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Editorial

Small Bite, Big Threat

Vectors like mosquitoes, ticks, and fleas transmit parasites, visuses, or bacteria between people or between animals and peopl.

Vector-borne diseases account for 17% of the estimated global burden of all infectious diseases. Global trade, rapid international travel, and environmental changes such as climate change and urbanization are causing vectors and vector-borne diseases to spread beyond borders. Many diseases including Malaria, dengue, Chagas disease, leishmaniasis, lymphatic filariasis, and onchocerciasis are vector-borne; other vector-borne diseases include typhus and spotted fevers.

Over half the world's population is at risk from vector-borne diseases such as malaria and dengue. Each World Health Day, the World health Organization (WHO) selects a theme that highlights a global public health issue. This year's theme is vector-borne diseases, with a first-time focus on dengue.

Forty percent of the world's population is at risk from dengue virus; there are an estimated 390 million dengue infections each year in over 100 countries. Dengue is the worlds fastest growing vector-borne disease, with a 30-fold increase in disease incidence over the last 50 years. South East Asia and Latin America are especially affected but dengue also occurs in Africa, where cases are less often diagnosed.

Malaria, a vector-borne disease that is one of the most severe public health problems worldwide is a leading cause of death in many developing countries, where young children and pregnant women are the groups most affected. The WHO estimates that in 2012, there were 207 million cases of malaria and 6,27,000 people died of malaria. Most deaths were among children in Africa. Programs that provide proven interventions — artemisinin-based combination therapies (ACTs), insecticide-treated bed nets (ITNs), and indoor residual spraying (IRS) — have achieved a 33% reduction in malaria deaths in the Arfican region and 1.1 million lives saved globally. A recent review of 75 malaria resurgences during the 20th century found that about 9 out of 10 were due in part to the weakening of malaria control programs, with resource constraints being the most common cause. If our investment tails off, it isn't a question of whether malaria will resurge, but rather how bad the situation will be when it does. Rapid diagnostic tests and advances in mobile technology now give us the tools to do surveillance better and faster, but these need well-trained staff to deploly them effectively.

The malaria parasites and their mosquito vectors change every day to evade the weapons we use to kill them. Increasing parasite resistance to drugs and increasing mosquito resistance to insecticides are sure bets. Our tools and strategies also need to adapt and evolve. Now is the time to invest in new drugs, insecticides, and approaches for the future. While developing brand new compounds takes many years, understanding how to use better our existing tools — novel combinations of vector control tools, or creative uses of drugs for prevention,

for example — can help us continue to stay ahead of our elusive foes.

Through vector-borne diseases have the biggest impact on the world's poorest people, everyone, rich and poor, is at risk for infections. The best way people can prevent these infections is by preventing tick or mosquito bites using repellents,wearing long sleeves and pants, and showering after going outside to wash unattached ticks off your skin, sleeping under a bednet & using insect repellents.

Mosquitoes, flies, ticks and bugs may be a threat to your health - and that of your family -- at home and when travelling. This is the message of this year's World Health Day, on 7 April.

World Health Day: Key Messages

Vectors spread diseases

Mosquitoes, flies, ticks, bugs and freshwater snails can spread diseases that cause serious illness and death.

Diseases are preventable

Diseases such as malaria, dengue, leishmaniasis and yellow fever are preventable, yet they have the biggest impact on some of the world's poorest people.

50% of population is a risk

More than half of the world's population is at risk of these diseases. Increased travel, trade and migration made even more people vulnerable.

Dr. D. Maji, Prof. & Head, Dept. of Medicine, RKMSP

Original Article

Study of Correlation Between OAE (Otoacoustic Emission) and ABR (Auditory Brainstem Response) Test Results for Assessment of Hearing Loss in Term & Preterm Newborns in NICU

Dr. Manomit Haldar[1], Dr. Asha Mukherjee[2]

Abstract

Introduction:

Universal newborn hearing screening with OAE test is now recommended to detect hearing impairment. Sensory-neural hearing loss is detected by ABR test. Any correlation between these 2 tests is very significant, as early detection of hearing impairment helps proper language acquisition.

Aims & Objectives:

1. To find out the incidence of hearing impairment in both NICU admitted & normal babies.
2.. Any correlation between OAE and ABR tests.
3. To find out the association between several risk factors and hearing loss.

Materials & Methods:

A prospective observational study was performed on both NICU-admitted (case) and normal post-natal ward (control) babies. Screening OAE was done on day-3 of life, for all babies. 2nd OAE was repeated at 6wks. ABR test was done at 3 months of age for all of them.

Results:

In the NICU, 17 out of 100 babies had 'REFER' result in their screening OAE, out of which 14 had persistent 'REFER' in repeat OAE. On ABR testing at 3 months, 5 showed abnormal ABR with deafness. In control group, 10 out of 100 had 'REFER' result, 6 of them had persistent 'REFER' in 2nd OAE; only 1 detected as deaf. Prematurity, Mechanical ventilation, Sepsis, Jaundice & Drugs were significantly associated with abnormal OAE & ABR test.

Conclusion:

1. Risk of sensory-neural hearing loss is more in NICU admitted babies.
2. Both abnormal OAE & ABR test results were having good correlation with deafness. Therefore, a combination of these 2 tests is well suited for use in hearing assessment in newborn & older.

Introduction:

Hearing loss is the most common treatable congenital anomaly occurring in approximately 2-4 infants per 1000 live births [1-3]. However 2 to 4 per 100 infants surviving neonatal intensive care have some degree of sensorineural hearing loss.

Family history of deafness, prematurity, low birth weight, ototoxic medications (Aminoglycoside, Vancomycin, Furosemide), jaundice, infection (meningitis, congenital infection, septicemia), multiple births, seizures, birth asphyxia are well known important risk factors for hearing impairment. Preterm and term infants who require newborn intensive care are often exposed to these factors.

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Several studies by Yoshinaga-Itano and her colleagues have shown that when children are identified with hearing loss at birth and receive intervention before 6 months of age, they catch-up with their normal-hearing peers and demonstrate essentially normal language development by 5 yrs of age[4]. Because OAE is generated at the outer hair cells, it does not detect neural dysfunction (i.e. Eighth nerve or auditory brainstem pathology). This is detected by ABR test. ABR is an effective and noninvasive mean of assessing the functional status of the auditory nerve and brain stem auditory sensory pathway. It is not significantly altered by state of consciousness, drugs and variety of environmental factors, including other sensory input to the cortex[5].

In one correlational study on normal hearing neonates, conducted by Cacae AT and Pinheiro JM, result shows that these two tests provide independent information about auditory system integrity and sensitivity. Therefore, a combination of both these tests is well suited for use within a pediatric test battery[6].

Aims and Objectives:

To find out the incidence of hearing impairment in both NICU-admitted and normal newborns, to evaluate any correlation between the OAE and ABR test results during assessment of hearing and to find out the association between several risk factors and hearing loss.

Methodology:

This study has been conducted in the Neonatal Intensive Care Unit (NICU) and Post-natal ward of the Pediatric department of Ramakrishna Mission Seva Pratishthan, VIMS, Kolkata. It is a city based general hospital with annual delivery rate of average 4,000 babies per year. Rate of preterm delivery is average 100-140 year.

100 newborns born in this institution and admitted at NICU, irrespective of gestational age and risk-factors, were taken as 'CASE'. 100 normal term babies from post-natal ward, without any risk-factor were selected as 'CONTROL'. This study was conducted during period of June 2011 to June 2012 over 1 year. Screening of all newborns (both NICU and post-natal ward) was done by first OAE test on Day-3 of life, by distortion produce OAE (DPOAE) method. OAE was repeated to all subjects at 6 weeks of age, during their first vaccination follow-up and ABR testing for all babies at 3 months of age.

Tools used for this study were history taking, clinical examination, otoscopic examination, audio logical test: Distortion Product Otoacoustic Emission (DPOAE) test and electrophysiological test, Auditory Brainstem Response (ABR) test.

Data Analysis:

Software used for this study was SPSS version 16 and Graphics: Microsoft Excel 2007.

Results:

Total out of 200 subjects 25 showed 'REFER' results and 175 had 'PASS' results. In NICU-admitted babies (CASE group), out of 100, 17 had 'REFER' result and 83 was normal. In normal post-natal ward babies (CONTROL group), 8 had 'REFER' result and rest were normal.

In the repeat OAE, total 20 of them were still showing 'REFER' result. In 'CASE group' 14 had REFER result and in normal babies (CONTROL group) 6 had persistent REFER result. During ABR testing at 3 months of age all babies, we found-among the 14 persistent 'REFER' cases in 'CASE' group, 5 had abnormal

ABR results, whereas rest 95 babies were showing normal ABR wave forms. In normal babies 1 out of 100, had abnormal ABR waveform. Rest had normal ABR.

Association with Risk-factors:
Prematurity, Sepsis, Jaundice, Mechanical ventilation, Medications were significantly associated with abnormal ABR result ($p<0.05$).

SENSITIVITY AND SPECIFICITY OF OAE 1:

Group	True Positive	True Negative	False Positive	False Negative	Sensitivity	Specificity	Positive Predictive Value	Negative Predictive Value
CASE	5	83	12	0	100.00	87.37	29.41	100.00
CONTROL	1	92	7	0	100.00	92.93	12.50	100.00

SENSITIVITY AND SPECIFICITY OF OAE 2:

Group	True Positive	True Negative	False Positive	False Negative	Sensitivity	Specificity	Positive Predictive Value	Negative Predictive Value
CASE	5	86	9	0	100.00	90.53	35.71	100.00
CONTROL	1	94	5	0	100.00	94.95	16.67	100.00

CORRELATION BETWEEN OAE 1 & ABR:

			OAE 1				
			ABNORMAL	NORMAL	TOTAL	p-VALUE	Significance
CASE	ABR	ABNORMAL	5 (29)	0 (0)	5 (5)	<0.001	Significant
		NORMAL	12 (71)	83 (100)	95 (95)		
		TOTAL	17 (100)	83 (100)	100 (100)		
CONTROL	ABR	ABNORMAL	1 (13)	0 (0)	1 (1)	<0.001	Significant
		NORMAL	7 (88)	92 (100)	99 (99)		
		TOTAL	8 (100)	93 (100)	100 (100)		

CORRELATION BETWEEN OAE 2 & ABR:

			OAE				
			ABNORMAL	NORMAL	TOTAL	p-VALUE	Significance
CASE	ABR	ABNORMAL	5 (36)	0 (0)	5 (5)	<0.001	Significant
		NORMAL	9 (64)	86 (100)	95 (95)		
		TOTAL	14 (100)	86 (100)	100 (100)		
CONTROL	ABR	ABNORMAL	1 (17)	0 (0)	1 (1)	<0.001	Significant
		NORMAL	5 (83)	94 (100)	99 (99)		
		TOTAL	6 (100)	94 (100)	100 (100)		

Discussion:

Over 278 million people are hearing impaired globally. According to the National Commission on population ’2010, 115 million people in India are deaf. In India over 25,000 children are born deaf every year. In at least 30% of them parents are not aware about child’s inability to hear. Not only have that, up to 30% of children born with sensory-neural deafness, also had other congenital anomalies. As we know children born with severe to profound deafness, never acquire fully normal speech, even with best efforts, if treated after 6 months. So, screening of all newborn is important. *Identification and intervention before 6 months can have a significant impact on the development of expressive and receptive language.*

The Joint Committee on Infant Hearing (JCIH) first published a set of risk indicators for hearing loss in 1971, which were used primarily for screening infants in the neonatal intensive care unit (NICU), because most infants with risk factors were found in the NICU. However, subsequent studies reported that 19 to 42% of profoundly hearing-impaired children would be missed with targeted, risk-factor based screening. *If feasible based on logistics, all newborns should be screened. High risk criteria can be used to identify children who are at risk for hearing loss.*

The initial screening can be performed using OAE or AABR or both. OAE alone is not sufficient screening tool in infants who are at risk for neural hearing loss, especially NICU-admitted babies. Re-screening or a second hearing screening can be performed, if an infant does not pass the initial test in one or both the ears.

In India, it would be practical to do the second test at 6 weeks when the infant comes for

immunization. If he does not pass the re-screening test or results cannot be obtained in one or both the ears, he should be referred for diagnostic audio logical evaluation which must include Diagnostic ABR.

Conclusion:

1. Risk of sensory-neural hearing loss is more in NICU-admitted babies.
2. Incidence of hearing loss is higher even in normal babies (1 in 100 live births in our study).
3. In both CASE and CONTROL groups, Sensitivity of OAE is 100%. So, OAE is a good screening test.
4. In both CASE and CONTROL group, 2nd OAE at 6 weeks is more specific than 1st one.
5. Prematurity, Mechanical ventilation, Sepsis, Jaundice, Medication and Congenital infection was significantly associated with abnormal OAE & ABR (p value < 0.05).
6. In both NICU and normal postnatal babies, both 1st and 2nd OAE had statistically significant correlation with abnormal ABR. So, although these 2 tests detect functional integrity of 2 different parts of hearing pathway, both of them should be performed for complete evaluation of hearing in newborn and children.

Acknowledgement:

The authors acknowledge the technical support provided by staff members of the Dept. of E.N.T. and Head and Neck Surgery for doing audiological test of the patients.

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Original Article

Analgesic Efficacy of Transverse Abdominis Plane Block in Gynaecological Surgery Under General Anaesthesia

Dr. Krishnendu Chandra[1], Dr. Pabitra Ghoshal[2], Dr. Tulsi Nag[3], Dr. Jayanta Bhattacharya[4], Dr. Kasturi H. Bandhopadhyay[5], Dr. Rita Paul[6]

Abstract:

The Transverse Abdominis Plane Block (TAP) block is a method for blocking the anterior abdominal wall neural afferents via Lumbar Triangles of Petit. In this randomized, controlled and double blinded study we evaluated the analegesic efficacy of TAP block & tramadol requirement during 1st 24 postoperative hours after abdominal surgery.

Sixty adult women undergoing Total Abdominal Hysterectomy & Bilateral salphingooporectomy under general anaesthesia were included in the study. Patients were divided into two equal groups. The study group received TAP Block with 25 ml of 0.25% Bupivacaine & clonidine (75mcg) before reversal. The control group received TAP block with 25ml of sterile water. Both the groups received Inj. Fentanly (2mcg/kg) & Inf. Paracetamol (1gm) in the intraoperative period. In the post operative period both the group received inj Diclofenac sodium 75mg im BD & inj Tramadol (100mg) iv on demand. Each patient was assessed in the post operative care unit at 0, 2, 4, 6, 12 & 24 hrs and VAS score recorded. Any events of adverse effect were also noted in the 1st 24 post operative hrs. Total amount of tramadol requirement was also noted in the 1st 24 post operative hrs.

The TAP Block reduced VAS Score (TAP V/S control, mean) on emergence (1.16 v/s 5.48, p=0.0373) & various post operative time points including at 12 hrs (2.53 v/s 5.87, p=0.05). Tramadol requirment in first 24 hrs were also significantly reduced (113.33+_34.57mg v/s 350+_50.85mg, p=0.002). There was no complications attributable to TAP block. All TAP block patients with bupivacaine and clonidine reported high levels of satisfaction with their postoperative analgesic regimen.

The TAP block provided highly effective post operative analgesia in 1st 24 postoprative hours after major abdominal surgery.

Keywords: TAP Block. Lumbar Triangle of Petit, Multimodal Analgesia, Tramadol, Postperative Analgesia.

Introduction:

The pain experienced by patient after any abdominal surgery is derived from abdominal wall incision.[1] Abdominal wall generally consists of 3 layers. Externaloblique, internal oblique and transversusabdominis and their fascialsheaths. The central abdominal wall also includes rectus abdominis muscles and their fascialsheaths. This mascular wall is innervated by nerve afferents that course through the transverse abdominis neurofascial plane.[2]

A new approach of providing post operative analgesia is to block the sensory supply to anterior abdominal wall.[3,4] But the current method of performing abdominal field block is uncertain due to lack of clearly defined anatomical landmarks.

