RE, Kliegman RM, Jenson HB, Stanton BF, Nelson textbook of pediatrics, 18th edition. Vol. I, Elsevier : Saunders; 2008.p. 502–17.
2. Hsia DY. Lethal and sub lethal malformations. In : Sorsby A (eds) Clinical Genetics, 2nd edition. Sussex: RJ Acford Ltd 1973; 47 – 57.
3. Tolmei JL. Down syndrome and other Autosomal Trisomies. In : Rimoin DL, Connor JM, Pyeritz RE, Korf BR (eds) Emery and Rimoin's

- Principles and practice of medical genetics, 4th edition. London:Churchill Livingstone Harcourt Publishers Ltd.; 2002.p.1129 – 83.
4. Nyberg DA, Jeanty P, Glass I. Syndromes and multiple anomaly conditions. In Nyberg DA, McGahen JP, Pretorius DH, Pihu G (eds) Diagnostic imaging of fetal anomalies Philadelphia: Lippincott Williams and Wilkins; 2003.p.133 – 220.
 5. Yeo L, Guzman ER, Day Salvatore D, Walters C, Chavez D, Vintzileos AM. Prenatal detection of fetal Trisomy 18 through abnormal sonographic features. J Ultrasound Med 2003; 22 (6) : 581 – 90 (quiz 591 – 2).
 6. Hepper PG, Shahidullah S. Trisomy 18 : behavioral and structural abnormalities. An ultrasonographic case study. Ultrasound Obstet. Gynecol. 1992; 2 (1) : 48 – 50.
 7. Tongsong T, Sirichotiyakul S, Wanapirak C, Chanprapaph P. Sonographic features of Trisomy 18 at midpregnancy. J Obstet Gynaecol Res. 2002; 28(5) : 245 – 50.



Airway management in temporomandibular joint ankylosis - a report on two cases

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Case 1

A 70-year old male patient presented to the Surgery out patient department, Regional Institute of Medical Sciences, Imphal with a history of inability to open the mouth following multiple sessions of surgical resection of a growth on the left side of the buccal cavity at a district hospital. He was posted for resection of the fibrosed area in the mouth followed by skin grafting under general anaesthesia. Preoperative assessment of the case revealed that the fibrosing growth produced a false ankylosis of the temporomandibular joint (TMJ) and the mouth opening was less than 2 mm (fig 1).



Fig 1. Picture showing mouth opening less than 2 mm.



Fig 2. ETT in situ after blind nasal intubation.

His neck mobility was normal and other physical and laboratory findings were within normal limits, and there was feasibility of ventilation with a face mask. On the day of the operation, he was premedicated with injection glycopyrrolate

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0.2mg intramuscularly and injection ranitidine hydrochloride 50mg intravenously 45 minutes before the scheduled time. Blind nasal intubation under inhalational anaesthesia with sedation was selected as the technique for securing the airway. The nasal mucosa was prepared with lignocaine and xylometazoline nasal drops. After administering propofol 1% at 1mg/kg intermittent bolus doses for sedation, blind nasal intubation was attempted under spontaneous respiration. The lubricated ETT was gently advanced along the floor of the nose, orienting the tube to aim at the larynx after putting the patient's head in the sniffing position along with cricoid pressure. Then, it was incrementally advanced during inspiration until the breath sounds conducted through the ETT become maximal after which the tube was passed beyond the vocal cords and the patient coughed (fig 2).

After confirmation of the proper placement of the ETT by auscultation and capnography, the surgical procedure was carried out under controlled ventilation with intermittent positive pressure ventilation without any intraoperative or post-operative complication.

Case 2

A 53-year old male patient attended the Surgery out patient department, Regional Institute of Medical Sciences, Imphal with a growth on the right side of the buccal cavity. Previously, the patient underwent multiple sessions of radiotherapy and was considered for surgical resection of the growth under general anaesthesia.

Preoperative assessment of the case revealed that the mouth opening was less than 2mm with the fibrosing growth restricting the TMJ mobility producing a false TMJ ankylosis (fig 3); his neck mobility was normal and other findings were within normal limits.



Fig 3. Picture showing mouth opening less than 2 mm.

Preoperatively, the patient was premedicated with injection glycopyrrolate 0.2mg intramuscularly and injection ranitidine hydrochloride 50mg intravenously 45 minutes before the scheduled time. Blind nasal intubation was selected as the technique for securing the airway. After preparing both the nares with xylometazoline (0.1%) nasal drops, it was packed with cotton gauge soaked with 4% lignocaine for 2-3 minutes after which blind nasal intubation was attempted under spontaneous respiration. However, blind nasal intubation was abandoned after two failed attempts, and it was switched over to retrograde intubation. The skin over the cricothyroid membrane was infiltrated with 0.5% lignocaine. Then, the larynx was stabilized between the thumb and the index finger of one hand while the cricothyroid membrane was punctured with the catheter over needle assembly (Jelco 18G) held at 45° pointing cephalad. After confirmation of tracheal placement of the i.v catheter by aspiration of free air, the catheter over the needle assembly was pushed further at an obtuse angle closer to the axis of the larynx. The needle stylet was withdrawn and after instillation of 3ml of 4% lignocaine into the lumen of the larynx, a 'J'- tipped guide wire was inserted through the IV catheter and advanced cephalad into the oro-nasopharynx.

