IMPORTANT TELEPHONE NUMBERS

Emergency / Casualty :	**2656 8063 / 2656 8064**
Ambulance :	97692 50010
Hospital Board Line :	022-2675 1000 / 2656 8000
Hospital Fax :	022-2640 7655 / 2640 5119
Admission Department :	2656 8080 / 2656 8081 / 2656 8082
TPA Cell :	2656 8089
Appointment-OPD :	2656 8050 / 2656 8051
Billing-Inpatient Department :	2675 1586 / 2675 1585
Billing-OPD Department :	2656 8052 / 2656 8053
Blood Bank Department :	2656 8214 / 2656 8215
Health Check-up Department :	2656 8355 / 2656 8354
Report Dispatch Counter :	2656 1620
MRI Department :	2656 8066
X-Ray, Sonography Department :	2656 8031
CT Scan Department :	2656 8044 / 2656 8045
Physiotherapy Department :	2675 1536 / 2675 1698 / 2675 1699

Lilavati Hospital and Research Centre

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LILAVATI HOSPITAL
MEDICAL TIMES

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EDITORIAL...

Changing Scenario: Healthcare In India

It's an absolute delight to be associated with Lilavati Hospital Medical Times (LHMT) since its launch. I am grateful to the readers and the clinicians who have enthusiastically come forward and given their bit of thought to our institution's finest thought sharing process which has taken shape in articulating what virtually we all think and dream in the form of LHMT.

Health care system comes in crises when we systematically neglect prevention and wellness. We may never understand the magnitude of other's illness but we have to understand a mechanism of prevention. Finding cure should not be the objective, Prevention is what required. Prevention is worth more than million pounds of cure. “Health is the greatest of all possessions; a pale cobbler is better than a Sick King”.

The field of medical science has come a long way and yet has to go miles further. I am of the opinion of having continues improvement in the field of medical know-how's and improvement which not only reflects capabilities of Lilavati Hospital but the health scene for our nation.

This edition of LHMT includes the aspects we should know about Lilavati Hospital; these are the essential features of the institute. A brief on our Physiotherapy Neuro Rehabilitation service; which we wish to share with our readers is presented. A variety of informative reports are presented by our experts on Anesthesiology, Gastro Surgery and Radiology, Pediatric Surgery and Pedicatrics, Histopathology, Nephrology and Gastroenterology. Besides this; we have a report on SEWA - our social wing, straight from the heart section illustrate appreciations we have received for our efforts. CMEs have been an ongoing feature of our institution which are regularly conducted and these academic sessions are spreading information to the medicos who want to keep pace with the cutting edge technologies and the latest medical systems practiced.

My participation in the LHMT is not restricted only to the eminence of the periodical but on the overall development of the content and articles. I would be glad to receive any feedback from you which will help me in making LHMT even better. We all at Lilavati Hospital and Research Centre always strive to improve in all areas of life and I look forward to your involvement to a greater extend to broaden our reach to larger section of people and taking LHMT to the next best possible level.

Dr. Narendra D Trivedi

LILAVATI HOSPITAL TODAY

Department in focus: Physiotherapy Neuro Rehabilitation

In today's times, due to the highly stressed and sedentary lifestyle episodes of 'stroke at young', 'frequent falls', 'slowness in doing daily activities' is increasing. These problems occur due to various changes in the brain. To combat these changes neurorehabilitation plays a very important role in one's life. Neurorehabilitation comprises of various treatments that are case specific focusing on patient's recovery with neurological disorders. Aim is to improve the quality of life of patients with post stroke limitations, Traumatic Brain Injury, brain or spinal cord injuries. Focused strategies are adopted to suit various patients during the therapy to overcome functional limitations. Neurorehabilitation also includes transfers, gait training, balance training and neuro muscular retraining, helping the patients in activities of daily living. Also, motor re-learning techniques which facilitates to alter to recovery of function is an important part of neurological physical therapy. The core of neurorehabilitation remains in improving strength, mobility targeting patient's independence and social well-being.