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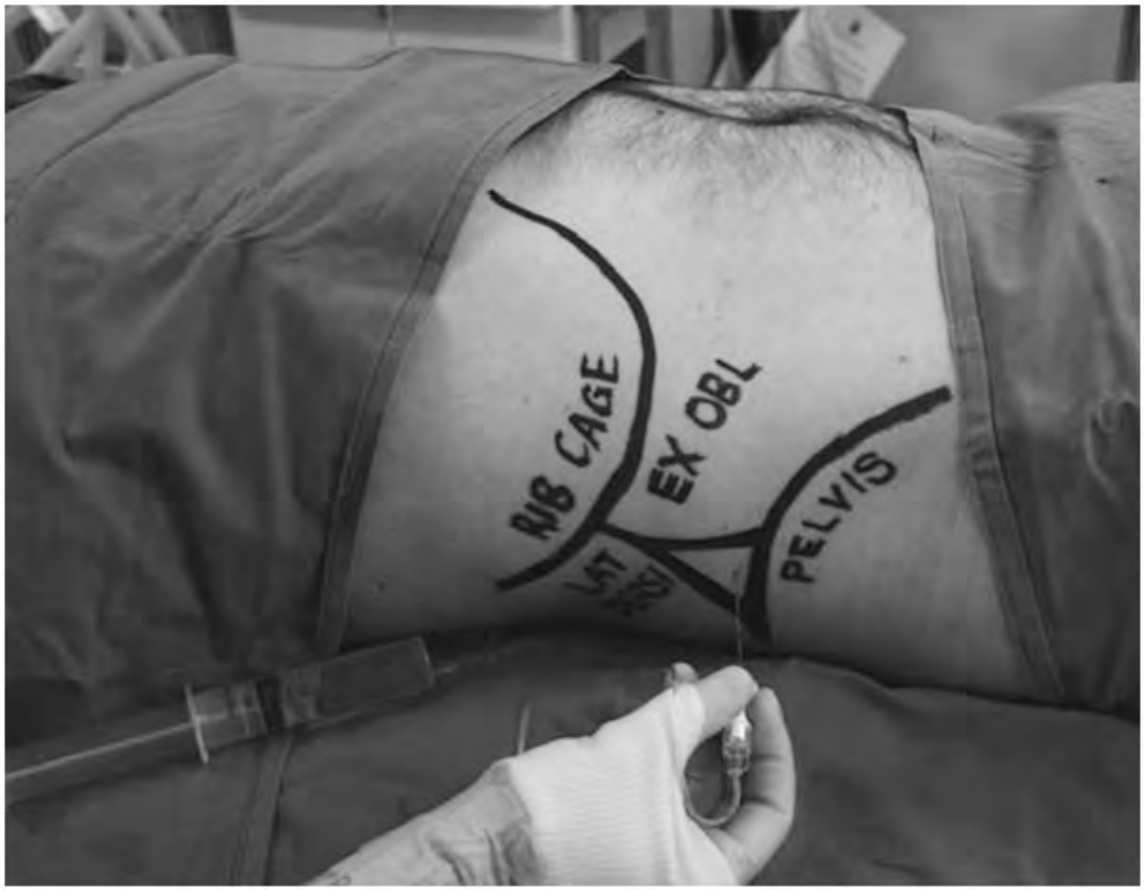
In this study, we tried to find out a new reliable approach of blocking anterior abdominal wall neural afferents. The neural afferents course through the neurofascial plane between the internal oblique and transverses abdominis muscles.[2] On the basis of clinical study we identified lumbar triangle of petit as a potential access point to this neurofascial plane. The triangle is bounded posteriorly by lattismusdorsi and anteriorly by external oblique with iliac crest forming the base of triangle and is a fixed and easily palpable landmark. By depositing local anaesthetics in the transverse abdomisnis plane via lumbar triangle of petit it is possible to block he sensory nerves of anterior abdominal wall before they pierce the musculature to innervate the abdomen. This novel block is called Transverse Abdominis Plane block or TAP block.

Methods and Materials:

After obtaintaing approval from hospital ethical committee sixty patients of ASA I and II undergoing Total abdominal hysterectomy and salphingooporectomy under conventional general anaesthesia were enrolled in the study. Informed written consent was taken from each of the patients and all the patients were explained about the procedure in their own language. The study was randomized, controlled and double blinded. Patients were randomized into two equal group of 30 patients. Study group received TAP block with injbuivacaine and clonidine and the control group received TAP block with normal saline. All the patient undergoing study received conventional general anaesthesia. On arriving to the operating room i.v access secured and all the standard ASA monitors (NIBP, ECG, PULSE OXIMETRY, ETCO2) were attached. Balanced anaesthesia was performed using iv induction agent propofol, muscle relaxant atracurium, opioidsfentanly and inhalational agent isoflurane. Both the group received iv fentanyl (2mcg/kg) and infusion paracetamol (15mg/kg) in the intraoperative period. The study group received TAP Block with 25 ml of 0.25% Bupivacaine & clonidine (75mcg) before reversal. The control group received TAP block with sterile water. The iliac crest was palpated from anterior to posterior until lattismasdorsi muscle was identified. The lumbar triangle of petit was identified just anterior to lattismasdorsimuscle. Using a 20 cc sterile needle the skin was pierced just cephalad to iliac crest over lumbar triangle of petit. The needle was advanced until the 1st resistance encountered which indicated that the needle is at external oblique muscle layer. Gentle advancement of the needle then resulted in a 'POP' sensation as the needle entered the plane between external and internal oblique fascia. Further advancement resulted in a second 'POP' sensation which indicated that the needle is at Transverse abdominisfascialplane. After aspiration to exclude any vascular puncture 25ml of 0.25% bupivacaine with clonidine (75mcg) was injected into that plane. TAP BLOCK was performed in the opposite side in the identical technique. On the other hand control group received TAP block with only 25ml normal saline. After complete reversal of anaesthesia all the patients were transferred to PACU for further management and monitoring. In the post operative period all the patient received injdiclofenac 75mg i.m BD and inj. Tramadol (100mg) iv on demand. Each patient was assessed in the post operative care unit at 0, 2, 4, 6, 12 & 24 hrs and VAS score recorded. Total amount of tramadol requirement was also noted in the postoperative period.

Anatomical Landmarks for TAP Block:



Aim of Study:

1. To evaluate analgesic efficacy of TAP block in post operative period in gynaecological surgery under general anaesthesia.

2. Tramadol requirement in first 24 hrs.

3. Any adverse events.

Results:

Sixty patient who underwent Total abdominal hysterectomy and bilateral salphingoophorectomy under general anaesthesia were included in the study total number of patients were randomized into two equal groups of 30 patients. The study group received TAP block with bupivacaine and clonidine & control group received sterile water instead of drugs before reversal.

All the patient underwent the same surgical procedure with conventional anterior abdominal wall incision. Both the groups were comparable with age, height, weight and operative procedure performed. Both the groups were comparable in their post operative heamodynamic status. SBP, DBP and Heart rate were very much comparable and the p value was insignificant.

Table 1: Demoraphic Data

	TAP GROUP	CONTROL GROUP	P VALUE
AGE	43±5.87	43.46±6.67	0.69
HEIGHT	156.76±3.56	157.1±3.41	0.72
WEIGHT	58.6±5.62	59.26±6.14	0.73

Table 2: Comparative Study of Post Operative Systolic Blood Pressure in Two Groups

	0 hrs.	2 hrs.	4 hrs.	6 hrs.	12 hrs.	24 hrs.
TAP GROUP	132	132	132	132	132	132
CONTROL GROUP	132	132	132	132	132	131
P Value	0.4	0.3	0.5	0.4	0.3	0.5

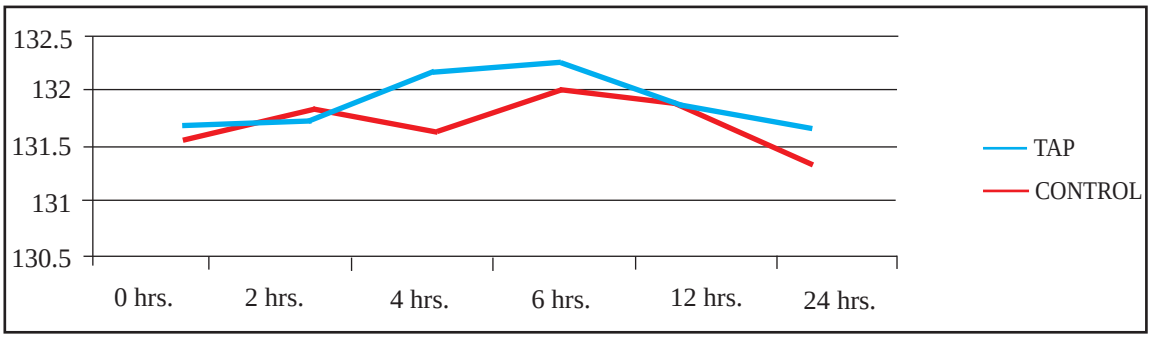


Table 3: Comparative Study of Post Operative Diastolic Blood Pressure in Two Groups

	0 hrs.	2 hrs.	4 hrs.	6 hrs.	12 hrs.	24 hrs.
TAP	81.9	81.7	81.5	81.5	81.6	81.6
CONTROL	81.9	81.9	81.1	81.5	81.8	81.7
	0.32	**0.46**	**0.61**	**0.32**	**0.42**	**0.35**

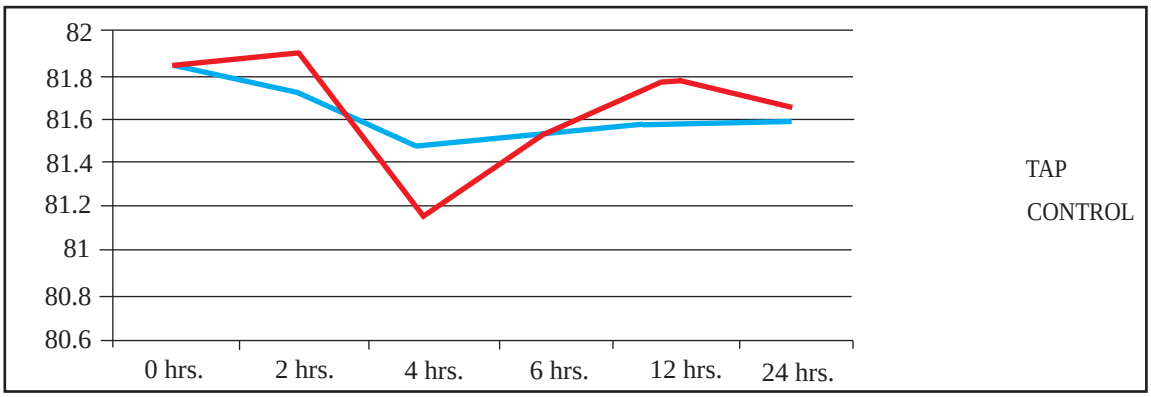
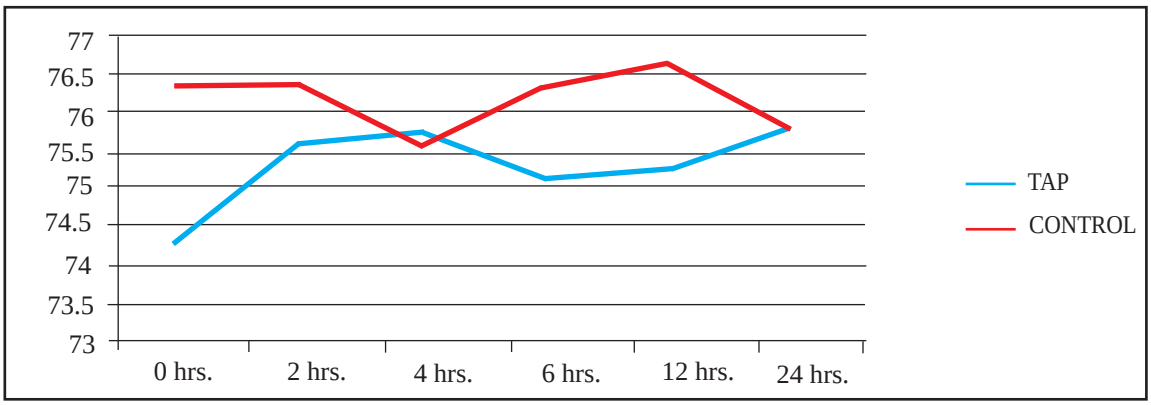


Table 4: Comparative Study of Post Operative Heart Rate in Two Groups

	0 hrs.	2 hrs.	4 hrs.	6 hrs.	12 hrs.	24 hrs.
TAP	74.3	75.6	75.7	75.1	75.3	75.8
CONTROL	76.4	76.4	76.5	76.4	76.7	75.8
	0.93	**0.66**	**0.69**	**0.86**	**0.94**	**0.37**

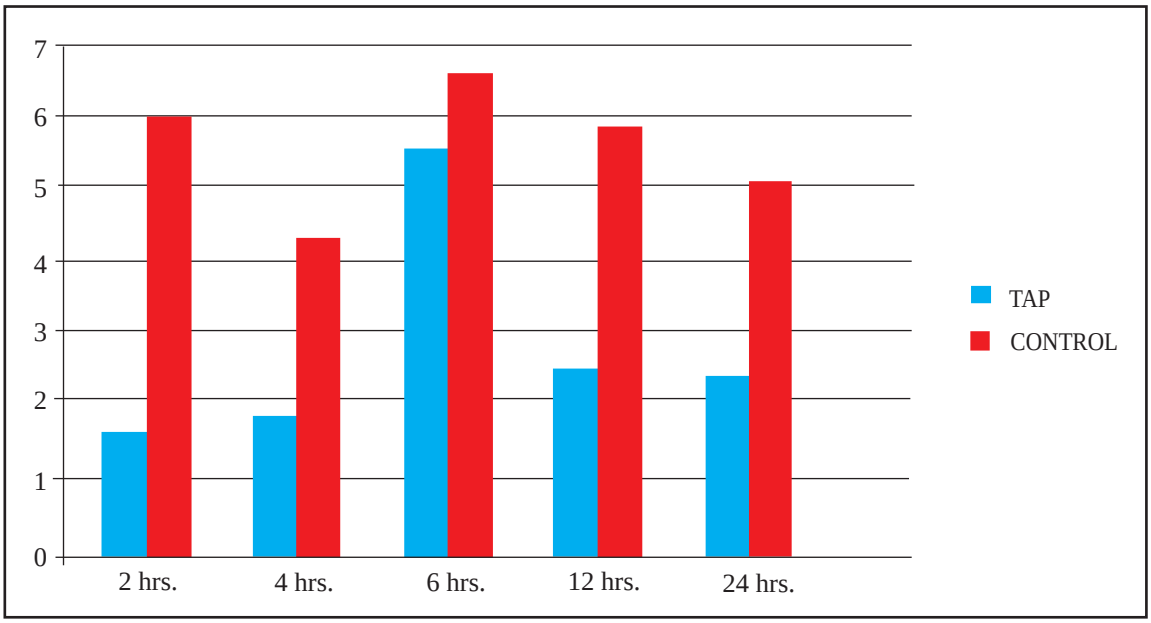


On the other hand, the TAP Block reduced VAS Score (TAP V/S control, mean) on emergence (1.16 v/s 5.48, p=0.0373) & various post operative time points including at 12 hrs (2.53 v/s 5.87, p=0.05). Tramadol requirment in first 24 hrs were also reduced significantly (113.33±34.57mg v/s 350±50.85mg, p=0.002). There was no complications attributable to TAP block. All TAP patients with bupivacaine and clonidine reported high levels of satisfaction with their postoperative analgesic regimen.

Table 2: Postoperative VAS SCORE

	0 hrs.	2 hrs.	4 hrs.	6 hrs.	12 hrs.	24hrs.
TAP Group	1.2	1.7	1.9	5.566667	2.5	2.5
CONTROL GROUP	5.5	6	4.3	6.645161	5.9	5.1
P VALUE	0	0.2	0	<0.0001	0.1	0.2

Bar Diagram Representing Table 2:



Discussion:

The benefits of adequate postoperative analgesia are clear and include a reduction in the post operative stress response, reduction in postoperative morbidity and in certain types of surgery improved clinical outcome. Effective pain control also facilitates rehabilitation and accelelarates recovery from surgery. Other benefits of effective regional techniques include reduced pain intensity, decreased incidence of side effects from analgesics and improved patient comfort.

Direct blockade of neural afferent supply of abdominal wall such as abdominal field blocks, ilioinguinal and hypogastric nerve blocks have long been recognized as capable of providing significant postoperative analgesia in patient undergoing abdominal surgical procedure such as caesarean delivery and inguinal herniorrhaphy [3,4].

However lack of clearly defined anatomical landmarks has meant that the full potential of abdominal wall blockade in patients undergoing major abdominal procedures remain to be

realized. Analternative, simple, reliable and effective regional analgesic technique is required.

Innervation of the anterolateral abdominal wall arises from the anterior rami of spinal nerves T7 to L1. These include the intercostal nerves (T7-T11), the subcostal nerve (T12), and the iliohypogastric and ilioinguinal nerves (L1). The anterior divisions of T7-T11 continue from the intercostal space to enter the abdominal wall between the internal oblique and transversus-abdominis muscles until they reach the rectus abdominis, which they perforate and supply, ending as anterior cutaneous branches supplying the skin of the front of the abdomen. Midway in their course they pierce the external oblique muscle giving off the lateral cutaneous branch which divides into anterior and posterior branches that supply the external oblique muscle and latissmusdorsi respectively. The anterior branch of T12 communicates with the iliohypogastric nerve and gives a branch to the pyramidalis. Its lateral cutaneous branch perforates the internal and external oblique muscles and descends over the iliac crest and supplies sensation to the front part of the gluteal region.

The iliohypogastric nerve (L1) divides between the internal oblique and transversusabdominis near the iliac crest into lateral and anterior cutaneous branches, the former supplying part of the skin of the gluteal region while the latter supplies the hypogastric region.

The ilioinguinal nerve (L1) communicates with the iliohypogastric nerve between the internal oblique and transversusabdominis near the anterior part of the iliac crest. It supplies the upper and medial part of the thigh and part of the skin covering the genitalia.

The aim of a TAP block is to deposit local anaesthetic in the plane between the internal oblique and transversusabdominis muscles targeting the spinal nerves in this plane. The innervation to abdominal skin, muscles and parietal peritoneum will be interrupted. If surgery traverses the peritoneal cavity, dull visceral pain (from spasm or inflammation following surgical insult) will still be experienced. The block can be performed blind or using the ultrasound.

In this randomized, controlled, double blinded clinical trial the TAP block produced effective and prolonged postoperative analgesia when compared with standard therapy, in patient undergoing surgery involving anterior abdominal wall. The TAP block reduced postoperative pain score and also reduced postoperative tramadol requirement compared to control group.