The guide wire was then retrieved from the nostril; and it was inserted into a well lubricated 7.5mm ETT (endotracheal tube) after making it taut by pulling at both ends (fig 4 & 5). The patient was asked to protrude his tongue to elevate the epiglottis and provide an easy access of the ETT into the glottis. Then, the surgical procedure was carried out under

general anaesthesia with controlled ventilation without any intra-operative or postoperative untoward incidence.

During the post operative period, both the patients were put in the intensive care unit for weaning on T-piece and extubated the next day without any untoward events or complications.



Fig 4. Guide wire and cannula in situ.



Fig 5. Showing ETT rail-loaded over the guide wire.

Discussion

In general anaesthesia, endotracheal intubation forms a major, significant and essential event with respect to maintenance of the airway of the patient, throughout the anaesthetic procedure. Interestingly, intubation of trachea is considered a simple procedure for anaesthesiologists.¹ However, there are many conditions which can make endotracheal intubation extremely difficult and TMJ ankylosis is one such condition where intubation poses a great challenge to the anaesthesiologists. Thus, when exposure of the vocal cords is difficult due to anatomical-congenital or acquired cases, many procedures have been used.² Some of the techniques include laryngeal mask airway, intubating laryngeal mask airway, McCoy laryngoscope, bougie, cricothyrotomy, blind nasal intubation, retrograde intubation, fiberoptic laryngoscope etc.

In TMJ ankylosis, the technique of blind nasal intubation was traditionally recommended.³ However, it may fail and repeated attempts may injure the involved structures resulting in complications like bleeding, airway obstruction, etc. The technique has been long recognized as relatively easy in spontaneously breathing patients and success rates from 72% to 86% have been reported.^{4, 5} However, in the presence of bleeding and frequently associated airway anomaly, failure of blind

nasal intubation is not uncommon.⁶ Interestingly, in our first case, no such untoward incident occurred and blind nasal intubation was carried out smoothly.

On the other hand, originally described in 1960, the technique of retrograde intubation is a two stage procedure which includes retrograde passage of a catheter or a guide wire from the larynx to mouth or nose followed by railroading an ETT over the guide wire or catheter.⁶ Since then several modifications have been reported. A number of technical and procedural problems may arise using this method. These may include catheter selection, site of puncture, route of advancement of tracheal tube over catheter (nasal or oral) and attendant complications.⁷ As observed in our second case, the use of a single flexible 'J' tipped guide wire for retrograde intubation makes it a one step

procedure, since it negotiates the bends and curves in the airway without much difficulty. However, Dhulkhed VK⁸ advocated the use of second guide wire and found it advantageous for retracting the epiglottis to make the passage of the endotracheal tube into the larynx easier.

Conclusion

Fibreoptic endoscope-assisted intubation is the first choice in such cases of difficult intubation; but in centres, where fibreoptic endoscopes are not available for routine use, the cases may still be effectively managed with the techniques like retrograde intubation or blind nasal intubation, etc. It is worth mentioning here that using such techniques should be with utmost precautions as unwanted complications like bleeding, infection, injury to respiratory tract, etc. may occur.

References

1. Lee YW, Lee YS, Kim JR. Retrograde tracheal intubation. *Yonsei Medical Journal* 1987; 28(3):228-30.
2. Salem MR, Mathrubhutham M, Bennett EJ. Current concepts: difficult intubation. *N Engl J Med*. 1976; 295:879-81.
3. Williamson R. The airway decides the anaesthetic approach before tracheal intubation. *Br J Anaesth* 1993; 70:601.
4. O'Brian DJ, Danzl DF, Hooker EA, Daniel LM, Dolan MC. Prehospital blind nasotracheal intubation by paramedics. *Ann Emerg Med* 1989; 18: 612-7.
5. O'Connor RE, Megargel RE, Schnyder ME, Madden JF, Bitner M, Ross R. Paramedic success rate for blind nasotracheal intubation is improved with the use of an endotracheal tube with directional tip control. *Ann Emerg Med* 2000; 36: 328-32.
6. Butler FS, Cirillo AA. Retrograde tracheal intubation. *Anaesth Analg* 1960; 39: 333-8.
7. Arya VK, Dutta A, Chari P. Difficult retrograde endotracheal intubation: the utility of a pharyngeal loop. *Anesth Analg* 2002; 94: 470-3.
8. Dhulkhed VK. Retrograde intubation in temporomandibular joint ankylosis-a double guide wire technique. *Indian Journal of Anaesthesia* 2008; 52(1):90-2.