At Lilavati Hospital, importance is on patient goal oriented treatment. This is achieved by using various motor learning exercises, repetitive exercises which are task oriented. These exercises promote neural plasticity in the higher levels of the brain. This type of training at LHRC is incorporated through advanced equipment in the department like.

1. **BALANCE MASTER**
2. **UNWEIGHING TREADMILL**
3. **SCIFIT**
4. **NEUROMUSCULAR ELECTRICAL STIMULATION**

BALANCE MASTER

Balance master allows the physiotherapist to perform an objective assessment on static and dynamic balance. It retrains the sensory system with visual feedback. This comprises of Assessment, Sequence training and Custom based training for evaluation and then do the treatment. Numeric and comprehensive progress reports can be generated when a patient's assessment is performed. Balance Master helps to overcome functional limitations like sit to stand, transfers, gait training, weight shifting, tandem walking etc.

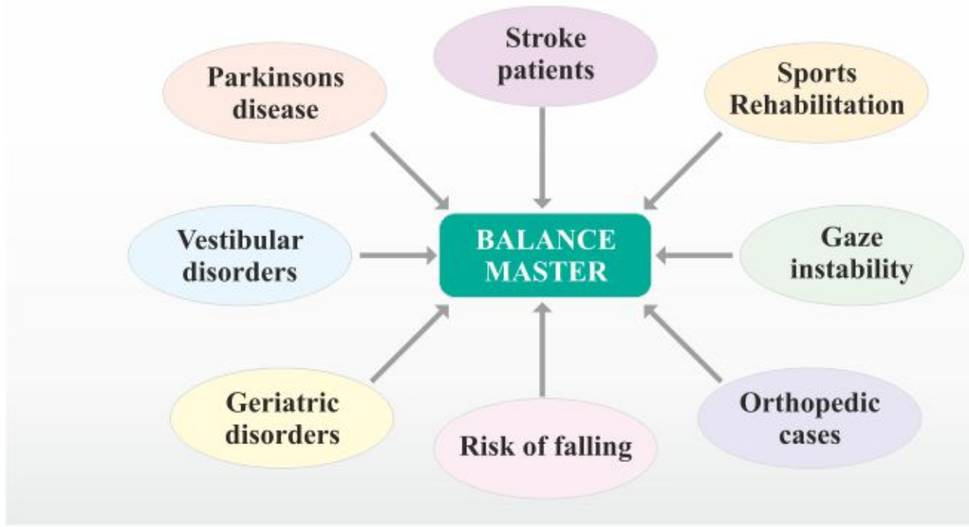
PATIENT BENEFITS

- To procure a baseline, status of the patient's condition.
- Enhances patients performance with visual feedback.
- Links perception to movement.
- Improves the postural alignment.
- Build's confidence in daily activities.

The balance master also allows us to do a detailed analysis of functional movements like "QUICK TURNS", "Weight shifting "etc. This analysis allows us to plan a treatment with effectiveness of therapeutic programme. Custom based training helps to treat goal oriented therapy.

LILAVATI HOSPITAL TODAY

CONDITIONS WHERE BALANCE MASTER CAN BE USED:



ACCESSORIES USED:

1. **Foam pad**
2. **Rocker board**
3. **Wedges and step stool**
4. **Step up blocks**
5. **Heel / toe wedges**
6. **Leveling block**
7. **Inversion / eversion**
8. **Curbs**

These accessories help in reducing the usefulness of somatosensory inputs for balance control, to train various strategies (hip, ankle), loading and unloading activities training (in sports rehabilitation), to improve medial lateral instability etc.

Thus, balance master can be used as an integral part in our treatment. This allows a person to live life more confidently, maximize the level of independence and improve the quality of life.