Gerard Curley, Mc Donald et al showed that TAP block as a component of multimodal analgesic regimen, provided superior analgesia when compared with placebo upto 48 post operativehrs after elective cesasearn section[5]. John G McDonnel, Brain O Donnel et al showed that TAP block provided highly effective post operative analgesia after first 24 hrs after major abdominal surgery. There was also no complication attributed to TAP block. Morphine requirment also significantly reduced ($p<0.05$) in the 1st 24 post op hrs compared to control group[6]. Analgesic potential of TAP Block in a series of patient undergoing radical prostatectomy has also been shownby O'Donnell BD, McDonnell JG, McShane AJ[7].

On the other hand Cristiano R et al showed that TAP block alone is not completely effective in the management of post operative pain & some form of extra analgesic agents always needed while better results were obtained when

resections performed via laparoscopic procedure[8]. R. C. N. McaMarrow et al showed spinal morphine but not TAP block improved analgesia after ceasareansection. The addition of TAP Block with bupivacaine 2mg/kg to spinal morphine did not further improve analgesia[9].

In our study we found that TAP block is highly effective as a component of multimodal analgesia after surgery involving ant. abdominal wall incision. TAP block significantly reduced post operative VAS score in 1st 24 hrs. TAP block also reduced tramadol requirment significantly in 1st 24 hrs as compared to control group. There was also no significant complications related to TAP block in the post operative period. All the TAP patients with bupivacaine and clonidine reported high levels of satisfaction with their post operative analgesic regimen. We may come to the conclusion that as a component of multimodal analgesia TAP block provived highly effective post operative analgesia in 1st 24 hrs after sugery involving ant abdominal wall incision.

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Original Article

Evaluation of Cervical Pap Smear by Digital Colposcopy

Dr. Rajmohan Ghosh[1], Dr. Sibani Sengupta[2]

Abstract:

Aim: To evaluate abnormal cervical Pap Smears and some normal Pap Smears with persistent clinical abnormality, by colposcopy supported by histopathology.

Materials and Methods: The study was conducted in the Dept. of Obstetrics and Gynecology at Ramakrishna Mission Seva Pratishthan, Vivekananda Institute of Medical Sciences, Kolkata, over a period of 1 year from from January 2011 to December 2011. 46 women were taken in each of the abnormal and normal Pap Smear group on outpatient basis. Colposcopy was done in all women and directed biopsy was taken as necessary.

Results: In normal Pap Smear group, 35 patients (76.09%) had a normal but 11 patients (23.91%) had abnormal Colposcopic findings. In abnormal Pap Smear group, 26 patients (56.52%) had abnormal but 20 patients (43.48%) had normal Colposcopic findings. In the group with normal Pap Smear, 10 patients (21.74%) had abnormal histopathology report and in those with abnormal Pap Smear, 25 patients (54.35%) had abnormal histopathology (HP) report- this confirmed that even though the cytology is normal there can be colposcopic abnormality (supported by HP) in some subjects. However, the distribution of abnormal Colposcopy and histopathology is significantly (p-value: 0.001) higher in abnormal Pap Smear group than in normal Pap Smear group. The sensitivity of the Pap Smear is 69.44% and the specificity is 62.50%.

Conclusion: All patients with abnormal Pap Smear and those with a normal Pap Smear butpersistent clinical abnormalities are to be considered for Colposcopic evaluation.

Keywords:

PAP Smear, Digital Colposcopy.

Introduction:

Cervical cancer is the fourth most common cancer in women worldwide (after breast, colorectal and lung cancer), with about 5,28,000 new cases and 2,66,000 mortalities in 2012. More than 85% of the global burden occurs in the developing countries. In India, there are about 1,34,000 new cases and 72,000 deaths due to cancer cervix, according to the GLOBOCAN cancer fact sheet, 2012[1]. This occurs despite considerable knowledge and understanding about the prevention, early detection, and treatment of the disease and is probably due to lack of infrastructure for organized screening, in addition to a multitude of other causes.

Cervical cancer screening using exfoliative cytology - the Pap Smear test - has been an extremely successful public health intervention where practised effectively, reducing the incidence by 79% and mortality by 70-80% since 1950s, since its introduction by George Papanicolaou [2,3].

This lesion may exist in the non-invasive stage for years and shed abnormal cells that can be detected on cytological examination and hence

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the efficiency of cytological screening. However, in recent years, it has been found that Pap Smear has significant limitations. The sensitivity of Pap Smear ranged from 47% to 62% and specificity from 60-95% in detecting CIN2 and 3 in a recent review [4-6]. Approximately 30% of new cancer cases each year result from women who have undergone Pap testing and had a negative report. This could be due to errors in sampling, fixation, and/or interpretation [7]. Thus, in order to make the screening more effective, Pap Smear needs to be supplemented by other tests like HPV DNA and follow up.

Detection of HPV is associated with a 250-fold increase in the risk of high-grade CIN. The percentage of intraepithelial neoplasia attributed to HPV infection approaches more than 90%; however, tests for HPV detection have certain drawbacks too[8].

After Pap Smear with or without HPV DNA, Colposcopy needs to be done. It has a central role in the diagnostic investigation and management of women with premalignant disease of the cervix detected by abnormal cervical cytology in the majority of (WHO recommended) cervical screening programmes [9,10]. In women with low grade abnormal cytology, in recent years, there has been a trend towards increasing referral for Colposcopy, rather than surveillance by repeat cytology tests [11]. This change in practice evolved from recognition that some women with low grade abnormal cytology, or even normal cytology, have underlying high grade cervical intraepithelial neoplasiaand might eventually require Colposcopy for persistent cytological abnormalities [12,13].

The present study is aimed at Colposcopic evaluation of cervical Pap Smears – both abnormal and normal with some persistent clinical suspicion.

Materials and Methods:

The present study was undertaken at Ramakrishna Mission Seva Pratishthan, Vivekananda Institute of Medical Sciences, 99, Sarat Bose Road, Kolkata – 26.

This study is a comparative study carried out from January 2011 to December 2011 (12 months).

Patient evaluation was done according to the previously designed proforma, which included demographic, cytological, colposcopic and histological parameters.

During the year under study, a total number of 1012 cases of PAP Smear were done, where 72 cases showed epithelial abnormality. 50 cases out of the 72 were enrolled for the study while 4 subjects dropped out. Therefore, study group B consisted of 46 cases and similarly 46 cases were enrolled for the study group A.

The number of subjects were placed in study group A and B.

Study Group A – This group consisted of 46 women with 2 consecutive normal Pap Smear at 3 months intervals. They were having:

Complaints of -

1. Persistent white discharge even after a course of treatment for vaginal infections
2. Post coital bleeding and
3. Intermenstrual spotting/bleeding per vagina where functional or uterine cause has been excluded, and

Clinical findings of -

1. Grossly enlarged angry looking cervix

2. Congested cervix which bleeds on introducing the speculum
3. Abnormal vessels.

Study Group B – This group consisted of 46 women with abnormal Pap Smear categorized according to the Bethesda III System 2001 which includes patients with reports of-

1. ASCUS (atypical squamous cells of unknown significance)
2. ASC-H (atypical squamous cells which high-grade lesions must be excluded)
3. LSIL (low-grade squamous intraepithelial lesions)
4. HSIL (high-grade squamous intraepithelial lesions)

   The normal subjects were reported as 'Negative for Intraepithelial Lesion or Malignancy (NILM)'

Both groups of patients were subjected to colposcopic evaluation supported by histopathology.

Sample Design:

Inclusion Criteria:

In this study patients who underwent routine cervical cytology (Pap Smear) and had abnormal Pap Smear reports were included.

Some women who had normal Smear reports but had persistent clinical abnormalities were also included.

Exclusion Criteria:

a) Atypical glandular cells (AGC)
b) Pregnancy
c) Patients with any gross pelvic pathology requiring immediate surgery.

Study Procedure:

Clinical Study:

All patients in our study were subjected to routine gynaecological examination and Pap Smear screening after recording of history in a pre-designed proforma.

A. History:

1. Age, parity and socioeconomic status were recorded. The patients were classified according to low, middle, high class according to education, occupation and income as per modified Kuppuswamy scale [14].
2. Chief complaints of every patient were recorded.
3. Details of menstrual history were recorded.
4. History of high risk factors like early sexual intercourse, age of first pregnancy, number of sexual partners, sexually transmitted disease, oral pill use and smoking habits.
5. Past medical, surgical and past obstetric history were recorded.
6. History of previous screening (Pap Smear) if any were recorded.

B. Clinical Examination:

1. General and Systemic examination was carried out to detect any organic disease.
2. Per speculum examination: after taking verbal consent and bladder evacuation, in presence of female attendant, was carried out in dorsal position, under proper illumination. Any abnormal finding like presence of erosions, white discharge, abnormal vascular pattern, presence of any growth or polyp were noted.

3. Bimanual examination was done following the speculum examination to detect any pelvic pathology.

Then the patient was sent for cytology by PAP Smear.

Cytological Screening:

All patients, who were sexually active or married for at least 3 years were subjected to routine Pap Smear screening according to World Health Organisation cervical cancer screening guidelines.

The Smears were taken in the Pathology Department and observed under microscope after staining by Papanicolaou stain.

The reporting is done according to **The Bethesda System 2001[15].**

Selection of subjects for Colposcopy based on cytology reports:

The patients with abnormal Pap Smear report and the patients with normal Pap Smear but with persistent clinical problems were taken for Colposcopy.

Colposcopy Procedure:

Colposcopy is done by a Digital Video Colposcope which is a computer aided device.

After explaining the procedure, consent is taken and patient particulars are recorded. Patient is placed in lithotomy position after evacuation of the urinary bladder.

Using a Cusco's speculum the cervix is exposed and cleared of any mucus by normal saline. It is then bathed in 5% Acetic acid (freshly prepared) for 1 minute and then painted by Lugol'siodine. After every step, photographs are taken and saved in the Computer. Any acetowhite and iodine negative area is noted and categorised according to modified Reid colposcopic index. Biopsy was taken and sent for HP examination. If visualisation of squamocolumnar junction (scj) is difficult, then the external os is opened by a long curved artery forceps and the scj is seen.

Results And Analysis:

Table - 1. Distribution of The Cases in Different Age Groups

Age groups (yrs.)	Normal Pap smear group No (%)	Abnormal Pap smear group No (%)	Total No (%)
<30	10 (21.74)	5 (10.87)	15 (16.3)
30-45	30 (65.22)	29 (63.04)	59 (64.13)
>45	6 (13.04)	12 (26.09)	18 (19.57)
Total	46 (100)	46 (100)	92 (100)

Mean age for normal Pap Smear group is 37.74 years and abnormal Pap Smear group is 39.11 years and the p value=0.159 (not significant).

Diagram - 1: Distribution of Normal and Abnormal Pap Smear in Different Age Groups

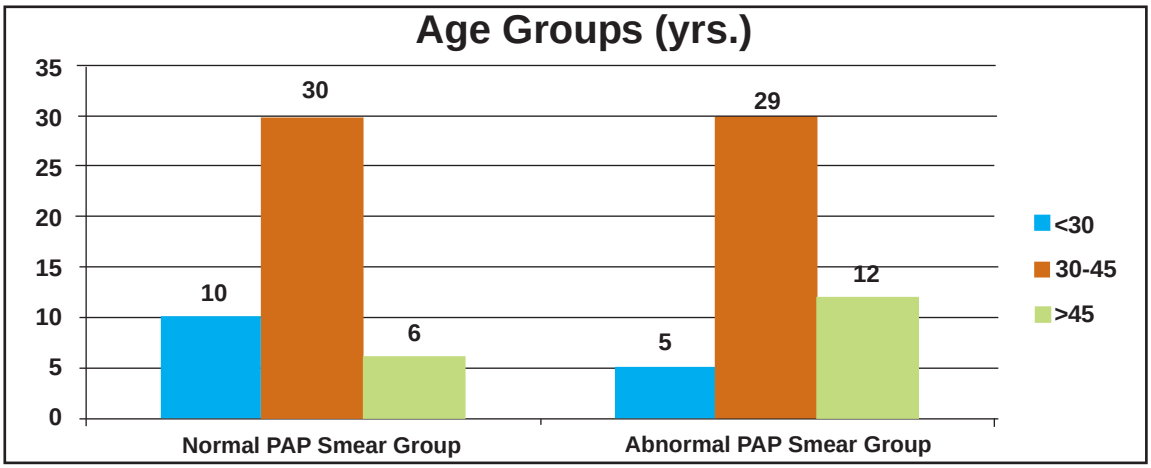

Table - 2: Distribution of Normal and Abnormal Pap Smear Among Different Socio Economic Classes

Socio Economic Status	Normal PAP smear group No (%)	Abnormal PAP smear group No (%)	Total No (%)
Lower	27(58.7)	31(67.39)	58(63.04)
Middle	16(34.78)	14(30.43)	30(32.61)
Upper	3(6.52)	1(2.17)	4(4.35)
Total	46(100)	46(100)	92(100)

p value=0.027 (not significant).

Diagram - 2: Distribution of Normal and Abnormal Pap Smear Among Different Socio Economic Classes

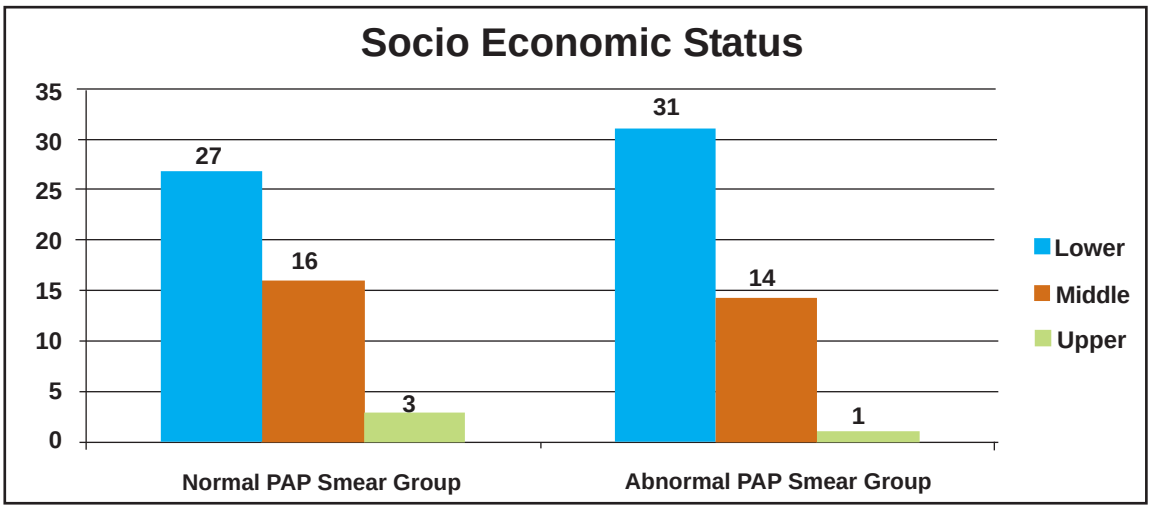

Table 2 and Diagram 2 show the distribution of normal and abnormal Pap Smear among different Socio Economic Classes In our institute, most of the cases (63.04%) are from the lower socio economic status.

Table - 3: Distribution of Colposcopic Findings in Normal and Abnormal Pap Smear Groups

	Normal PAP Smear	Abnormal PAP Smear	P Value
Normal Colposcopy	35/46	20/46	0.001
Abnormal Colposcopy	11/46	26/46	

Diagram - 3: Distribution of Colposcopic Findings in Normal and Abnormal Pap Smear Groups

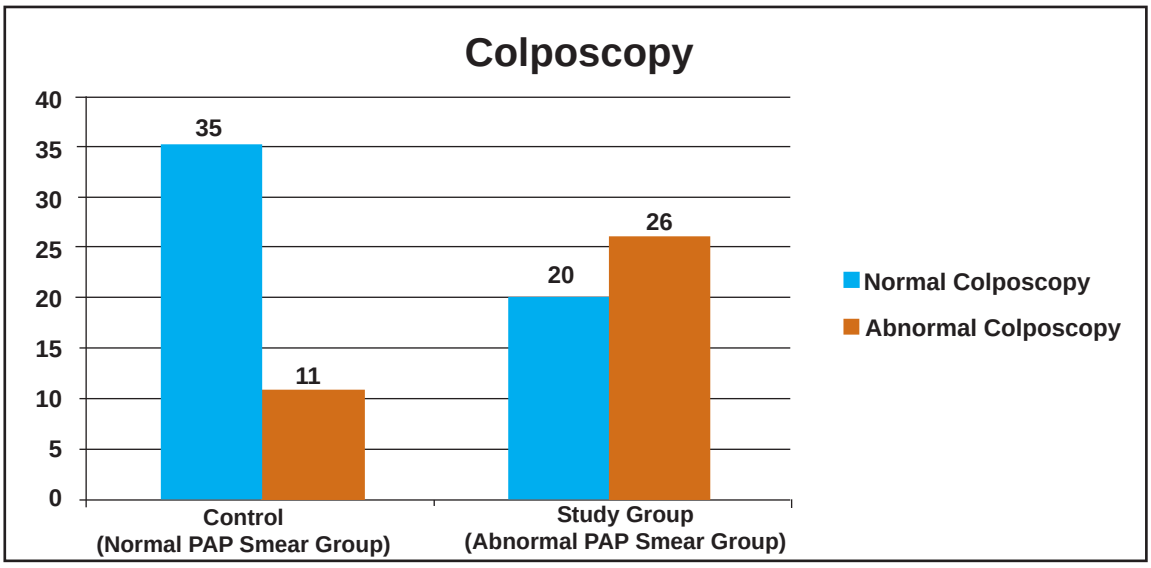

Table 3 and Diagram 3 show the distribution of Colposcopic findings in normal and abnormal Pap Smear groups. The distribution of abnormal Colposcopy is significantly (p-value: 0.001) higher in abnormal Pap Smear group than in normal Pap Smear group.