Struma ovarii - a case report

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A 50 year old female presented with 24 weeks size lump was admitted to gynaecology department of Regional Institute of Medical Sciences (RIMS) Imphal on 29-9-2004. The lump was firm, mobile and non tender. It was associated with ascitis. and mild pain abdomen for the last one year. She had a small lump in the thyroid which was diagnosed as thyroid goiter with focal hyperplastic activity. Ultrasound thyroid and thyroid profile remained normal. Chest radiology was normal. Ultrasound lower abdomen revealed an adnexal mass with multiple solid areas, and ascitis.

She underwent total adominal hysterectomy and bilateral salpingo-oophorectomy. There was no feature of matastasis to omentum and lymph nodes. Ascitic fluid was not haemorrhagic.

Grossly the left ovarian mass was 12.5x8x9 cm in size, brownish, with smooth glistening surface. Cut section showed multiloculated cystic spaces filled with brownish gelatinous fluid, whitish solid areas and thick yellowish areas. Cytoanalysis

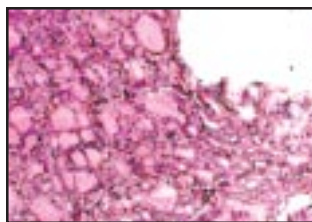


Fig 1. Photomicrograph of ovary showing colloid filled thyroid follicles, H&E, X 10.(Stroma ovarii).

of the ascitic fluid found no malignant cells. Histopathology revealed a benign struma ovarii (Fig 1). Postoperative thyroid function was normal. During two years of follow up patient was found healthy.

Discussion

Struma ovarii is a rare monodermal ovarian teratoma composed predominantly of mature thyroid tissue. Of these cases, 5-8% are clinically hyperthyroid and 5-10 % of these tumor are malignant.¹ Malignant struma ovarii is rare and makes up 0.1- 0.3 % of all ovarian teratomas.

The usual age of presentation of struma ovarii is 51-60 years.² In our case the chief complain was abdominal distension which is similar to the cases reported by Kumar V et al². These authors opined that the thyroid tissue in a teratoma may show pathological changes as seen in normally placed thyroid including diffuse a nodular hyperplasia thyroiditis, carcinoma and malignant lymphoma. Therefore a careful histopathological examination of such case is necessary.

Preoperative diagnosis is difficult because this rare tumour has no differentiating signs or symptoms. Occasionally, pre-operative scintigraphy of the pelvis may be helpful.³ Criteria of malignancy such as microscopic cellularity, cellular pleomorphism and mitotic activity are accepted universally.⁴

Pardo-Mindam FJ and Vazquez JJ⁵ have emphasised that the malignant tumour must show

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capsular and / or vascular invasion or metastasis or both and morphology of the tumour must resemble the follicular or papillary type. Very infrequently peritoneal implants of benign thyroid tissue, termed "strumosis" occur in struma ovarii and should not be confused with malignancy.

Because of its rarity, there is no consensus about its diagnosis and management in the literature.⁶ Surgical management is the better

treatment for struma ovarii which also represents a preventive measure for possible future malignant transformation.

Although the vast majority of struma ovarii are benign, they can present mimicking malignancy. Surgery is treatment of choice. Extent of surgery depends on the age of the patient and her desire to preserve fertility. Only histopathology can diagnose the disease.

References

1. Hatami M, Breining D, Owers RL, Del Priore G, Goldberg GL. Malignant struma ovarii - a case report and review of the literature. *Gynecol Obstet Invest.* 2008 ; 65 (2) : 104-7.
2. Kumar V, Gupta N, Srinivasan R, Nihawan R, Rajwanshi A. Struma ovarii-A report of seven Cases. *J obstet Gynecol India.* 2007 Jul-Aug ; 57 (4) : 350-1.
3. Joja I, Asakawa T, Mitsumori A, Nakagawa T, Hiraki Y, Kudo T et al. Struma ovarii : appearance on MR image. *Abdom Imaging* 1998 ; 23:652-6.
4. Kempers RD, Dockerty MB, Hoftmann DL. Struma Ovarii : ascitic, hyperthyroid and asymptomatic syndromes. *Ann Intern Med* 1970 ; 72:883-93.
5. Pardo-Mindam FJ, Vazquez JJ. Malignant struma ovarii : light and electron microscope study. *Cancer* 1983 ; 51:337-43.
6. Doganay M, Gungor T, Cavkaytar S, Sirvan L, Mollamahmutoglu L. Malignant struma ovarii with a focus of papillary thyroid cancer ; a case report. *Arch Gynecol Obstet.* 2008 Apr ; 277 (4) : 371-3.