LILAVATI HOSPITAL TODAY

UNWEIGHING SYSTEM WITH TREADMILL

"The 1st step is the hardest", this old proverb suddenly seems more relevant than ever for patients facing a new challenge: To regain their ability to walk. The unweighing provides the optimum conditions to take that 1st step. It goes on to play an important supporting role for every step during the initial stages of patient's rehabilitation and ambulation training in a safe environment.

The unweighing System enables partial weight bearing therapy to be conducted with the assurance of patient comfort, safety and with convenient access to the patient for manual observation and assistance. The electrical unweighing trainer is designed to apply vertical support to remove the stress of bearing body weight, digitally controls weight bearing and promotes proper posture and balance over the treadmill or ground. The system allows patients to stand upright and use both arms freely. Harness gives security to patients with limited trunk strength. The adjustable suspension bar and harness accommodates individuals of all body sizes.

The rehab treadmill features accurate speed, adjustable inclines, movable control panel and a true zero start. It is powerful enough to move a 200kg patient at speeds as low as 0.1 mph.

SALIENT FEATURES:

- Low step height for patients to easily step on and off.
- Distance readout in feet and miles for easy charting.
- Large walking surface.
- Incline upto 10% powered by high capacity motor.
- Zero start capability which is a must for rehabilitation. It allows patients to stand on the belt when starting the treadmill. Patients do not need to straddle the belt when safely supported in the unweighting harness. The speed ranges from 0.1 to 6.0 mph and is adjustable in 0.1 mph increments. Speed is calibrated accurately at even the lowest rates, important in the rehab settings.

All above features make it the most advanced solution for Gait Therapy.

Research has shown good results in ambulation training of patients with conditions such as Stroke, Parkinson's Disease, GBS, Spinal Cord Injuries, Neuropathies, Transverse Myelitis, Cerebellar Ataxias, Head Injuries Etc.

Thus the unweighing system with Treadmill helps begin the gait training earlier in the rehabilitation process. It not only regains patients ability to walk but makes them independent in walking.

LILAVATI HOSPITAL TODAY

SCI FIT

The SCI FIT formula for success:
MOVE + PROVE = IMPROVE

MOVE - The equipment is designed to make it easy to start moving. Extreme accessibility, low starting resistance and easy interaction are just the beginning.

PROVE - People with goals achieve more. Sci fit makes it easier for everyone to set, review, and ramp up realistic yet challenging goals for measurable performance.

IMPROVE - Sci fit lets people experience the motivated thrill of improvement.

When the nervous system is affected, muscles may be weak / floppy or very tight with spasms / tremor and movement may be uncoordinated. Sci fit helps in bringing about coordination of movements, near normalization of tone, reduce stiffness, maintain mobility, improve cardiac functioning or iso-strengthening and an overall rehabilitation of an individual / patient through the variable programs it provides.

PRO 2 is an upper body exerciser and lower body recumbent bike in one for a total body solution. Dependant upper and lower cranks enable passive assistance. Some of its features are :

- Knee to elbow motion for cardio to the core
- Easy access with true, adjustable step through
- Removable seat for wheelchair accessibility
- Adjustable seat options
- Bidirectional exercise
- In cases when the patient cannot do movements independently, the therapist can assist them through the assist pedal

Depending on the individual / patient status and requirement, appropriate program of the sci fit can be chosen. They provide with options for gradually increasing resistance or constant or general workout with continuous monitoring and adjustable duration, speed and resistance and help in overall rehabilitation.

NEUROMUSCULAR ELECTRICAL STIMULATION

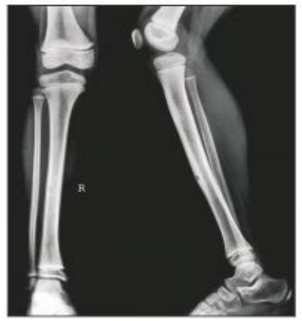
Neuromuscular Electrical Stimulation has been well established in enhancing performance of innervated muscle as well as stimulation of denervated muscle.