Table - 4: Distribution of Histopothologic Findings in Normal and Abnormal Pap Smear Groups

	Normal PAP Smear	Abnormal PAP Smear	P Value
Normal Histopathology	36/46	21/46	0.001
Abnormal Histopathology	10/46	25/46	

Diagram - 4: Distribution of Histopathology Findings in Normal and Abnormal Pap Smear Groups.

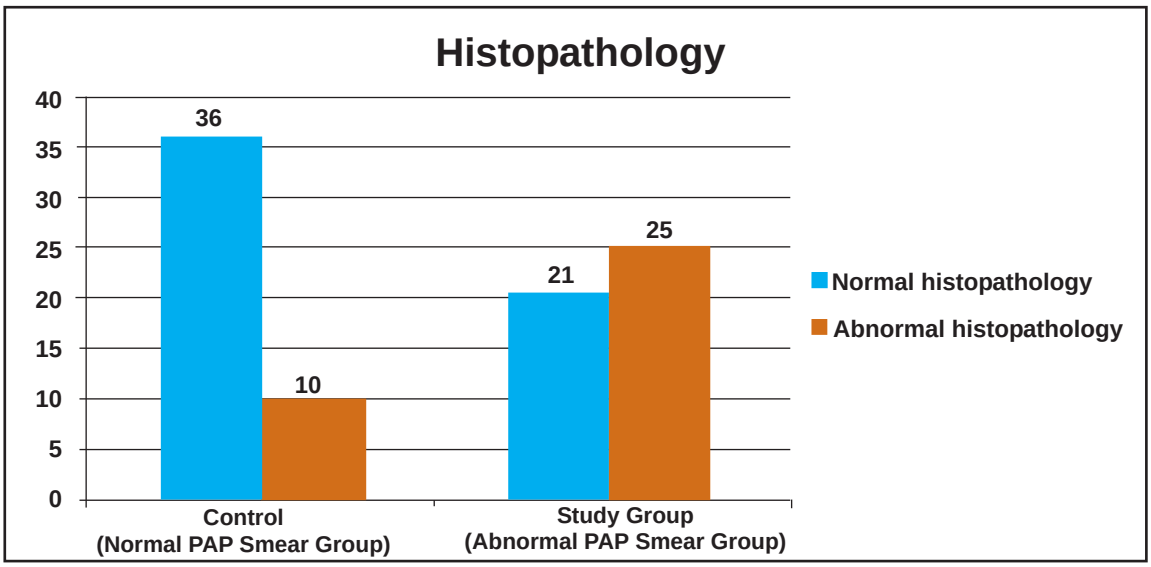

Table 4 and Diagram 4 showthe distribution of histopathologic findings in normal and abnormal Pap Smear groups. The distribution of abnormal histopathology is significantly (p-value: 0.001) higher in abnormal Pap Smear group than in normal Pap Smear group.

Table - 5: Shows the Sensitivity, Specificity, Positive Predictive Value, Negative Predictive Value and Diagnostic Accuracy of Pap Smear in Our Study.

Sensitivity	Specificity	PPV	NPV	Diagnostic accuracy
69.44	62.50	54.35	76.09	65.22

Diagram - 5: Shows the Sensitivity, Specificity, Positive Predictive Value and Negative Predictive Value of Pap Smear in Our Study

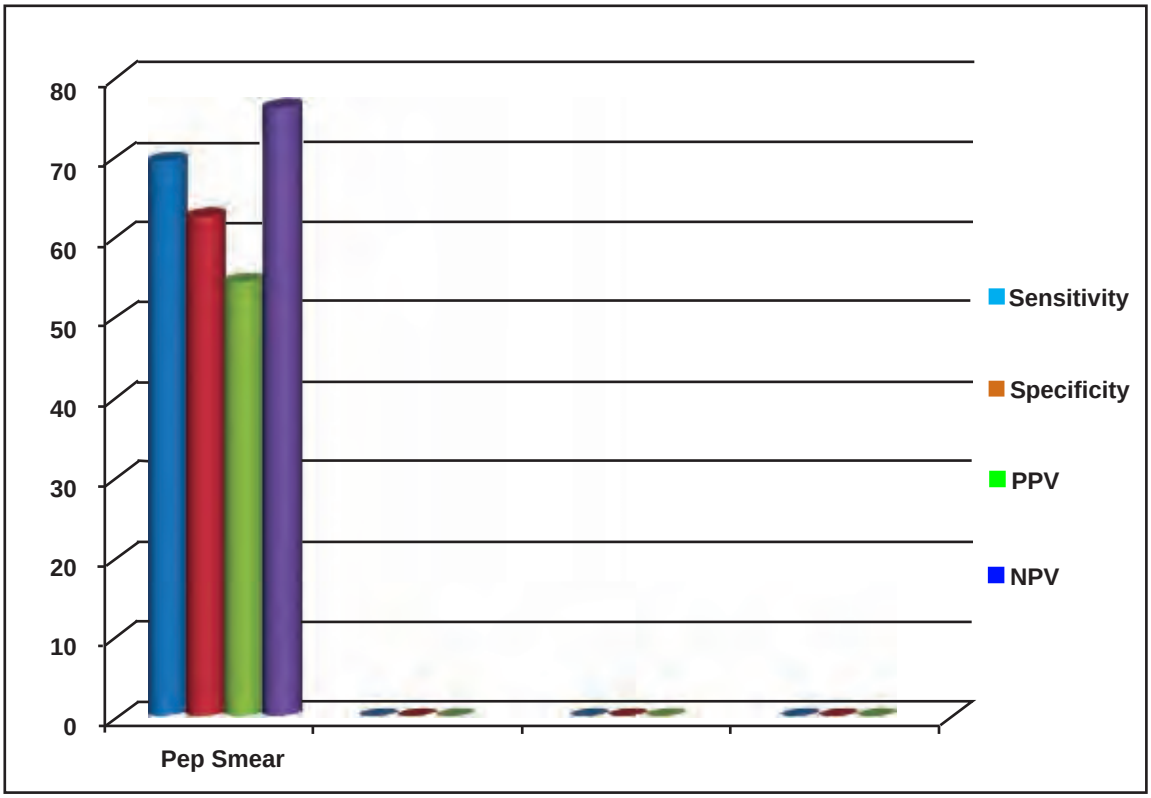

Table 5 and Diagram 5 show the sensitivity, specificity, PPV and NPV of Pap Smear in our study. The sensitivity of the Pap Smear is 69.44% and the specificity is 62.50%. The PPV and NPV of Pap Smear in our study is 54.35% and 76.09% respectively.

Table - 6: Shows the Sensitivity, Specificity, Positive Predictive Value and Negative Predictive Value of Colposcopy in Our Study

Sensitivity	Specificity	PPV	NPV
100	96.49	94.59	100

Diagram - 6: Shows the Sensitivity, Specificity, Positive Predictive Value and Negative Predictive Value of Colposcopy in Our Study.

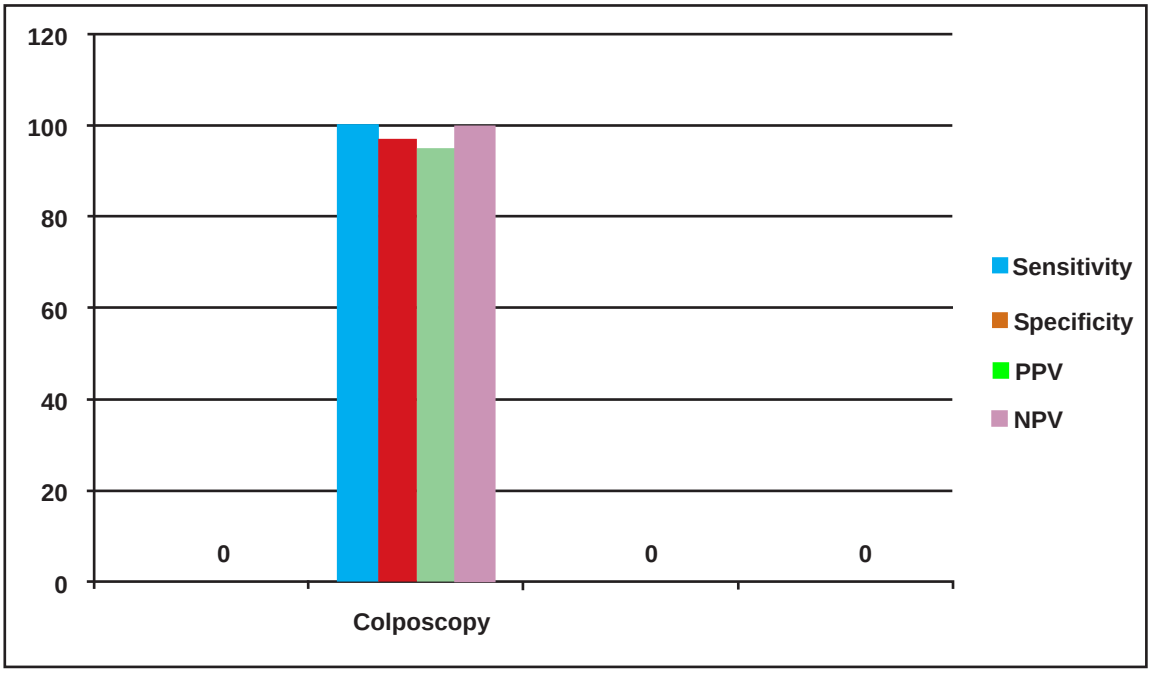

Table 6 and Diagram 6 show the sensitivity, specificity, PPV and NPV of Colposcopy in our study. The sensitivity of the Colposcopy is 100% and the specificity is 96.49%. The PPV and NPV of Colposcopy in our study are 94.59% and 100% respectively.

Discussion:

Women with cervical cancer die about 18 years earlier than they would have otherwise. Thus, cervical cancer is an important public health problem that deserves urgent attention. Screening with Pap Smear has resulted in a dramatic reduction in the incidence of invasive cervical cancer in thedeveloped world, but has not been able to completely eliminate the disease even in those countries. This is because of the many pitfalls of the Pap Smear which alone is clearly inadequate for the purpose.

The application of the detection of HPV DNAhas its drawbacks and is still being evaluated for its implementation.

In our study we,therefore,evaluated the Pap Smear reports with Colposcopy and histopathology.

The patients who attend the Gynaecology OPD and are married for at least 3 years undergo a Pap test. Many of these patients, however, donot come back for further evaluation, even if the Pap reports are abnormal, and cannot be contacted either, since many of themcome from remote areas and have no contact number. So we selected those patients who came back with the Pap reports for further evaluation.

In one study performed in Bangladesh, Urmila Banik et al showedepithelial cell abnormality in 139 (8.18%) patients out of a total of 1699

patients who had their Pap Smear done. In their study they found 6.36% cases of LSIL, 1.18% cases of HSIL and 0.18% cases of ASC-US among the total cases of Pap Smear[16].

In another work, Edelman et al [17] studied Pap Smears from 29295 females over a period of one year and the Pap Smear abnormalities were as follows: 9.9% ASC-US, 2.5% LSIL, 0.6% HSIL, and 0.2% invasive cancer.

In our hospital, a total of 1012 Pap Smears done during one year shows 72 (7.11%) cases with epithelial cell abnormality; among them, 4.55% cases were LSIL, 1.38% cases were HSIL and 0.8% cases were ASC-US. These findings are almost similar to the findings obtained by Urmila Banik, et al, in Bangladesh but not with the study of Edelman et al. This difference may be due to different study population - different race and geographical area with different socio economic status and also the sample size.

In our study 73.91% cases of abnormal Pap Smear was in the age group of 45years or less and 26.09% cases of abnormal Pap Smear was in the age group of more than 45 years of age. This finding slightly differs from the study performed in Bangladesh by Urmilla Banik, et al, [16] which shows 53.96% of cases of abnormal Pap Smear in the age group below 45 years and 46.04% of cases above 45 years. The mean age of our study population was 38.42 years whereas the mean age in the study by Banik was 41.97 years, which probably may explain the difference.

In the present study, in normal Pap Smear group 58.7% cases were from low socio economic group, 34.78% cases were from middle socio economic group and 6.52% cases were from upper socio economic group. In abnormal Pap Smear group 67.39% cases were from low socio economic group, 30.43% cases were from middle socio economic group and 2.17% cases were from upper socio economic group.

In our study, 46 cases of normal Pap Smear and 46 cases of abnormal Pap Smear were evaluated by Colposcopy and histopathology. 76.09% of cases of normal Pap Smear had normal Colposcopy and 78.26% of cases had normal histopathology. 43.48% of cases of abnormal Pap Smear had normal Colposcopy and 45.65% of cases had normal histopathology. So in our study the sensitivity of the Pap Smear is 69.44% and the specificity is 62.50% for detecting epithelial cell abnormality. In a recent review the accuracy of cervical cytology assessment, the sensitivity of the Pap test in detecting CIN 2 and 3 ranged from 47% to 62% and the specificity ranged from 60% to 95% [4-6] These findings are almost similar to our study.

In the year of 2011, Katyal Surabhi and Mehrotra Ragini, in their study found that the sensitivity and specificity of Pap Smear was 69.2 and 63.72% while for HPV DNA testing was 92.3 and 84% respectively. Sensitivity and negative predictive value of combined Pap Smear and HPV DNA test increased to 100% [20]. This study also has a similar finding for the sensitivity and specificity of the Pap Smear.

The Agency for Health Care and Policy Research, now renamed the Agency for Healthcare Research and Quality (AHRQ), undertook a literature review of conventional cervical cytology testing techniques and compared them with newer technologies designed to reduce the false-negative rate [21]. For this project, five reports were analyzed, and the conclusion was that the sensitivity of conventional cytologic testing in detecting

cervical cancer precursor lesions was 51%. This is a false-negative rate of 49%. In three recent reviews of the accuracy of cervical cytology assessment, the sensitivity of the Pap test in detecting CIN 2 and 3 ranged from 47% to 62% and the specificity ranged from 60% to 95% (4,5). The present study results for the sensitivity and specificity of Pap Smear is similar to the review done by The Agency for Health Care and Policy Research.

Ratnam et al [22] found sensitivity of Pap Smear 45% and specificity 64% which is similar to present study. Clavel et al [23] and Kumar et al. [24] found sensitivity of combined Pap and HPV DNA test 93.3% and negative predictive value of combined test to be 98.8%. HPV testing in conjunction with cytology improves the screening efficacy of cytology alone and may allow for a more effective and safe primary screening programme with increased screening interval.

In our study the abnormal Pap Smear were evaluated by colposcopic examination and directed biopsy. In the present study, we found the sensitivity of the Colposcopy 100% and specificity 96.49%. In one study by Mitchell et al, [5] in 1998, Colposcopy had a reported sensitivity ranging from 87% to 99% to diagnose cervical neoplasia, but its specificity is lower, between 23% and 87% [11] In another study by Daniel et al found that the Colposcopy yielded a sensitivity of 92% and a specificity of 64.9% [25].

In our study, 35 out of 37 (94%) cases of abnormal Colposcopy had abnormal histopathology which is comparable to one study done by Staff A et al. In their study, they found the correlation between the colposcopic impression and the histologic diagnosis after colposcopically directed punch biopsy is very high with an accuracy of greater than 85% [26].

In the present study, the distribution of abnormal Colposcopy and histopathology is significantly higher in abnormal Pap Smear group than in normal Pap Smear group with a p-value of 0.001. That indicate a referral for Colposcopy should be done in all cases of abnormal Pap Smear which is in accordance with the ASCCP guideline for management of cervical intraepithelial neoplasia [27]. Importantly, there were 10 cases of abnormal histopathology obtained by colposcope guided biopsy in 46 women who had normal or negative cytology reports - the colposcopy being done because of persistent clinical abnormality.

The accuracy of the Pap test has traditionally been determined using the diagnoses on the follow-up histologic samples as the gold standard (4,28). Although undoubtedly practical, this determination is potentially fraught with many errors related to factors arising in the interim, such as regression of human Papillomavirus infection, new lesion acquisitions or Colposcopy-associated variability. In the study of Mayeaux et al [29], a repeat conventional Pap Smear prior to Colposcopy had a sensitivity of 48% for CIN.

Infection with high risk Human Papilloma virus (HR-HPV) has now been found to be the major risk factor for the development of cervical cancer. HPV is highly prevalent among sexually active persons and can be easily detectable by newer technology. While Pap Smear detects the effects of HPV infection, these newer tests determine the presence of HPV infection itself; however, the HPV testing has its drawbacks. In women less than 30 years of age, prevalence of HPV is

very high but the majority of the infection reverses spontaneously. HPV testing is therefore being evaluated as a potential alternative or adjunctive to cervical cytology for early detection of cervical cancer precursors in women over 30. Many recent studies show that the LBC and HPV DNA detection appears to be superior screening methods for cervical carcinoma. Our limitation is that these tests are not readily available in our setup and even when available, most of our patients cannot afford to pay the high cost of these tests [30, 31].

Conclusion:

All women attending Gynaecology OPD, in the reproductive age group and married for more than 3 years should undergo routine Pap Smear screening irrespective of clinical symptoms.

All patients with abnormal Pap Smear reports should be referred for a Colposcopic evaluation and directed biopsy as and when necessary as the sensitivity and specificity of Colposcopy is high.

All patients with persistent symptoms and unhealthy looking cervix but with a normal Pap Smear may be considered for Colposcopic evaluation.