Treatment of pancreatic fistula with octreotide (somatostatin analogue sms 201-995)

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CASE I

The patient was 40 years old man who had sustained abdominal injury by a buffalo horn on 12-12-2001. There was an abrasion over the left hypochondrium just 2 cm lateral to the midline. Abdominal paracentesis confirmed haemoperitonium. On palpation a gap was felt over the site of abrasion because of muscle rupture. On laparotomy, there was a longitudinal laceration at the body of the pancreas along with tear in the omentum. There was associated bleeding from the site of laceration but injury to the major duct was not present (Lucas grade II). The lacerated injury was closed with atraumatic silk 2-0. The abdomen was closed after putting a malecot's drain in morrison's pouch. Postoperatively the patient developed pancreatic fistula. The amylase study of which revealed >1000 IU/l in the drainage fluid. The patient recovered well from surgery except for the pancreatic fistula. He was discharged on 10th January 2002 with the expectation of spontaneous closure. However the patient reported back on 25/02/2002 with the same problem with drainage fluid more than 500-600 ml everyday. On 27th/02/2002 we started therapy with injection octreotide 50 microgram in normal saline slow intravenously 8 hourly followed by 50 micrograms subcutaneously 12hourly. Tthe

drainage fluid suddenly dropped next day and stopped completely in 5 days. The drain was removed on 07/03/2002. No side effect was observed during the treatment.

CASE II

Another patient, 32 years old male had two stab wounds at the abdomen, one at the umbilical area with omentum prolapsed and another at the left iliac fossa with intestine prolapsed, along with multiple incised wounds at both arms and left anterior chest wall. The patient was resuscitated properly. Six units of compatible blood were transfused during the entire period of treatment. At laparotomy, on 11/07/2003, there were perforating injuries at both anterior and posterior wall of the body of stomach near the greater omentum, incised wound at the anterior surface of body of pancreas (Lucas grade II), perforation of the jejunum 4 feet distal to the duodeno-jejunal junction and haematoma of the lesser sac. The perforation of stomach and jejunum were repaired in two layers.

The incised wound of the pancreas was repaired with atraumatic silk 2-0. The peritoneum was toiled thoroughly and a malecot's drain was put. Postoperatively, patient developed pancreatic fistula with drainage fluid reaching up to 500 ml. On 16/07/2003, we started Injection octreotide 50 microgram in normal saline intravenously 8 hourly followed by 50 microgram subcutaneously 12 hourly till 22/07/2003. Tthe drainage fluid was found to be reduced next day. The discharge fluid was completely

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stopped in four days. Drain was removed on 22/07/2003 and the patient was discharged fit on 23/07/2003.

Discussion

Pancreatic injury is uncommon, occurring in approximately 10% of abdominal injuries and it seldom occurs in isolation because of its anatomical position.¹ About one third of pancreatic injury developed pancreatic fistula.² Pancreatic fistula is a cause of concern to the surgeons because of its mortality and morbidity. It is traditionally treated either by surgery or conservative management which consists of antibiotics, I.V. fluids including total parenteral nutrition.

With the discovery of somatostatin, a naturally occurring tetradecapeptide with antisecretory property in the gastrointestinal tract in the early 1970s and its first clinical use in 1979 by Klempa in pancreatic surgery,³ another armamentarium was added to the management of pancreatic surgery. The short acting somatostatin (half life 1 – 3 minutes) leads to the search of another long acting analogue, octreotide. Octreotide has a half life of 90 to 120 minutes which is convenient for clinical use. Large clinical trials over the controversy of use of octreotide and its benefits in the prevention and management of pancreatic fistula have been undertaken.

In our two cases, the pancreatic injuries were categorized as Lucas II grade. Pancreatic repairs were complicated by development of fistula. We used intravenous octreotide in the

postoperative period only.

Bassi C et al³ advocated the routine use of octreotide in pancreatic surgery. Leandros E et al⁴ studied with controls and found a better rate of spontaneous closure of pancreatic fistula in 65% whereas only 27% in the control group. Boike GM et al⁵ found that administration of octreotide in enterocutaneous pancreatic fistula resulted in the rapid and significant reduction of fistula output within twenty-four hours of treatment and closure of fistula in two days.

Our cases I and II showed reduction in the amount of fluid discharge in the next day and fistula closure in five and four days of octreotide administration respectively. Alghamdi AA et al,⁶ after a systemic review, concluded that pre-operative and post-operative use of octreotide was associated with a significant reduction of fistula output even though the mortality rate was not changed.

In our cases, we found good response to octreotide administration as there was marked reduction of fistula output associated with rapid closure of the fistula. In addition to octreotide administration, other factors like the patients' general good health, young age, less devitalized pancreas in stabbed or penetrating wounds unlike in tumours, have the advantage of rapid reduction in fistula discharge and closure of the fistula tract. Octreotide has a role to play in the management of pancreatic fistula.