The waveform used is symmetrical biphasic waveform with a 100 microseconds interphase interval. Because of its short pulse this waveform has low skin load, making it suitable for applications requiring high intensities such as in muscle strengthening protocols.

It can be used for variety of patient populations to address impairments of muscle strength, muscle spasticity, muscle spasm, swelling and tissue extensibility to enhance functional outcomes and also urinary incontinence and to reduce shoulder subluxaton in stroke patients. NMES when applied during functional activity (Gait Training) is referred as Functional Electrical Stimulation.

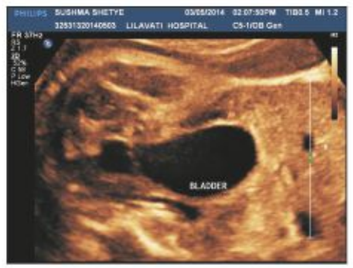
Thus, all the above equipments help improve patient's physical and social well being. Neurorehabilitation helps incorporating the patients in the community much earlier thus improving their quality of life.

IMAGES OF THE MONTH

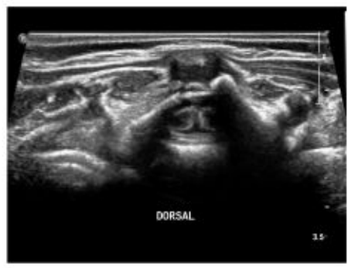


Osteoid Osteoma

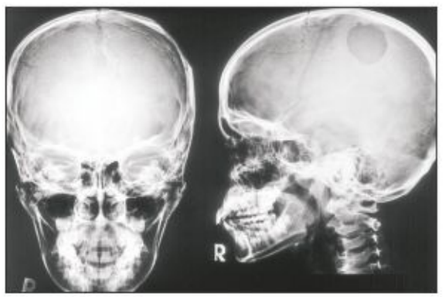
AP & Lateral view of right leg showing sclerotic thickened anterior cortex of right tibia with well defined radiolucent nidus suggestive of Osteoid Osteoma



Distended Fetal urinary bladder with echogenic posterior urethral valves

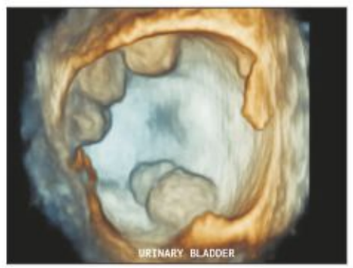


Diastometamyelia with bony spur



Eosinophilic granuloma

Skull X-Ray AP and lateral view shows a well defined punched out lytic lesion with bevelled edges in left parietal bone suggestive of eosinophilic granuloma.



Papillomas

Multiple polypoidal lesions seen arising from wall of urinary bladder suggestive of Papillomas

STRAIGHT FROM THE HEART

"Lilavati a Healthcare institute - So caring, warm and generous with amazing checkups and facilities".

Kareena Kapoor,
Film Actress

"Thank you all for the lovely experience have at Lilavati. Excellent service and of course doctors".

Karishma Kapoor,
Film Actress

"Amazing coordination & great team work of various departments, skillfull staff. Keep up the wonderful work so that the hospital grows from Strength to strength".

Asha Pandit

"The doctors are very helpful and supportive to the patients, hospital is very nice & each and every thing is excellent".

Samina Shaikh

"Excellent System, Infrastructure, Security, Doctors & Nursing staff. All nurses attending the patient were excellent and has performed their duties genuinely, professionally and sweetly. They are the backbone of this hospital".

Suhas Joshi

"Consultants are very good, nursing staff are simply great and no where else have I experienced such good nursing care".

Saama Pandit

"The love and care of the nursing staff is par excellence. Always ready to help and extremely professional. A variety of facilities are provided to the patient making them extremely comfortable. Keep up the good work of serving your patient. It is a very noble job. It is truly more than Health Care, Human care".