The sensitivity and the specificity of the conventional Pap Smear is low so it should be supplemented by other newer screening methods like LBC and HPV DNA detection from cervix, followed by Colposcopy.

Overall a well-organized screening programme, public education, awareness and follow up is needed to reduce the incidence and mortality of cervical carcinoma- the dreaded disease that takes away a woman in the prime of her life.

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Review Article

Laryngo Pharyngeal Reflux Disease: Myth or Reality?

Dr. S. Ghosh[1], Dr. A. Roychoudhury[2], Dr. B. K. Roychaudhuri[3]

Introduction:

Laryngo pharyngeal reflux disease (LPRD) has been the centre of a lot of controversies and discussions in terms of nomenclature, diagnosis and management over the last few decades. The dilemma about its existence is truly reflected by quite a number of its synonyms like: *atypical reflux, gastropharyngeal reflux, extraesophageal reflux, pharyngosupraesophageal reflux, laryngeal reflux, reflux laryngitis* and so on. [1,2] In spite of innumerable clinical trials and studies, LPRD still stands on a controversial etiopathogenesis; presents with a wide variety of symptoms, some of which are unexplainable and the treatment is still empirical and by trial in vast majority of cases. Presently LPRD has been universally accepted and established as a completely separate clinical entity.

History:

Fabricius described the gastro esophageal (GE) junction in 1618 and referred to it as "cardia" after Galen. Galen in 200 AD suggested the term "cardia" as symptoms arising from GE junction resembled those associated with cardiac ailments[1]. In the late 19th century Chevalier Jackson invented the distally illuminated esophagoscope and he believed inflammatory diseases to be the cause for esophageal strictures. Gastro Esophageal Reflux Disease (GERD) gained recognition as a clinical entity in the mid 1930s and is probably the most prevalent gastroenterological (GI) disease in clinical practice today. GI symptoms and airway diseases were associated by Bray in 1934, whereas acid related laryngeal ulcerations and granulomas were first related by Chery in 1968[3].

Definition:

The term "*reflux*" literally suggests "backflow" and is derived from two latin words "*re*" which means "back" and "*fluere*" which means "to flow"[2].

Gastro esophageal reflux (GER) refers to backflow of stomach contents into the esophagus. It is a physiological entity and up to 50 episodes of reflux can occur daily, mostly after meals.[2,4,5] GERD is a clinical entity with GER in excess, that results in tissue damage in the form of esophagitis and clinical manifestations like heartburn[2].

Laryngo pharyngeal reflux (LPR) is backflow of stomach contents into the throat i.e. laryngopharynx and LPRD is its clinical manifestation.

GERD vs LPRD:

The pathophysiology, pattern of reflux, symptomology, manifestations and management protocols are quiet different for LPRD and GERD. Only 40% patients of LPRD present with heartburn and less than 25% have documented esophagitis[2] and quiet naturally most of them tend to visit the otolaryngologist first in place of the gastroenterologists[1]. The differences between GERD and LPRD can be briefly summarized as in Table: 1.

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Manifestations	GERD	LPRD
Position of reflux	Supine & nocturnal	Supine, upright & daytime
Exposure of acid	Prolonged	Short periods with high damage
Esophageal dysmotility	Present with prolonged acid clearance	Absent
Site of dysfunction	Lower esophageal sphincter	Upper esophageal sphincter, probably both
Defense mechanism	Esophageal protective barrier is more active (carbonate production, mucosal barrier, peristalsis)	No protective mechanism in the larynx
Susceptibility to pepsin injury	Lesser than laryngeal mucosa	100 times more susceptible than esophageal mucosa

Inspite of all these differences, most patients with LPRD usually do not have GERD symptoms whereas some may present with features of both.

Diagnosis:

The symptoms of LPRD may be "chronic" or "chronic–intermittent". Some of these are truly attributable to LPRD while some may not be substantiated by any valid pathophysiological basis.

A detailed history and thorough clinical examination are usually sufficient to establish LPRD as the causative factor. Often more sophisticated laboratory tests may be necessary as an adjunct to the diagnosis. The symptoms and clinical manifestations of LPRD are summarized in Table:2.

Organ affected	Symptoms	Clinical Manifestations
Larynx	Dysphonia (Chronic/intermittent), voice break/fatigue, chronic throat clearing, Post nasal drip, chronic cough	Reflux laryngitis, subglottic stenosis, carcinoma larynx, endotracheal intubation injury, contact ulcers & granulomas, posterior glottic stenosis, arytenoid fixation, paroxysmal laryngospasm, paradoxical vocal fold movement, vocal nodules, polypoidal degeneration of vocal folds, pachydermia laryngis,
Esophagus	Dysphagia, globus symptoms	Globus pharyngis, recurrent leucoplakia
Airway	Intermittent/chronic airway obstruction, wheezing	Laryngomalacia, exacerbation of asthma
Miscellaneous		SIDS, sinusitis, adenoid hypertrophy, OSA, dental caries, apthous ulcers

A quality of life questionnaire, *reflux symptom index (RSI)*, has been suggested, which is a self-[administered nine-item outcomes instrument for evaluating LPRD. RSI has been proven to be easily administered, highly reproducible, and exhibits excellent construct and criterion-based validity. [15]

A thorough examination of the larynx with a rigid or flexible scope is mandatory to clinch the diagnosis and at times stroboscopy may also prove to be contributory to the test battery. The laryngeal findings may include: pseudo sulcus vocalis, obliteration of the ventricle, hyperemia and edema of the vocal folds, hypertrophy of the posterior commissure, posterior glottic granuloma and thick mucus.[1]

An endoscopic grading scale, *Reflux Finding Score (RFS)*, has been proposed to document the physical findings and severity of LPR. The scoring system is made up of eight findings that are graded on severity and that yield a score from 0 to 26. A score of more than seven is associated with a high likelihood of dual pH probe positivity.[14]

Over and above the standard clinical procedures, an ambulatory 24 hour double probe pH monitoring (simultaneous esophageal and pharyngeal) is considered the gold standard when the diagnosis is in question.[2,4,5,6,7,8] The double probe pH testing may also be used to evaluate drug efficacy while treating LPRD.[2] Barium esophagography or esophagoscopy are not as sensitive as pH monitoring[6,9] but may be performed to screen the esophagus for associated pathology.[6,10]

Management:

Though there are different treatment modalities for LPRD, often the protocol needs to be tailor made according to the patient's symptoms and response to treatment.[1,2]

1) **Lifestyle Modification:** This is of prime importance and consists of the following modalities —

a. Dietary habits: Patients are asked to have frequent small meals, avoid foods with spice, fat, caffeine, alcohol and tobacco. They should also avoid drugs that promote reflux like calcium channel blockers.

b. One should also maintain ideal body weight, avoid going to bed before 1½ to 2 hours of having food and elevate head end of bed by 6-8 inches.

2) **Pharmacotherapy:** H_2 antagonists and antacids may be required 3-4 times a day to combat symptoms. Proton pump inhibitors (PPI), usually with a gastric prokinetic, are the gold standard for treating LPRD and usually require a twice daily dosage pattern for a minimum duration of 6 weeks.[1,2] Usually it takes about 2-3 months time for the patients to report symptomatic improvement whereas laryngeal findings may take up to 6 months to resolve.[11,12] In some patients PPIs may have to be supplemented with H_2 antagonists.[2,13] Mild to moderate LPRD is usually managed with lifestyle modifications and H^2 antagonists whereas more severe cases require prolonged therapy with PPIs.[2]

3) **Surgical Intervention:** Patients resistant to conservative management are usually selected for Nissen's fundoplication. This surgical procedure tightens the lower esophageal sphincter by wrapping a part of the stomach around the lower esophagus.[1,2]

Conclusion:

Laryngopharyngeal reflux disease (LPRD) has emerged as one of the commonest clinical condition in routine otolarygological practice. It is a distinct clinical entity separate from GERD. There is need for an universal diagnostic and therapeutic protocol for LPRD. As per current evidence, majority of the cases may be diagnosed on clinical grounds and the treatment consists of pharmacotherapy, supported by lifestyle modifications.

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Review Article

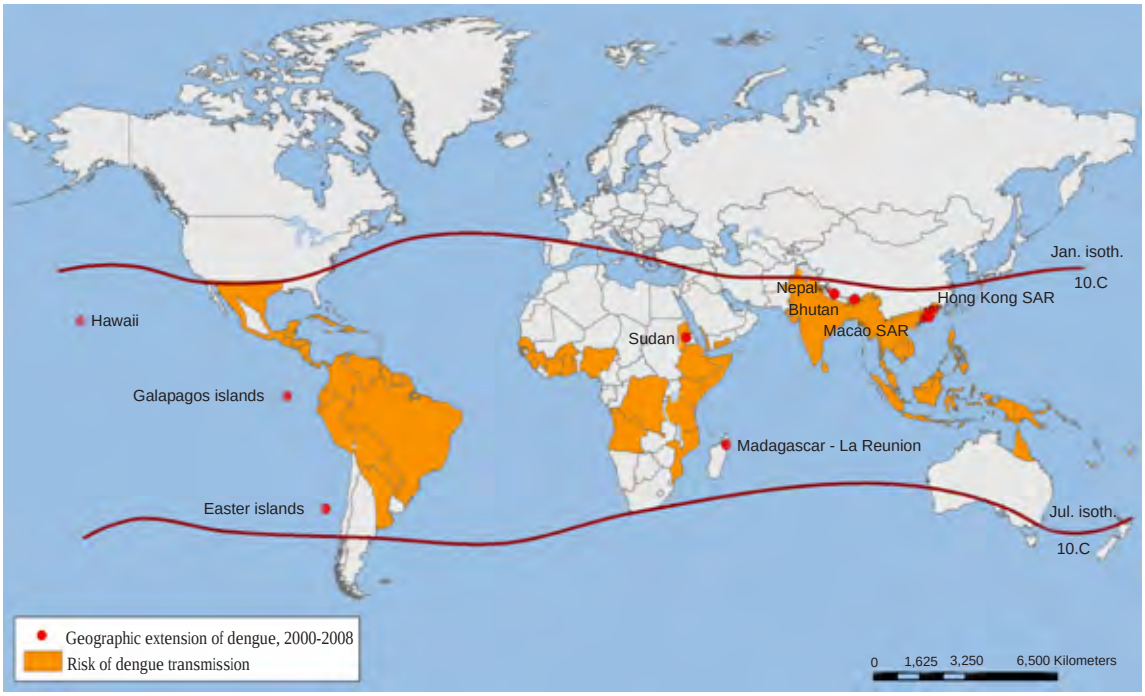
Dengue: Management Guidelines

Dr. Pallavi[1], Dr. Kaushik Sur[2]

Introduction:

Dengue is the most rapidly spreading mosquito-borne viral disease in the world. It is caused by the Dengue virus having four different serotypes and transmitted principally by the mosquito *Aedes aegypti*. Sudden rise in density and geographic distribution of the Aedes aegypti and a large transmission has led to an exponential increase in the emergence and re-emergence of Dengue Fever (DF) and Dengue haemorrhagic fever (DHF).

Figure Showing Countries & Areas at Risk of Dengue Transmission:



Source: Dengue Net, WHO, 2008. www.abc.net.au/rn/backgroundbriefing/documents/20100221_map.pdf

New Classification:

As the magnitude of the problem is increasing day by day, updated knowledge and specific guidelines for diagnosis and treatment are needed. The new guidelines of World Health organization (WHO), published in 2009, provides practical information on the diagnosis, treatment, prevention and control of Dengue fever. As per the new guidelines this disease is now classified

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into three categories:

1. Dengue
2. Dengue with warning signs
3. Severe Dengue

The clinical course of the disease is divided in three phases

1. Febrile
2. Critical
3. Recovery

Table - 1. Classification of Dengue Case and Levels of Severity

DENGUE ± WARNING SIGNS? SEVERE DENGUE

CRITERIA FOR DENGUE	WARNING SIGNS*	CRITERIA FOR SEVERE DENGUE
PROBABLE DENGUE Live in /travel to dengue endemic area. Fever and 2 of the following criteria: l Nausea, vomiting l Rash l Aches and pain l Tourniquet test positive l Any warning sign Laboratory Confirmed Dengue	l Abdominal pain or tenderness. l Persistent vomiting l Clinical fluid accumulation. l Mucosal bleed l Lethargy, restlessness l Liver enlargement >2cm l Lab increase in HCT concurrent with rapid decrease in platelet count . *requiring strict observation and medical treatment	Severe plasma leakage Leading to: l Shock (DSS) l Fluid accumulation with respiratory distress. Severe bleeding As evaluated by clinician Severe organ involvement l Liver : AST or ALT >= 1000 l CNS : impaired consciousness l Heart and other organs

Adapted from WHO guidelines for Dengue 2009

Management of Dengue:

Group A

DENGUE WITHOUT WARNING SIGNS

(*May be sent home*)

Patients not having warning signs and who are able:

- l To tolerate adequate volumes of oral fluids.
- l To pass urine at least once every 6 hours

Laboratory Tests:

- l Full blood count (FBC)
- l Haematocrit (HCT)- stable HCT can be sent home

Treatment Advice:

- l Adequate bed rest, fluid intake and paracetamol.
- l Avoid aspirin and other non-steroidal anti-inflammatory agents (NSAIDs), or steroids.
- l Antibiotics are not necessary.

Monitoring:

Daily review for disease progression:

1. Decreasing white blood cell count, defervescence and development of warning signs (until out of critical period).

Group B

Dengue with warning signs *(Referred for in-hospital care)*

These patients are likely to pass in the critical phase and need hospitalization.

Laboratory Tests:

1. Full blood count (FBC)
2. Haematocrit (HCT)

Treatment Advice:

Encourage oral fluids.

If not tolerated, start maintenance intravenous fluid therapy 0.9% saline or Ringer's Lactate:

In presence of warning signs or deteriorating clinical status- treat with I.V fluid, 5- 7 ml /kg/hr for 1-2 hours till hemodynamic stabilization, then taper to 3-5 ml/kg/hr for 2-4 hrs and then to 2-3 ml /kg/hr or less according to the clinical response.

Monitoring:

1. Temperature and Intake-output chart, warning signs
2. HCT, white blood cell and platelets.

Group C

Severe dengue *(Require emergency treatment)*

Severe dengue should be considered if the patient is from an endemic area of dengue risk having fever for 2–7 days plus any of the following:

Evidence of plasma leakage, such as:

- high or progressively rising haematocrit;
- pleural effusion or ascites;
- circulatory compromise or shock (tachycardia, cold extremities, capillary refill time > 3 seconds, weak or undetectable pulse, narrow pulse pressure

OR

unrecordable blood pressure in late shock).

Significant Bleeding:

Altered level of consciousness (lethargy or restlessness, coma, convulsions).

Severe gastrointestinal involvement (persistent vomiting, intense abdominal pain, jaundice)

Multi-organ dysfunction (acute liver failure, acute renal failure, encephalopathy or encephalitis, or other unusual manifestations, cardiomyopathy) or other unusual manifestations.