References

1. Madiba TE, Mokoena TR. Favourable prognosis after surgical drainage of gun shot, stab and blunt trauma of pancreas. *BJS* 1995; 82:1236-9.
2. Lucas CE. Diagnosis and treatment of pancreatic and duodenal injury. *Surg Clin North Am* 1977; 57:49-65
3. Bassi C, Falconi M, Peerzoli P. Role of somatostatin and its analogues in the treatment of gastrointestinal diseases: prevention after pancreatic surgery. *Gut* 1994; supplementary 3:S20-2.
4. Leandros E, Antonakis PT, Albanopoulos K, Dervenis C, Konstadoulakis MM. Somatostatin vs octreotide in the treatment of patients with gastrointestinal & pancreatic fistula. *Can J Gastroenterol* 2004 May; 18(5):303-6
5. Boike GM, Sightler SF, Averette HE. Treatment of small intestinal fistula with octreotide, a somatostatin analog. *J Surg Oncol* 1992; 49:3-5.
6. Alghamdi AA, Jawas AM, Hart RS. Use of octreotide for the prevention of pancreatic fistula after elective pancreatic surgery: a systemic review and meta analysis. *Can J Surg*. 2007Dec; 50(6):459-66.



Spontaneous uterine rupture in a primigravida - a case report

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Mrs. LD, a 38 years old, Gravida 1, Para 0 ($G_1P_{0+0+0+0}$) 39 weeks of pregnancy presented at the Department of Obstetrics and Gynaecology, Regional Institute of Medical Sciences, Imphal, at 10.15 PM. on 1st September, 2005 with complaints of pain abdomen and difficulty in breathing. The pain started suddenly in the morning around 6.00AM while she was walking. Then the pain increases without any relieving factor. There was no history of mismanaged labour or any history of trauma. There was no history of bleeding or leaking per vaginam. There was no significant medical and surgical history in the past.

On examination, she was conscious, pulse rate was 130/minute, feeble with systolic blood pressure of 70mmHg, markedly pale, with severe abdominal distension. Chest and cardiovascular systems were normal. Per abdominal examination revealed full term uterine size with foetus in breech presentation, two to three mild uterine contractions each lasting for 25-30 seconds every 10 minutes with intact uterine contour. Guarding of abdominal muscle and generalized uterine tenderness was present. Fetal heart sound could not be localized by stethoscope. On per vaginal examination, the cervix was 1.5 cm dilated, 50% effaced, soft and central in

position with tense bag of membrane. Buttock was at -2 station and there was no evidence of foeto-pelvic disproportion. Catheterization was done and clear urine was obtained. Her haemoglobin was 6 gm%, urine had no sugar or albumin and blood group was O Rh positive. Blood sugar, serum urea and electrolytes were within normal limits. Abdominal paracentesis revealed haemoperitoneum. Hence clinical diagnosis of uterine rupture with intra uterine fetal death was made. After initial resuscitation with intravenous fluids and blood she was shifted to operation theatre at 11.00PM, where emergency laparotomy was performed under general anaesthesia. Intraoperatively 3 litres of fresh haemoperitoneum was present. There was a 5mm size rupture on the posterior body left side 2 cm below the origin of round ligament. A tortuous vessel (?aberrant) was seen nearby. There was rapid oozing of blood. A fresh still born male baby weighing 3 Kg was delivered at 11.30PM by lower transverse uterine incision. Placenta and membrane were not remarkable and there was no retroplacental clots. There were two palpable intramural fibroids, one at the fundus about 5 cm in diameter and another of 2 cm diameter at the left lateral angle of the uterine incision. Uterus was closed in two layers. Repair of the rupture was done by using vicryl 1-0 suture materials. The rest of the uterus, both tubes and ovaries were normal. No other concomitant lesions were found inside the abdominal cavity. She received four units of whole blood intra operatively and two units in the post operative period. She recovered well and her post operative periods were uneventful.

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Discussion

Uterine rupture is a catastrophic obstetrics complication. Spontaneous uterine rupture is a life threatening event and the diagnosis is difficult in an unscarred uterus.^{1,2} Likewise our case was a life threatening one. The main risk factors of uterine rupture are uterine scars due to previous Caesarean section, myomectomy, hysterotomy, invasive placentation, manual removal of placenta and trauma due to external and internal podalic versions. Uterine rupture in primigravid women is a very rare event. Traditionally, the primigravid uterus has been considered almost immune to spontaneous rupture. Walsh CA et al³ reported a case of ruptured uterus in a primigravida of 33 years old at term pregnancy who presented with severe abdominal pain and signs of haemodynamic instability which were similar to our findings. They performed the exploratory laparotomy and the ruptured uterine wall was repaired in two layers. Somewhat related case was described by Fakhoury GW et al⁴ where a primigravida presented at 32 weeks of gestation following

trivial abdominal trauma with features of haemoperitoneum and shock. Repairment of uterus should only be performed when the rupture is simple and is in the lower uterine segment with no signs of infection.⁵ Interestingly, in our case the rupture was small and was in the lower segment. So, successful repairment could be performed. But unfortunately, foetus could not be saved because of the late reporting of the patient. Hence, prompt recognition of uterine rupture and expeditious recourse to laparotomy are critical in influencing both perinatal and maternal morbidity and mortality.

This case is being reported for its rarity. It is remarkable for a number of reasons. Firstly, she is a primigravida. Secondly, she has no history of dilatation and curettage and no history of mismanaged labour or trauma. Thus it can be concluded that although uterine rupture occurs more commonly in the multiparous populations, it cannot be assumed that the primigravid uterus is immune to rupture.

References

1. Sakr R, Berkane N, Barranger E, Dubernard G, Darai E, Uzan S. Unscarred uterine rupture-case report and literature review. *Clin Exp Obstet Gynecol.* 2007; 34(3):190-2.
2. Musa J, Misauno M. Uterine rupture in a primigravidae presenting as an acute abdomen post delivery; a case report. *Niger J Med.* 2007 Jul-Sep; 16(3):274-6.
3. Walsh CA, O' Sullivan RJ, Foley ME. Unexplained prelabour uterine rupture in a term primigravidae. *Obstet Gynecol.* 2007Feb; 109(2):455.
4. Fakhoury GW, Das E. Continuation of pregnancy and vaginal delivery following incomplete uterine tear during pregnancy: Case Report. *Br J Obstet Gynecol* 1984; 91:299-300.
5. Vedat A, Hasan B, Ismail A. Rupture of the uterus in labour: a review of 150 cases. *Isr J Med Sci.* 1993 Oct; 29(10):639-43.



Disseminated Langerhans' Cell Histiocytosis(LCH) – a case report

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A 16 - months old male presented with multiple swellings in the neck, axilla and right temple for 2 weeks. Concurrently there was another swelling over the left shoulder blade accompanied by red rashes on the trunk (fig. 1). Abdomen was also distended causing mild discomfort to the child. There were accompanying fever and off and on diarrhea but no appreciable weight loss. The child had a normal milestone of growth and development. Family and birth histories were non-contributory. There was no congenital abnormality. General physical examination revealed bilateral multiple cervical lymphadenopathy of 0.5 - 2 cm. in sizes, firm and mildly tender; a non healing operative scar on right temporal scalp with serous discharge, and a soft boggy swelling over left scapula.



Fig 1. Erythematous rashes on the trunk and left scapular swelling.



Fig 2. Hepato splenomegaly and abdominal erythematous rashes.

There were erythematous maculo-papular rash all over the trunk. Per abdominal examination revealed moderately enlarged hepato-splenomegaly (fig. 2) otherwise all other systems were normal. One month back the patient underwent an excisional biopsy of temporal scalp swelling at a private clinic which was reported as eosinophilic granuloma. Investigation showed Hb - 9.8 gm/dl, TLC - 8000cells/cu mm, DLC - P_{60}, L_{35}, E_2, M_3 , Platelet - 1.69 lacs, ESR - 34 mm1st hr; GBP - mild hypochromic RBCs, otherwise no abnormal detected. BT, CT, PT, LFT,KFT and urine RE were within normal limit. On skull X-ray, there was right temporal erosion with overlying soft tissue swelling. X-ray chest showed left scapular soft tissue swelling with irregularly resorbed bone(fig 3a). CT scan brain showed soft tissue swelling over right temporal bone and osteolytic lesion in right temporal and zygoma of skull(fig 3b). Brain parenchyma was normal. USG of abdomen

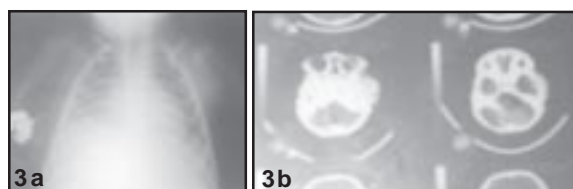


Fig 3a & 3b. Chest X-ray and CT scan skull showing bony lesions.

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revealed moderately enlarged hepato-splenomegaly with no focal lesions detected. FNAC of cervical LN shows reactive lymphadenitis. FNAC of scapular swelling findings were suggestive of Histiocytosis X (fig 4a & 4b). Bone marrow examination was not done. A final diagnosis of disseminated

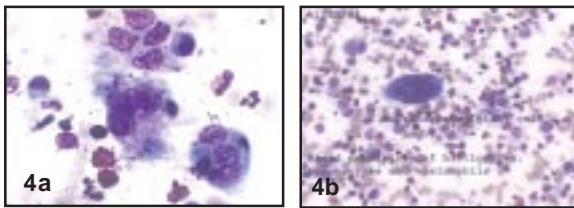


Fig 4a & 4b. Microphotographs showing mixed populations of histiocytes, lymphocytes, Eosinophils and multinucleated giant cells.

Langerhans' cell histiocytosis was made.

Systemic chemotherapy with Inj. Vinblastine 3mg. IV weekly(as per BSA). was given. Treatment was planned to be given for 6 cycles followed by maintenance with 6-MP and steroid with vinblastine at 3 wkly intervals for 18 weeks more. The chemotherapy was well tolerated. Patient defaulted after 3 cycles. However, the patient reported after default(after 6 months) and upon subsequent follow up was clinically and radiologically disease free (complete response) at 26 months (fig 5a & 5b).