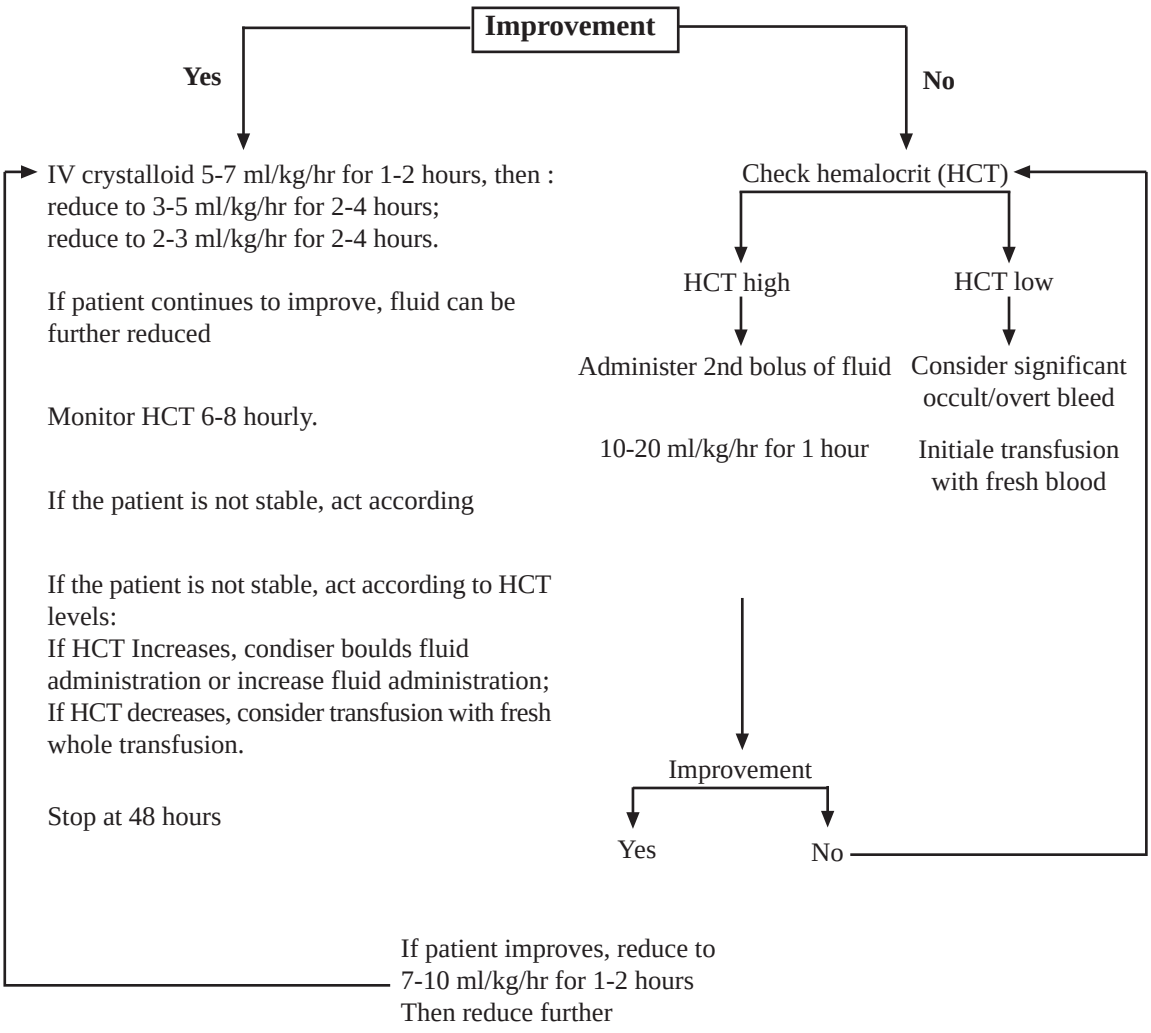
Emergency Treatment Protocol and Monitoring of Compensated Hypotension:

Algorithm for Fluid Management in Compensated Shock Compensated Shock

(Systolic pressure maintained but has signs of reduced Perfusion)

Fluid resuscitation with isotonic crystalloid

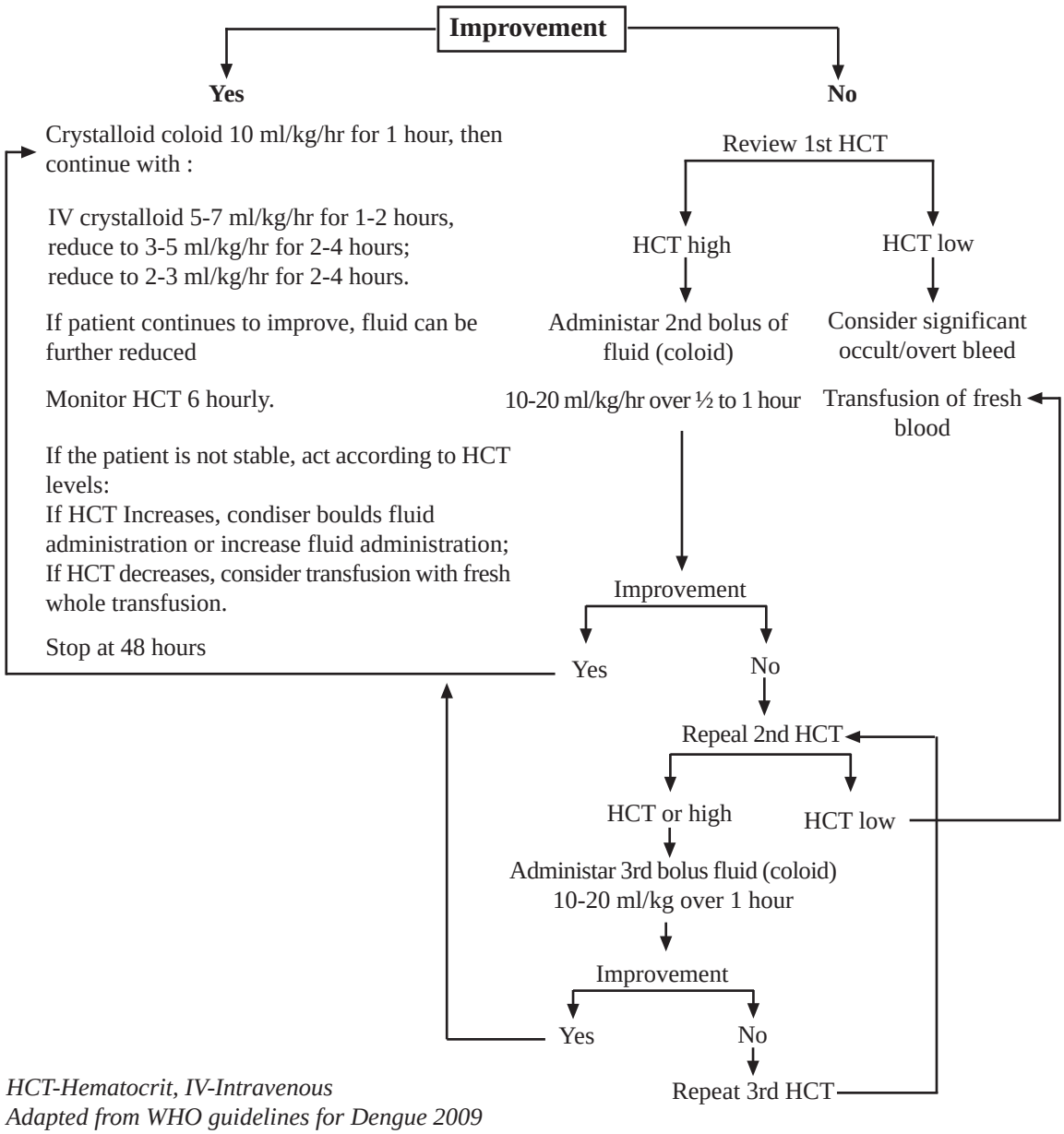
5 - 10 ml/kg/hr over 1 hour



HCT-Hematocrit, Iv-Intravenous
Adapted from WHO guidelines for Dengue 2009

Management of Decompensated Shock:

Algorithm for Fluid Management in Hypotensive Shock



HCT-Hematocrit, IV-Intravenous
Adapted from WHO guidelines for Dengue 2009

Management of Complications:

1. Major bleeds : 10 ml /kg of PRBC or 20 ml/ kg of whole blood should be transfused.
2. Platelet transfusion: In case of significant bleed with thrombocytopenia, platelet should be transfused.
3. Fluid overload: Oxygen supplementation has to be given. If respiratory distress is present, Inj. Frusemide 0.1 -0.5 mg/kg/dose once or twice daily may be used.
4. Management of dyselectrolytemia, hypoglycaemia.
5. Vasopressors and inotropes : Used in severe fluid unresponsive dengue shock .

Discharge Criteria:

(All of the following conditions must be present)

Clinical: No fever for 48 hours. Improvement in clinical status (general well-being, appetite, haemodynamic status, urine output, no respiratory distress).

Laboratory: Increasing trend of platelet count. Stable haematocrit without intravenous fluids.

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Review Article

Perinatal & Neonatal Outcome of Mothers Having Pregnancy Induced Hypertension

Dr. Debashis Mondal[1], Dr. Soumitra Masani[2], Dr. Asha Mukherjee[3]

Introduction:

Pregnancy Induced Hypertension (PIH) is an important cause of maternal morbidity & mortality and it leads to adverse perinatal & neonatal outcome. About 5-8% of all pregnancies worldwide anually are complicated by hypertension acquired during pregnancy. This is a significant disease burden. Approximately 70% of women who are diagnosed with hypertension during pregnancy will have gestational hypertension or preeclampsia. Hypertensive disorders are one of the most common medical complications in pregnancy, its pathophysiology is complex and incompletely understood, its diagnosis may be delayed at times and there is no effective treatment. Antenatal care involves a difficult balance between the risks for women to continue pregnancy and those for the baby's early birth and subsequent complications.[1,2]

Risk factors for Pregnancy Induced Hypertension are young age, black race, obesity, past history of PIH, family history of PIH, nulliparity, multifoetal gestation and co-existent diabetes mellitus, thrombophilia and renal disorder. Maternal complications of Pregnancy Induced Hypertension are abruptio placentae, hepatic failure, renal failure, cerebral oedema manifested as seizures and HELLP Syndrome (Hemolysis, Elevated liver enzymes and Low platelet count).[3]

Perinatal mortality rates range from 59 in 1,000 in developed countries to more than 300 in 1,000 in low-income countries. Rates greater than 200 in 1,000 are usually reported in severe PIH (at 24 to 34 weeks of gestation). Current stillbirth rates in PIH range between 9 and 51 in 1,000 births. Perinatal & neonatal mortality is increased due to IUGR or asphyxia, prematurity, uteroplacental insufficiency, placental abruption. In addition, the influence of maternal disease severity and onset of hypertension, increased proteinuria or the presence of HELLP syndrome on perinatal & neonatal outcome has been emphasized by several studies. Increased NICU admission, Respiratory distress syndrome, Broncho-pulmonary Dysplasia, Thrombo-cytopenia, Neutropenia and increased susceptibility to Neonatal sepsis, Necrotizing Enterocolitis & Intraventricular hemorrhage/ Periventricular leukomalacia are other proposed perinatal & neonatal complications of PIH.[1,4].

Back Ground:

Hypertensive disorders are one of the most common medical complications in pregnancy and major contributor to maternal, fetal and neonatal morbidity and mortality. The most widely accepted definition and classification of hypertensive disorders of pregnancy was proposed by the National High Blood Pressure Education Program (NHBPEP) Working Group on High Blood Pressure in Pregnancy (2001), which used a blood pressure of 140/90 mm Hg or higher on two separate occasions at least 4 hours apart as the diagnostic criterion for hypertension. The NHBPEP defined four hypertension categories in pregnancy: chronic

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hypertension, gestational hypertension, Pre-eclampsia (PE) and Pre-eclampsia superimposed on chronic hypertension.[5] Pre-eclampsia is characterized as severe according to hypertension degree (arterial pressure 160/110mmHg on two occasions _6 hours apart); proteinuria (5 g/24 hours or 3+ in two urine samples); or any of the following: cerebral/visual disturbances, abdominal pain, abnormal liver function, oliguria, pulmonary edema, thrombocytopenia, or fetal growth restriction.[6]

Criteria for Diagnosis of Preeclampsia (5):

The National Institute of Health (NIH) working group on hypertension in pregnancy has classified hypertensive disorders of pregnancy in four main categories:

1. **Chronic Hypertension:**

   Systolic blood pressure > 140 mmHg.

   Diastolic blood pressure > 90 mmHg; occurs prior to pregnancy or prior to the 20th week of gestation.

2. **Gestational Hypertension:**

   It occurs when blood pressure is elevated after 20 weeks of gestation with no prior history of hypertension and proteinuria is absent.

3. **Preeclampsia (PE):**

   Systolic blood pressure > 140 mmHg; Diastolic blood pressure > 90 mmHg with proteinuria.

4. **Preeclampsia Superimposed on Chronic Hypertension:**

   It is recognized to impart a more severe course and higher incidence of maternal and fetal complications than preeclampsia alone.

Severe Preeclampsia/ Eclampsia is Confirmed When Any of the Following Criteria is Present:

- Systolic blood pressure > 160 mmHg .
- Diastolic blood pressure > 110 mmHg (on two occasions at least 6 hours apart while the patient is on bed rest).
- Proteinuria of 5000mg (5g) or higher on a 24-hour urine collection or at least 3+ on two random urine samples collected at least 4 hours apart.
- Oliguria < 500 mL urine output in 24 hours.
- Cerebral or visual functional disturbances (CNS irritability) .
- Pulmonary edema or cyanosis (not due to excessive intravenous volume replacement).
- Epigastric or right-upper quadrant abdominal pain .
- Impaired liver function on laboratory analysis (elevated AST/SGOT, ALT/SGPT, or LDH)
- Thrombocytopenia (platelet count < 150,000/uL).
- Fetal growth restriction.[5]

The major perinatal consequences are high perinatal mortality rates and increased neonatal morbidity due to prematurity, IUGR[7,8]. In addition, the influence of maternal disease severity such as degree of hypertension, increased proteinuria or the presence of HELLP syndrome, abruption placentae, premature onset of labour on perinatal outcome has been emphasized. It has been demonstrated in several studies that the perinatal outcome could be modulated by the obstetric management of severe PE and also by anticipation and early diagnosis of perinatal ailment and their prompt and appropriate management. [9,10]

Several studies elucidated correlation between PIH and its perinatal outcome. Naeye RL, Friedman EA et al. (1979), pioneered the study to identify causes of perinatal death associated with gestational hypertension and proteinuria[11]. The first documented Indian study was done by Deorari AK et al. to demonstrate perinatal & neonatal outcome in hypertensive disease of pregnancy and was published in 1985 Indian Pediatrics journal [12].

W Szymonowicz et al (1987), from Queen Victoria Medical Centre, Melbourne, Australia, studied the effect of severe pre-eclampsia on the outcome of infants of very low birth weight in a prospective case control study design of 35 pairs of infants of comparable gestation. Significantly more infants were delivered before term by induction of labour or by caesarean section in the group with pre-eclampsia and these babies tended to be smaller and had a higher incidence of hyaline membrane disease, patent ductus arteriosus, pulmonary air leak and hypotension. They also required more intensive treatment with oxygen and mechanical ventilation. [13]

J. Nadkarni etal. (2000), From the Department of Pediatrics, Mahatma Gandhi Memorial Medical College, Indore, showed Hypertensive disorders of pregnancy predisposes women to acute or chronic uteroplacental insufficiency, resulting in ante or intrapartum anoxia that may lead to fetal death, intrauterine growth retardation and preterm delivery. The percentage of preterm and low birth weight babies was high in their study. Prematurity was the most important factor responsible for increased perinatal & neonatal morbidity and mortality. Birth asphyxia was the commonest neonatal complication.[14]

Attiya Ayaz etal. (2009), from J Ayub Med College Abbottabad, showed a perinatal mortality of 328 neonates per 1000 total births, major cause being still births and intrauterine death (IUD). Various complications seen were IUD, low APGAR score, low birth weight, intrauterine growth restriction (IUGR) and increased need for admission to Neonatal Intensive Care Unit (NICU).[15]

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CME Article

Migraine: Current Trends in Diagnosis & Management

Dr. Arabinda Mukherjee

Abstract:

Migraine is a recurrent headache disorder that is one of the most common complaints in medicine. Migraine is the commonest of all "Primary headache disorder". In the United States, more than 30 million people have 1 or more migraine headaches per year. Approximately 75% of all persons who experience migraines are women. The term migraine is derived from the Greek word *hemikrania.*

Migraine was previously considered a vascular phenomenon that resulted from intracranial vasoconstriction followed by rebound vasodilation. Currently, however, the neurovascular theory describes migraine as primarily a neurogenic process with secondary changes in cerebral perfusion. Approximately 70% of patients have a first-degree relative with a history of migraine. In addition, environmental and behavioral factors may precipitate migraine attacks in persons with a predisposition to migraine.

The classic migraine episode is characterized by unilateral head pain preceded by various visual, sensory, motor symptoms, collectively known as an aura. Most commonly, the aura consists of visual manifestations such as scotomas or visual scintillations (eg, bright zigzag lines). In practice, however, migraine headaches may be unilateral or bilateral and may occur with or without an aura. In the current International Headache Society (IHS) categorization, the headache previously described as classic migraine is now known as migraine with aura, and that described as common migraine is now termed migraine without aura. Migraines without aura are the most common, accounting for more than 80% of all migraines.

The diagnosis of migraine is clinical in nature, based on criteria established by the International Headache Society. A full neurologic examination should be performed during the first visit; the findings are usually normal. Neuroimaging is not necessary in a typical case.

Migraine treatment involves acute (abortive) and preventive (prophylactic) therapy. Patients with frequent attacks usually require both. Measures directed toward reducing migraine triggers are also generally advisable. Preventive treatment, which is given even in the absence of a headache, aims to reduce the frequency and severity of the migraine attack, make acute attacks more responsive to abortive therapy, and perhaps also improve the patient's quality of life. [1-8]

Migraine Classification [9-12]:

The second edition of the International Classification of Headache Disorders lists the following types of migraine:

1.1 Migraine without aura

1.2 Migraine with aura:

Typical aura with migraine headache

Typical aura with nonmigraine headache

Typical aura without headache

MD, DM (Neuro), Prof. of Neurology, VIMS Kolkata

Familial hemiplegic migraine

Sporadic hemiplegic migraine

Basilar artery migraine

1.3 Childhood periodic syndromes commonly precursors of migraine:

Cyclical vomiting

Abdominal migraine

Benign paroxysmal vertigo of childhood

1.4 Retinal migraine

1.5 Complicated migraine:

Chronic migraine

Status migraine

Migrainous infarction

Migraine triggered seizures

1.6 Probable migraine

Migraines typically present with recurrent severe headache associated with nausea, vomiting photophobia and phonophobia either singly or in combinations. An aura only occurs in a small percentage of people. The severity of the pain, duration of the headache, and frequency of attacks is variable. A migraine lasting more than 72 hours is termed status migrainosus. The four possible phases to a migraine attack are listed below, although not all the phases are necessarily experienced. Additionally, the symptoms experienced can vary from one migraine attack to another in the same person.

Phases of Migraine:

1. The prodrome which occurs hours or days before the headache
2. The aura, which immediately precedes the headache
3. The pain phase also known as headache phase
4. The postdrome

Prodrome:

Prodromal symptoms occur in 40–60% of those with migraines. This phase may consist of altered mood, irritability, excessive sleepiness, craving for certain food. This phase usually precede the headache phase of the migraine attack by several hours or days, and experience teaches the patient or observant family how to detect a migraine attack is near.