Fig 5a & 5b. Photographs and showing healed lesions after treatment.

Discussion

LCH is a very rare disease with a reported incidence of 5 per million.¹ Affecting any age group without sex predilection, it is a clonal disorder of Langerhans' dendritic cells, associated with polymorphic cellular infiltration and damage at either unifocal sites or in multiple organs and tissues.² Historically, localized type is known as Eosinophilic granuloma, and clinical variants of multisystem disease as Histiocytosis X, Hand-Schüller-Christian disease, Letterer-Siwe disease. Histologically, LCH contains a mixture of Langerhans' cells that express surface antigen CD_{1a} and cytoplasmic S-100, in a

background of eosinophils, neutrophils and lymphocytes.¹ Demonstration of Birbeck granules by electron microscopy is diagnostic.³ Some contain central necrosis and in long standing cases fibrosis with foamy macrophages. Several cytokines produced by the LCH are responsible for the pathological damage. In fact there is a cytokine storm mainly produced from the T-cells. Although the clonality suggests a neoplastic disorder with varied biological behavior, it doesn't necessarily indicate a malignant process.⁴ The case reported here is a multisystem or disseminated form of LCH. Usually this form has less favorable prognosis than the localized form. Treatment also requires multiagent aggressive chemotherapy to be supplanted by radiotherapy whenever necessary, especially when there are brain and bony involvements.⁵ Single site or multiple sites within the same tissue can be treated by surgical resection or bony curettage and local irradiation with TD as low as 4 to 8 Gys. Disease free survival with local LCH is >95% but recurrence is common and hence close monitoring is required. With multisystem LCH response to therapy occurs in 60 to 90%, but multiple organ involvement with dysfunction the overall response rate and survival decrease to 40 to 66%. Initial suboptimal therapy in multiple visceral involvement is a major cause of concern for recurrence and treatment failure. Our case has been treated suboptimally, that is going by the guidelines of International Histiocytosis Society(HIS)⁶. However, LCH is also known for spontaneous regression even without treatment and sometimes with minimal treatment many are cured.^{4,5} Although our patient refused further treatment for 6 months, a close monitoring(clinical examination and imaging) was done to detect early relapse. Overtreatment with multiagent therapy in pediatric age group is also fraught with danger since there is risk of inducing long term sequelae like secondary malignancies. Thus the disease remains an enigma both in terms of origin, pathogenesis, natural course and treatment outcome. The HIS⁶ gives only a broad guideline of management but treatment should be individualized based on the merit of each case.

References

1. Kenneth L. McClain. Histiocytic Disorders. Cancer treatment. C. M. Haskell(Ed.) 5th Edit. Philadelphia : W. B. Saunders; 2001.p.1236-41.
2. Egeler RM. LCH:The symptoms, diagnosis and treatment. The Netherlands current time. 2004; 03:42-26.
3. Birbeck MS, Breathnach AS, Everall JD. An electron microscopic study of basal melanocyte and high level clear cell (Langerhans cell) in vitiligo. J invest dermaol 1961;37:51-67.
4. Disorders of histiocytes and dendritic cells. ASH-SAP: American society of hematology. Michael E. Williams et al, editors 2nd edit.New York: Blackwell publishing. 2005.p.140-44.
5. Gadner H. Heitger A, Grois N, et al. Treatment strategy for disseminated Langerhans cell histiocytosis. Med. Pediatr Oncol 1994;23:72-80.
6. The Histiocytic Society – at <http://www.HistiocyteSociety.org>.



Sclerosing stromal tumor of ovary - a case report

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A 22 year-old women, gravida 0, para 0, who had irregular heavy menstrual bleeding for 2 months with lower abdominal pain was admitted in department of gynecology on 15th march 2008. She had been married for 1 year. Physical examination showed marked pallor, with BP of 100/70mmHg, PR 92/min. Respiratory and cardiovascular systems were within normal limits. Bimanual pelvic examination showed anteverted normal sized uterus with solid, non-tender mass over the right adnexal area.

Blood examination showed Hb% of 6.6gm%, TLC was 8080 cells/cumm, DLC showed N_{63} L_{30} E_3 B_2 , and ESR was 32 mm/1st hour. KFT, LFT and Urine R/E were within normal limits. Serum markers of alpha-fetoprotein, beta-human chorionic gonadotropin, CA-125 were also within reference range. Ultrasonography showed a well defined solid mass measuring 6cm x 5cm over the right adnexa with hyperechoic shadow. Patient underwent laparotomy after 3 units of blood transfusion. A smooth white glistening tumor of 6 cm x 5cm x 4cm was enucleated from right ovary. The uterus, the left ovary and both fallopian tubes appear normal. There

were no other visible tumors in the peritoneal cavity.

Gross examination showed a soft white, solid tumor with smooth outer surface with scattered



Fig 1. Gross specimen showing smooth, glistening white and solid sclerosing stromal tumor of ovary.

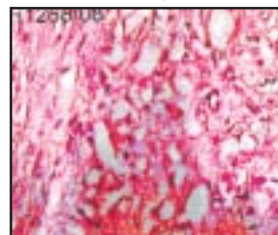


Fig 2. Sclerosing stromal tumor showing admixture of fibroblasts admixed with epithelioid cells and

yellowish nodular areas on cut surface (fig1). Microscopic examination shows paucicellular and hypercellular areas in a distinct lobular architecture and contain admixture of benign spindle and epithelioid cells with pleomorphic nuclei and vacuolated cytoplasm. Stromal collagen of the cellular areas encase prominent thin-walled branching capillaries and depicts hemangiopericytoma like pattern (fig 2). No laying down collagen with abnormal mitosis, entrapped capillaries areas of haemorrhage and necrosis are seen in any of the sections studied.

The overall morphological features are fairly consistent with sclerosing stromal tumour of ovary.

After an uneventful post-operative recovery, patient developed normal menstruation within 1month.

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Discussion

Sclerosing stromal tumor (SST) of the ovary is a rare ovarian disease with prevalence of 1.5% to 6% of ovarian stromal tumors.¹ It was first delineated as a distinct entity in the ovarian sex cord stromal tumors in 1973 by Chalvardjian and Scully.² Till date on Medline search only about 100 cases had been reported in world literature. In India too, few cases have been reported.^{3,4} It occurs predominantly in young women.³ More than 80% of SSTs occur in patients below the age of 30 years.^{2,5} Tumor size varies from 1.5cm to 20cm in diameter.⁶ The typical clinical presentation is with menstrual irregularities or pelvic pain. Most tumors are nonfunctional² but occasional patients present with symptoms caused by hormones secreted from their tumor.^{7,8} Sonographic findings of SST showed a well defined solid mass with hyper echoic honeycomb structures in our patient, which are also the characteristics of mixed heterogenous tumor without focal calcification.⁹ Histologically, SST is characterized by cellular heterogeneity,

prominent vasculature and pseudolobular appearance composed of both cellular and hypocellular area.¹⁰ The cellular areas of the tumor tend to undergo collagenous sclerosis and so is called as sclerosing stromal tumor.¹ Infertility and endometrial hyperplasia concomittent with SST have also been described which may indicate excessive hormone production.⁵ In several patients with irregular menses,⁸ there was normal menses following excision of the tumor was noticed. In our patient too, no clinical virilization and her menstruation returned to normal cycle after surgery. Complete excision cures the tumor.¹⁰ Although many patients presented with irregular menstrual abnormalities, only a few had documented biochemical evidence of hormone production of the tumor.¹ SST is a rare solid tumor and because of its solid nature, malignancy is highly suspected in young women with menstrual irregularity and pelvic mass. In conclusion, before embarking on any radical surgery, one should be cautious as enucleation alone of such tumor cure the disease.

References

- Peng HH, Chang TC, Hsueh S. Sclerosing stromal tumor of ovary. *Chang Gung Med J* 2003; 26: 444-8.
- Chalvardjian A, Scully RE. Sclerosing stromal tumors of the ovary. *Cancer* 1973; 31: 664-70.
- Gupta S. *Indian J Pathol Microbiol*. Sclerosing stromal tumor of ovary- a case report. 1999; 42(1): 97-9.
- Pai RR, Shaktawat SS, Khadilkar UN, Lobo FD, Rao VS, Philipore R. Sclerosing stromal tumor of the ovary-a clinicopathologic spectrum. *Indian J Pathol Microbiol*. 2005; 48(3): 370-2.
- Gee DC, Russell P. Sclerosing stromal tumors of the ovary. *Histopathology* 1979; 3: 367-76.
- Saitch A, Tsutsumi Y, Osamura Y, Watanabe Y. Sclerosing stromal tumor of the ovary. Immunohistochemical and electron-microscopic demonstration of smooth muscle differentiation. *Arch Pathol Lab Med* 1989; 113: 372-6.
- Cashell AW, Cohen ML. Musculinizing sclerosing stromal tumor of the ovary during pregnancy. *Gynecol Oncol* 1991; 43: 281-5.
- Damajanov J, Drobnjak P, Grizelj V, Longhino N. Sclerosing stromal tumor of the ovary : a hormonal and ultra structural analysis. *Obstet Gynecol* 1975; 45: 675-9.
- Stylianidou A, Varras M, Akrivis C, Fyhaktidou A, Stefanaki S, Antoniou N. Sclerosing stromal tumor of the ovary: A case report and review of the literature. *European J Gynaecol Oncol* 2001; 22: 300-4.
- Kawauchi S, Jsuru T, Kaku T, Kamara T, Nakano H, Tsuneyoshi M. Sclerosing stromal tumor of the ovary: a clinicopathologic, immunohistochemical ultrastructural, and cytogenetic analysis with special reference to its vasculature. *Am J Surg Pathol* 1998; 22: 83-92.