Aura:

About 15% of people experience migraines with an aura. This aura comprises focal neurological phenomena that precede or accompany the attack. They appear gradually over five to 20 minutes and generally last fewer than 60 minutes. The headache phase of the migraine attack usually begins within 60 minutes of the end of the aura phase, but it is sometimes delayed up to several hours, and it can be missing entirely. The pain may also begin before the aura has completely subsided. Symptoms of migraine aura can be visual, sensory or motor in nature. Visual aura is the most common and can occur without any headache. There is a disturbance of vision consisting often of unformed flashes of white and/or black or rarely of multicolored lights or formations of dazzling zigzag lines (scintillating scotoma) Some patients complain of blurred or shimmering or cloudy vision, as though they were looking at an area above a heated surface, looking through thick or smoked glass or in some cases, tunnel vision or hemianopsia. The somatosensory aura of migraine may consist of digito-lingual or cheiro-oral paraesthesia. Other symptoms of the aura phase can include auditory, gustatory or olfactory hallucinations, temporary dysphasia, tingling or numbness of the face and extremities, and hypersensitivity to touch.

Pain:

The typical migraine headache is unilateral, throbbing, and of moderate to severe intensity and can be aggravated by physical activity. The pain may be bilateral at the onset or start on one side and become generalized, and may occur primarily on one side or alternate sides from one attack to the next. The onset is usually gradual. The pain peaks and then subsides and usually lasts 2 to 72 hours in adults and 1 to 48 hours in children. The frequency of attacks is extremely variable, from a few in a lifetime to several a week, and the average sufferer experiences one to three headaches a month. The pain of migraine is invariably accompanied by other features. Nausea occurs in almost 90 percent of patients, and vomiting occurs in about one third of patients. Many patients experience sensory hyperexcitability manifested by photophobia, phonophobia or osmophobia and seek a dark and quiet room. Blurred vision, delirium, nasal stuffiness, diarrhea, tinnitus, pallor or sweating may be noted during the headache phase. There may be scalp tenderness, prominence of a vein or artery in the temple, or stiffness and tenderness of the neck (allodynia). Impairment of concentration and mood are common.

Postdrome:

The effects of migraine may persist for some days after the main headache has ended. Many sufferers report a sore feeling in the area where the migraine was, and some report impaired thinking for a few days after the headache has passed. The patient may feel tired and may have cognitive difficulties, gastrointestinal symptoms, mood changes, and weakness.

Migraine Variants:

Abdominal Migraine:

The diagnosis of abdominal migraines is controversial. There is some evidence that recurrent episodes of undiagnosed abdominal pain which occur in the absence of a headache may be a type of migraine. These episodes of pain may or may not follow a migraine like prodrome and typically last minutes to hours. They often occur in those with either a personal or family history of typical migraines.

Vestibular Migraine:

Vestibular migraine may affect both adult and children. The diagnosis of vestibular migraine to be suspected when episodic vestibular symptoms are associated with migraine headache. This condition responds to migraine medications.

Ophthalmoplegic Migraine: Migraine headache associated with reversible ophthalmoplegia.

Pathophysiology of Migraine [14, 15]:

The pathophysiology of migraine is complex and involves a number of factors like genetic, electrophysiological, chemical and neurovascular changes**.**

Genetics:

Currently it is established that the propensity to experience recurrent migraine pain with aura is mostly genetically determined. It is clear from clinical experience that many of the 1st degree relatives of migraine patients also suffer from migraine. The genetic locus of familial hemiplegic migraine (FHM) has been attributed to chromosome 19p13.

Electrophysioloical Depolarization:

The phenomenon known as cortical spreading depression (CSD) which is associated with the

aura of migraine has been theorized as a possible cause of migraines. In cortical spreading depression (CSD) neurogenic activity is initially activated, then depressed over an area of the cerebral cortex followed by decrease in blood flow in the cerebral cortex. CSD propagates from occipital cortex anteriorly. This situation has been suggested to result in the activation of trigeminal vascular system with release of inflammatory mediators like substance P, neurokinin P and calcitonin gene related peptides (CGRP) leading to neurogenic inflammation and pain.

Vascular:

Vascular theory postulates that migraine is due to constriction of the intracranial vessels resulting in aura phase followed by dilatation of extracranial vessels which cause headache. However, to presume that migraine is generated from within the central nervous system, based on the available evidence is not always correct. The emerging evidence would suggest that just as alterations in neuronal activity can lead to downstream effects on the cerebral blood vessel, so too can changes within endothelial cells or vascular smooth muscle lead to downstream alterations in neuronal activity. Therefore, there are likely patients, and/or at least attacks in certain patients, where primarily vascular mechanisms predominate. It is probably because both vascular and neurogenic mechanisms for migraine exist and are important.

Triggers:

Migraines may be induced by triggers. Many things have been labeled as triggers, however the significance of these relationships are uncertain. Common triggers quoted are stress, hunger, and fatigue. Migraines are more likely to occur around menstruation. Other hormonal influences also play a role. Other common triggers include change of temperature, strong smell, heat, fasting, certain food. The classical triggers like cheese, Chinese food and chocolates are rarely found in Indian population.

Diagnosis:

The diagnosis of migraine according to the International headache society can be made according to the following criteria.[7]

Table - 1

ICHD 2 Diagnostic Criteria for Migraine Without Aura

Repeated episodes of headache (4-72 hours)<15 days/month with one of the following features:
Headache has at least any 2 of Unilateral Throbbing Worsened on movement Moderate to severe intensity
During headache one of the following : Photophobia/phonophobia Nausea /vomiting
Not attributable to any other cause

Most migraine headache fulfill the ICHD 2 criteria described above. However in pediatric patients the duration of headache may be shorter and less than 1 hour, the associated symptoms are different and include recurrent abdominal pain or cyclical vomiting.

Table - 2

ICHD 2 Diagnostic Criteria for Migraine with Aura

A. At least 2 attacks fulfilling criteria below
B. At least 3 of the following present:
One aura symptom developing over >4 minutes
No aura last for more than 60 minutes
Headache follows aura within 60 minutes of disappearance of aura
No evidence of organic disease or suggestions for a secondary cause

Neuroimaging in Headache:

Migraine is a clinical diagnosis. Neuroimaging is not routinely indicated for diagnosis. However imaging should be considered in following situations

A. Temporal and Headache Profile:

The first or worst headache

Subacute headache with increasing frequency and severity

A progressive headache or NDPH

Headache always on same side

Headache not responding to treatment

B. Demographics:

New onset headache in patients of cancer or HIV positive

New onset headache after 50

Patients with headache who have seizures

C. Associated symptoms and signs:

Headache with fever, stiff neck

Headache with focal neurological signs

Headache with papilloedema

Prevalence and Incidence of Migraine:

The published migraine prevalence varies wildly. Before puberty, prevalence of migraine is higher in boys than girls. As puberty approaches girls have higher prevalence. The peak prevalence is between 25-55 years. In American migraine prevention and prevalence study the prevalence of migraine was 18% in women an 6 % in men. Worldwide, migraines affect more than 10% of people. In the United States, about 6% of men and 18% of women get a migraine in a given year, with a lifetime risk of about 18% and 43% respectively. In Europe, migraines affect 12–28% of people at some point in their lives. Based on the results of a number of studies, one-year prevalence of migraine ranges from 6–15% in adult men and from 14–35% in adult women. After menopause, attacks in women tend to decline dramatically, so that in the over 70s, approximately equal numbers of males and females are sufferers, with prevalence returning to around 5%. At all ages, migraine without aura is more common than migraine with aura, with a ratio of between 1.5 and 2.0:1. The studies from India reflects that the prevalence and incidence are similar to western countries.

Management of Migraine [16-21]:

There are three main aspects of treatment:

Avoidance of triggers

Treatment of acute attacks

Migraine prophylaxis

Treatment of Acute Headaches:

Pharmacological agents for treatment of acute migraine attacks include :

Analgesics

NSAID

Ergot preparations

Triptans

Medications are more effective if used earlier in an attack. The frequent use of medications may, however, result in medication overuse headache in which the headaches become more severe and more frequent. These may occur with triptans ergotamines and analgesics, especially narcotic analgesics.

Analgesics:

A number of analgesics are effective for treating migraines including:

- Paracetamol/acetaminophen, either alone or in combination with metcloprpamide is effective for migraines.
- Simple analgesics combined with caffeine may help. Even by itself, caffeine can be useful during an attack, A 1000Êmg dose of aspirin could relieve moderate to severe migraine pain, with similar effectiveness to sumatriptan.

NSAIDS:

Ibuprofen provides effective pain relief in about half of people. Naproxen can abort about one third of migraine attacks, which was 5% less than the benefit of sumatriptan.

Triptans:

Triptans such as sumatriptans are effective for both pain and nausea in up to 75% of people. The different forms available include oral, injection, and oral dissolving tablets. Most side effects are mild, such as flushing; however, rare cases of myocardial ischemia have occurred. They are not addictive, but may cause medication overuse headaches if used more than 10 days per month. Several other triptans have been approved for acute migraine headache which includes rizatriptan, zolmitriptan, eletriptan, almotriptan. In general all of them have equal efficacy but individual patients may benefit more from one triptan than another. The choice of triptans for individual patients therefore depends on acceptance, medical co morbidities and preferred route of administration.

Ergotamines:

Ergotamine and dihydroergotamine (DHE) are older medications still prescribed for migraines, the latter in nasal spray and injectable forms They were the primary drugs available to abort a migraine prior to the triptans, and are much less expensive than triptans. DHE is recommended for treatment of status migraine but it is not available in India.

Abortive Therapy for Migraine

Specific Treatment

Drug	Dose	Route
Ergot Alkaloids		
Ergotamine	1-2 mg/d; max-6 g/d	Oral
Dihydroergotamine	0.75-1 mg	SC
5-HT receptor agonists		
Sumatriptan	25 - 300 mg 6 mg.	Orally SC
Rizatriptan Zolmitriptan	5mg/10mg 2.5/5mg	Oral, ODT Oral, intranasal

Migraine Prophylaxis:

Preventive treatments of migraines can be an important part of migraine management and include: medications, migraine surgery, nutritional supplements, lifestyle alterations, such as increased exercise, and avoidance of migraine triggers. The goals of preventive therapy are to reduce the frequency, painfulness, and/or duration of migraines, and to increase the effectiveness of abortive therapy. Another reason to pursue these goals is to avoid medication overuse headache (MOH). This is a common problem and can result in chronic daily headache. Many of the preventive treatments are quite effective.

Medication:

Preventive migraine drugs are considered effective if they reduce the frequency or severity of migraine attacks by at least 50%. The most effective preventive medications include: beta-blockers, flunarizine, valproate, topiramate and amitryptiline. 5HT antagonists, gabapentine, cyproheptadine are also effective. Recently botulinum toxin injection has been approved for migraine prevention. Other agents which have been used include riboflavin, co enzyme Q, feverfew.

Table - 3

Indications for Prophylactic Pharmacotherapy in Migraine:

More than 4 attacks per month

Migraine attacks with no satisfactory response to acute medicines

Intolerable adverse effect with acute attack treatment

Migraine attacks lasting longer than 48 hours

Regular rebound headache after triptan administration

Migraine attacks that are perceived intolerable by the patient

Complicated migraine (eg FHM)

History of migrainous infarct

Patient preference

The aim of preventive therapy are :

A. To reduce the use of acute medication
B. To sensitize brain to effect acute medication
C. To improve quality of life in migraine patients
D. To prevent complications like development of chronic migraine or migrainous infarction

The available migraine preventing drugs are classified in various ways. Some are based on mechanisms of action while others are classified according to efficacy. The major migraine preventive agents belong to four groups: beta-blockers, calcium channel antagonist, anti epileptics and antidepressants.

Table - 4

Classification of Drugs Effective for Migraine Prophylaxis:

A. High efficacy: Low to Moderate Adverse Effects (AE)

Propanolol, timolol, valproate, topiramate, flunarizine, amitryptiline

B. Low Efficacy: Low to Moderate AE

Atenolol, metoprolol, nadolol

Verapamil

Gabapentine

Pizotifen

Vitamin B2, magnesium, feverfew

Nsaid

C. Unproven Efficacy: Low to Moderate AE

Venlaflaxin

Doxepin

Sertaline

Paroxetine

Fluvoxamine

D. Proved Not Effective or Low Efficacy:

Carbamazepin

Lmotrigine

Pindolol

Nefidipine

Indomethacin

For individuals the selection of a preventive medication depends on various factors. Since many of the migraine patients have co morbidities the issue should be properly addressed before prescribing. For example beta blockers should be avoided in patients with COPD. heart blocks, diabetes and depression as beta blockers can aggravate them. For obese patients valproates and flunarizine are not suitable.

Some of migraine preventive drugs can treat the co morbidities as well. Ideal example is valproic acid which is the drug of choice when migraine is associated with epilepsy. The general rule is to start with one drug with low dose and gradually build up the dose till "success" is achieved or intolerable side effect(s) appear. We consider success when there is >50% reduction in headache severity and frequency. A three months trial is required before discarding a drug as not effective. If one drug is not effective it is recommended to try another first line drug preferably with a different mechanism of action.

Migraine Prophylaxis: Choice of Drugs

DRUGS	INDICATIONS
Flunarizine	Non-obese, sleep disturbance, vertigo children
Propanolol	Trenor, angina, pregnancy, children, hypertension
Divalproate	Seizure, MDP, non obese
Topiramate	Seizure, obese
Amitryptiline	Seizure, obese

Nonpharmlogical Agents:

Medical devices, such as biofeedback and neurostimulators, have some advantages in the migraine treatment, mainly when common anti migraine medication is contraindicated or in case of medication over use. Biofeedback helps people to be conscious of some physiologic parameters to control them and try to relax and may be efficient for migraine treatment. Neurostimulation uses implantable neuro-stimulators similar to pacemakers for the treatment of intractable chronic migraines with encouraging results for severe cases.

Migraine Diary:

A migraine diary allows the assessment of headache characteristics, to differentiate between migraine and tension-type headache and to record the use and efficacy of acute medication. A diary also helps to analyze the relationship between migraine and menstruation. Finally, the diary can help to identify trigger factors. A trigger may occur up to 24 hours prior to the onset of symptoms; the majority of migraines, though, are not caused by identifiable triggers.

Prognosis:

Though migraine has been in general considered as benign several serios neurological complications may occur in a small percentage of cases. The risk of stroke may be increased two- to three-fold in migraine sufferers. Young adults and women using hormonal contraception appear to be at particular risk involved . Women who experience auras have been found to have twice the risk of strokes and heart attacks over non aura migraine sufferers and women who do not have migraines. Death from cardiovascular causes was higher in people with migraine with aura in a study, but more research is needed to confirm this. Studies have demonstrated higher incidence of cerebral micro infarcts in patients with frequent migraine. Silent posterior circulation infarcts is another documented complication. The other complications include conversion of episodic migraine to chronic migraine which occur in about 33% patients and precipitation of seizures by migraine attacks. [22-24].

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Institute News

16th Micro-Ear Endoscopic Sinus Surgery workshop, CME and Cadaver direction Course held on 20th - 23rd March, 2014, organised by the Dept. of ENT & Head & Neck Surgery, RKMSP, VIMS.

MCI inspection for recognition of one permitted seat in Orthopedics and two in Paediatrics were held on 26th & 29th April 2014, respectively.

The results of Post Graduate degree and diploma examination for this year have been declared recently. All of the candidates, appearing from this institute were declared Passed, except two in MS (Obs & Gynae), who will sit for the supplementary examination sometimes in this year.

Achievements of Dr. Ujjwal K. Debnath in 2014

1. Concluded successfully "Operation Straight Spine" with US and UK team at RKMSP between 10-22nd February 2014.
2. Invited by Bangladesh Spine Society for presenting lectures as foreign faculty on "Current Concepts in management of Early Onset Scoliosis".
3. Presented paper on Operation Straight Spine in RKMSP (which has completed 8 years successfully) in "Scoliosis Resarch Society USA".
4. Elected executive member for "Indian Orthobiologics Society".

Newer Surgical Technique

Single Incision Laparoscopic Surgery (SILS) Without Any Special Instrument

Dr. Kalyan Ashis Mukherjee

Single incision laparoscopic surgery (SILS) is a newer surgical technique which provides cosmetically superior result. These procedures are costly as they require special instruments. But here we are doing SILS with normal laparoscopic instruments thereby avoiding the cost issue. Here at Ramakrishna Mission Seva Pratisthan we are mainly performing SILS-appendectomy.

Steps:

General Anesthesia, Supine position:

Port Position – A 2cm vertical intra umbilical incision made. Three 5mm ports are inserted. Centre one for camera and others for hand instrument. (Pic. 1)

Appendix is dissected through traditional way. Ultrasonic devise is necessary when there was inflamed retro ceacal appendix.

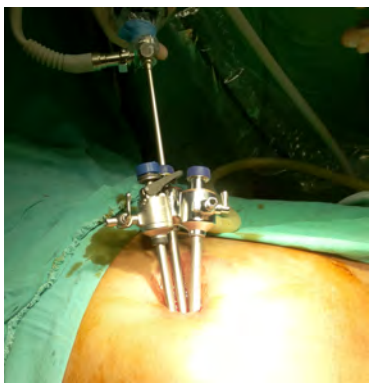
Appendicular artery is coagulated either by bipolar cauterisation or by ultrasonic device

Base of appendix is isolated and ligated by preformed catgut loop knot

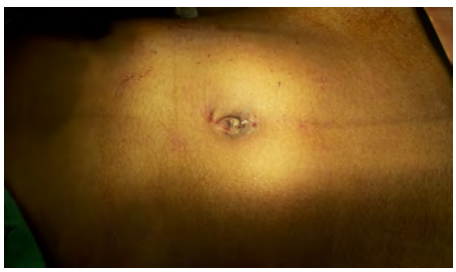
Appendix is cut and removed, if it is grossly swollen (not possible to insert inside the port) then it is better to put it into an endo bag and remove.

All three port site are closed with delayed absorbable suture and skin closed meticulously with subcutaneous suture. (Pic. 2)

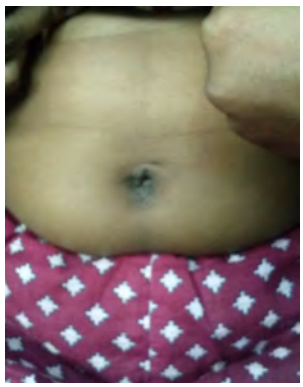
Patient need to follow up after 1 week (Pic. 3) for scar condition and after 6 months for port site hernia



Port Position



Immediate Post Operation



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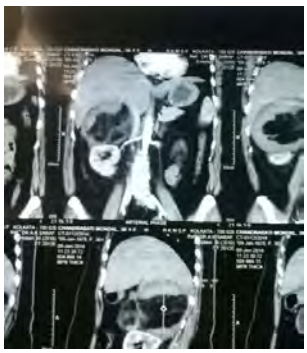
Consultant Surgeon

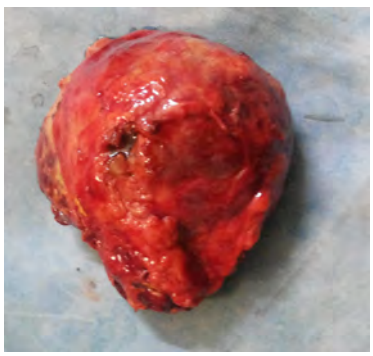
Case Report

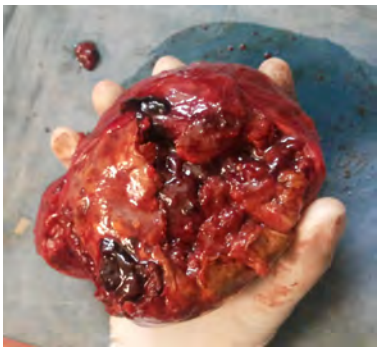
Rare Case of A Large Benign Adrenal Myolipoma

Dr. Kalyan Ashis Mukherjee[1], Dr. A. K. Saraf[2], Dr. Mahendra Gupta[3]

A 36yrs housewife from Kolkata was presented to our Hospital with vague abdominal pain at right ride of abdomen for 5 months without any associated symptoms. Physical examination was within normal limits, abdomen was soft, non tender, no mass palpable. Patient was investigated, her USG showed retroperitoneal lump, CECT abdomen showed-a non homogenous right adrenal mass of 10x9x8 cm compressing inferior vena cava. (Pic. 1) Further biochemical investigations excludes functional adrenal tumour. Patient was planned for open right adrenalectomy under GA. Surgery was performed through a midline incision from xiphoid inferiorly circumscribing the umbilicus, tumour was separated from IVC, and vascular branches were ligated. Single globular mass of 12x10x8 cm was removed by mobilising adrenal gland medial to lateral. (Pic. 2) Post operative period was uneventful except initial hypo-peristalsis for 3 days. Histopathological examination of the mass was diagnosed as adrenal myolipoma. Myelolipomas are usually small, asymptomatic and non-functional benign tumour of unknown etiology.[1] But rarely it can be large as it was in our case.[2] The asymptomatic small lesions of less than 4 cm should be followed up with CT scan or MRI; though some advocate just a clinical follow-up without routine follow-up with radiological investigations.[3] Surgery is indicated in symptomatic patients, or lesion of more than 4 cm in size due to rare chances of rupture[4], or if malignancy is suspected.







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[1]Consultant Surgeon, [2]Prof. of Surgery & Principal RKMSP, VIMS, [3]PGT 1st Year

Case Report

Non Compaction of The Left Ventricle

Dr. S. Guha[1], Dr. S. Ghosh[2], Dr. R. S. Ghose[3], Dr. S. Basu[4], Dr. D. Mondal[5], Dr. D. K. Mukherjee[6]

Abstract:

Noncompaction of left ventricle is an extremely rare cardiomyopathy resulting from a defective morphogenesis of the endomyocardium. It results in an architecturally aberrant ventricle wall consisting of two layers-a compacted layer and a loose interwoven mesh work with prominent trabeculae and deep intratrabecular recesses that communicate with the left ventricular cavity. This report describes the case of a three month male infant with non compaction of the left ventricle who presented with cardiomegaly and feeding difficulties.

Introduction:

Isolated noncompaction of the left ventricle (LVNC) is a rare disorder, classified as a primary genetic cardiomyopathy by the American Heart Association[1]. The European Society of Cardiology Working Group on Myocardial and Pericardial Diseases classified LVNC as an unclassified cardiomyopathy[2]. LVNC was previously also named spongy myocardium or hypertrabeculation syndrome; these terms should not be used as interchangeable terms[3].

LVNC is characterized by the following features:

- An altered myocardial wall with prominent trabeculae and deep intertrabecular recesses resulting in thickened myocardium with two layers consisting of compacted and noncompacted myocardium[5].
- Continuity between the left ventricular cavity and the deep intratrabecular recesses, which are filled with blood from the ventricular cavity without evidence of communication to the epicardial coronary artery system.

Case Report:

A three months old male baby, delivered by caesarian section with no history of consanguinity with birth weight of 2.3 kg admitted with feeding difficulty and respiratory distress from day 7 of life. He was on decongestive medications on admission. The infant was not in congestive cardiac failure. Respiratory rate was 35/min, heart rate was 120/min, all peripheral pulses equally palpable with no radio-radial or radio-femoral delay. Capillary refilling time <3 secs. Cardiovascular examination revealed left ventricular hypertrophy with grade 3/6 pansystolic murmur at the apex with no basal crepitations. There was soft non-tender hepatosplenomegaly (Liver 3 cm, Spleen 2cm). Other systemic review revealed no abnormality.

There was a history of three sibling loss with loss of first female child at three months of age, second male child at day 15 of life, third again a male child at the age of 6 months.

The chest X-ray showed cardiomegaly. Sinus tachycardia with left ventricular hypertrophy was present in ECG. Echocardiography at our hospital demonstrated grossly enlarged left ventricle. A two layer structure was seen with a compacted thin epicardial band and a thick non compactedepicardial layer with coarse trabeculations having deep trabecular recesses

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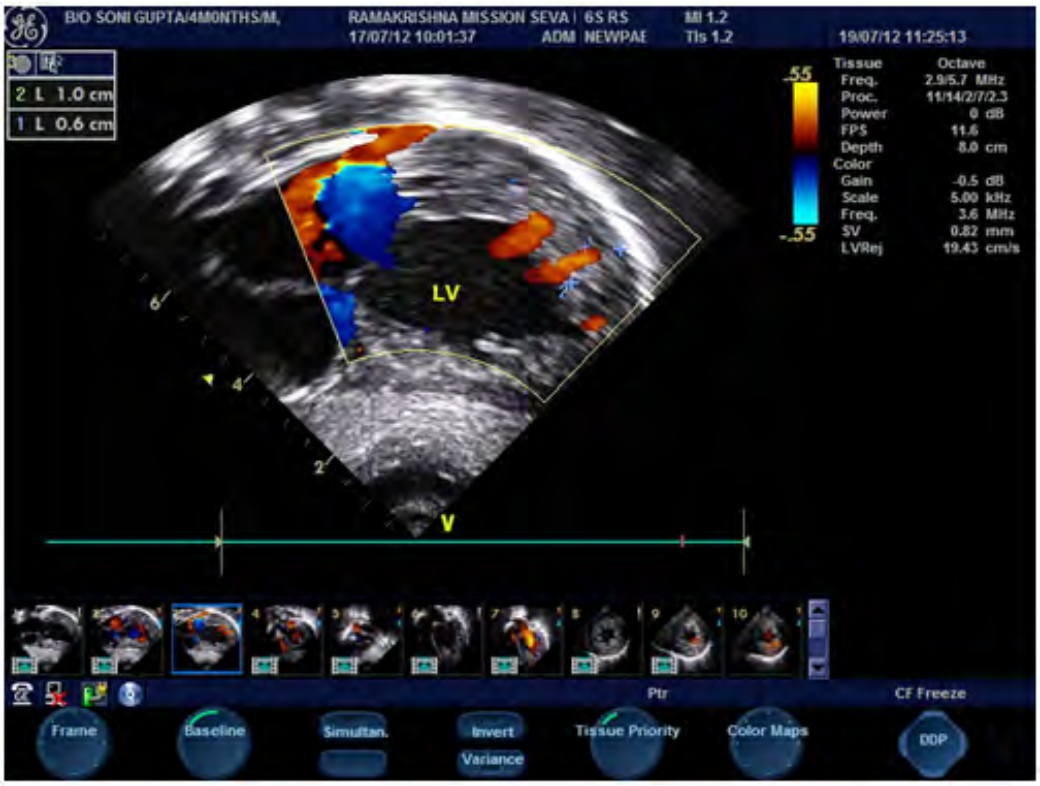
at the apical and lateral wall segments of left ventricle. The end systolic ratio of non-compacted to compacted layers was 3:5. The color Doppler images showed blood filling the recesses from ventricular cavity. The origin of both coronary arteries were normal. The M-mode showed left ventricular end diastolic diameter (LVEDD) 3.5mm, left ventricular end systolic diameter (LVESD) 2.9mm (body surface area .3kg/m^2), left ventricular ejection fraction (LVEF) 28% with shortening fraction(SF) 0.16. There was mild mitral regurgitation. The right ventricle was normal. These findings were consistent with left ventricular noncompaction.



2D Mode Echocardiograhy Showed Spongy Appearance of The Left Ventricular Wall

The infant was discharged on decongestive therapy (digoxin and furosemide), aspirin at antiplatelet dosage and carnitine with beta blocker. The echocardiography of both parents were normal.

Discussion:

Non compaction cardiomyopathy was first identified as an isolated condition in 1984 by Engberding and Benber.[4] They reported on a 33 year old female presenting with exertional dyspnea and palpitations. Investigations concluded persistence of myocardial sinusoids (now termed non compaction). Prior to this report, the condition was only reported in association with other cardiac anomalies, namely pulmonary or aortic atresia.

Before the fifth week of intrauterine life, the myocardium forms a loose network of fibers

and sinusoids which are in continuity with the ventricular cavity. Subsequently, the meshwork of fibers becomes 'compacted' and the sinusoids disappear. Pathological arrest of this compaction process leads to the persistence of ventricular hypertrabeculation, so called spongy myocardium or left ventricular (LV) non-compaction (NC).

Non compacted myocardium may also be seen in association with other abnormalities –

1. Congenital right ventricle or left ventricle outflow tract abnormalities- e.g. pulmonary atresia with intact intraventricular septum[5]. Coronary artery abnormalities are also common.
2. Non compacted myocardium is occasionally seen accompanying other congenital cardiac disorders e.g. Ebstein anomaly, bicuspid aortic valve, congenital corrected transposition of great arteries, hypoplastic left heart syndrome [6]. It may also be seen in patients with ventricular septal defect, patent ductus arteriosus [8].
3. Non compaction of the left ventricle can also be seen in metabolic diseases and genetic syndrome e.g. Barth syndrome, Charcot-Marie-Tooth disease, as well as nail patella syndrome.[9]

Common symptoms associated with a reduced pumping performance of the heart include breathlessness, fatigue, swelling of the ankles, limited physical capacity and exercise intolerance. Two conditions though that are more prevalent in noncompaction cardiomyopathy are: tachyarrhythmia which can lead to and clotting of the blood in the heart.

Diagnosis is made with 2-dimensional echocardiography, cardiac magnetic resonance imaging, or LV angiography. The natural history of LVNC is largely unresolved but includes LV systolic dysfunction and heart failure (and some cases of heart transplantation), thromboemboli, arrhythmias, sudden death and diverse forms of remodeling. Both familial and nonfamilial cases have been described. In the isolated form of LVNC, ZASP (Z-line) and mitochondrial mutations and X-linked inheritance resulting from mutations in the G4.5 gene encoding tafazzin (including association with Barth syndrome in neonates) have been reported. Noncompaction associated with congenital heart disease has been shown to result from mutations in the α-dystrobrevin gene and transcription factor NKX2.5

Diagnostic Criteria:

There are 2 sets of echocardiographic criteria for IVNC diagnosis: the Jenni criteria, which stress the presence of a 2-layered structure and the Chin criteria, which focus on the depth of the recess compared with the height of the trabecula[7]. In both sets, it is important that there are no other cardiac structural abnormalities, such as semilunar valve obstruction or coronary artery anomalies.

Due to its recent establishment as a diagnosis, and it being unclassified as a cardiomyopathy according to the, it is not fully understood how common the condition is. Some reports suggest that it is in the order of 0.12 cases per 100,000[8].

In a study (2006) carried out on 53 patients with the condition in Mexico, 42 had been diagnosed with another form of heart disease and only in the most recent 11 cases that ventricular noncompation was diagnosed and this took several echocardiograms to confirm. The most common misdiagnoses were (30 Cases), (6 Cases)

Conclusions:

NVM is recently included in the 2006 classsification of cardiomyopathies as a Genetic Cardiomyopathy.[3] When diagnosing LVNC, end-systolic as well as end-diastolic images have to be considered. The presence of more than three trabeculations as well as a two-layered myocardium are required. Since these criteria are not anatomically controlled, a comparison of echocardiographic images with pathoanatomic findings for assessing sensitivity and specificity is urgently needed. The familial character of LVNC has been recognized and probably up to 44% of patients do have affected family members. Although there are no systematic investigations on the familial occurrence of LV NVM, there are single reports that demonstrate LV NVM to occur in multiple family members.

In conclusion, echocardiography is the method of choice to establish a diagnosis and determine a treatment plan for patients with NVM. Despite the widespread use of echocardiography, NVM is commonly misdiagnosed because of the lack of knowledge of this disorder, with a significant negative impact on the prognosis of these patients. Therefore, it is still important to make echocardiographers more familiar with this condition and its pathology.

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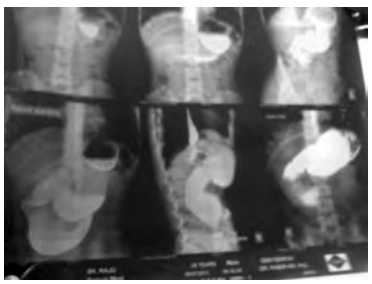
Case Report

Duodenal Carcinoid Tumour with Liver Metastasis

Dr. Kalyan Ashis Mukherjee[1], Dr. A. K. Saraf[2]

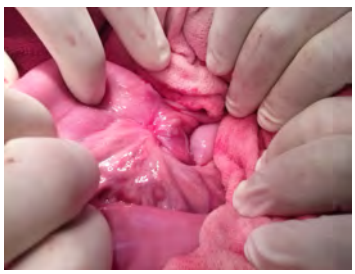
A 34 years carpenter from Howrah presented to our OPD with gradually deteriorating features of upper GI obstruction for two and half years. His vomitus contained bile and old food material but no fresh or altered blood. He was cachectic and had pallor. All other physical examinations were normal. No Abdominal mass was palpable, no organomegali. He was being investigated thoroughly. Upper GI endoscopy showed normal study up to second part of duodenum. Barium study showed dilated stomach (Pic 1) and duodenum till third part and sudden narrowing after that. To detect the cause of obstruction we had done CT scan abdomen which also supported the findings previous study along with it excluded luminal growth. (Pic 2) So our working diagnosis was extra-luminal obstruction of third part of duodenum, and suspicion was superior mesenteric syndrome.

Exploratory laparotomy was done under general anesthesia. After exploration we found there is moderate ascitis, a growth was palpable at third part of duodenum (Pic 3) and multiple liver nodules. Indurations around duodenal growth was extending medially and reaching splenic vein where it joins Superior Mesenteric Vein to form Portal vein. So if we want to remove the whole tumour then there is a high possibility of portal vein injury. Considering the existing metastasis we reject the plan of curative surgery and finally we did an anti-colic Gastro-jejunostomy. Post operative day one and two patient had two episodes of hypoglycemia (Capillary Blood Glucose <50 mg %). Then we thought of about the possibility of neuro-endocrine tumour and planned to investigate. But patient was taken to a Government Hospital by his attendents due to economic reason. Histopathology report showed Well differentiated neuro endocrine tumour, suggestive of Carcinoid. Unfortunately patient was expired after 11 days of surgery. Carcinoid is a rare tumour and carcinoid metastasized to liver even rarer. Interestingly, these tumours usually not produce typical systemic features of carcinoid. Study showed satisfactory result after surgery. Even after metastasis survival is better than duodenal adenocarcinoma. But here our patient was suffering for more than two and half years which probably allow the disease to spread to such extent.



Carcinoid Barium (Pic. 1)

Carcinoid CT (Pic. 2)



Carcinoid 3rd Part of Duodenum (Pic. 3)

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[1]Consultant Surgeon, [2]Prof. of Surgery & Principal RKMSP, VIMS

Obituary

We express deep sorrow at the passing away of —

DR. SUNIL KUMAR BAGCHI, MBBS, DOMS, FRCS (Edin.) *who was attached to the department of Opthalmology in this institute from 1968-69 to 2000 in different capacities, the last being an Hony, Consultant, passed away on 24th January, 2014.*

We remember with respectful gratitude the dedicated and selfless services rendered by him during his long association with this Institute.

We whole heartedly pray to the Almighty for the eternal peace of the departed soul.

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