Nishika Choudhary

"Well planned and organised care. Prompt explanation given by the doctors regarding the care helps to reduce the anxiety in the patient and relatives. Has a very good system of administration without causing any inconvenienceto the patient".

Mary Ipe

"The friendly nature of everyone at the hospital actually make the patient recover faster. The food is really fresh and tasty".

Anil Palekar

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CASE REPORT: ANAESTHETIC MANAGEMENT FOR TRACHEAL RESECTION AND RECONSTRUCTION

Dr. Ami Merchant *MD,* **Dr. Vaibhavi Baxi** *DA, FCPS, DNB,*
Dr. Sanjay Sharma *MS, FICS, FACS, FAIS,* **Dr. Bhuvaneshwari Balasubramanian** *MD.*

ABSTRACT :

We report the anaesthetic management of a patient with severe tracheal stenosis planned for tracheal resection and end to end tracheal reconstruction. General anaesthesia was administered with tracheostomy tube in situ. Post induction, tracheostomy tube was replaced with a flexometallic endotracheal tube (ET). Another flexometallic endotracheal tube was inserted through right nostril under laryngoscopic guidance and placed just beyond the larynx. The stenosed tracheal segment was resected after mobilization of the trachea both proximally and distally. The trachea was mobilized and posterior layer anastomosis was done while the ventilation continued through distal tracheal opening. The nasal ET tube was then introduced beyond the larynx across the suture line into the lower part of trachea making sure the cuff is beyond the anastomotic site and anterior anastomosis was completed. Patient received intermittent positive pressure ventilation (IPPV) with intermittent apnoea to facilitate surgery. All precautions to maintain oxygenation and ventilation during tracheal resection were taken.

INTRODUCTION:

Tracheal resection is indicated for patients with tracheal obstruction due to tracheal stenosis, tracheal tumours, congenital anomalies etc. Tracheal surgery is always a special endeavour as the airway is shared by the surgeon and the anaesthetist. Knowledge of various techniques of airway management is crucial since the principle anaesthetic consideration is ventilation and oxygenation in the face of an open airway. Anaesthetic management in tracheal resection varies depending upon the location of tracheal lesion and method of resection. This article discusses the successful management of a patient undergoing high tracheal resection and primary reconstruction.

CASE REPORT:

A 22yrs old young man with history of road traffic accident with a subdural haemorrhage underwent emergency craniotomy three months ago. Postoperatively he was placed on mechanical ventilation with an oral endotracheal tube due to poor neurological status. Tracheostomy was performed on 7th postoperative day in view of prolonged ventilation. He improved neurologically and respiration improved over the next few days. Removal of tracheostomy tube was attempted but it had to be reinserted due to respiratory distress. Fibreoptic bronchoscopy through nose was attempted in the intensive care unit but could not be negotiated about 2-3cm beyond the larynx. A computed tomographic (CT) scan showed a high grade narrowing of the trachea of length 1.3cm just above the tracheostomy opening causing almost complete obstruction of trachea. This stenotic lesion was approximately 3.4cm distal to the vocal cords. No evidence of tracheomalacia inferior to tracheostomy site was reported in the CT scan. [fig 1. CT scan - tracheal stenosis just above tracheostomy]

Preoperative evaluation by history, clinical examination, hemogram, baseline arterial blood gases, X-ray chest and CT chest ruled out the presence of any preoperative lung disease severe enough to indicate a the need for postoperative positive ventilatory support; which is a relative contraindication for tracheal resection.

Anaesthesia was induced with 6% sevoflurane in 100% oxygen with 7 no. tracheostomy tube in situ, in addition to intravenous 100mic fentanyl, 0.2 mg glycopyrrolate and 50mg atracurium. By gentle airway instrumentation the tracheostomy tube was then replaced by a sterile 7 no. cuffed flexometallic endotracheal tube to which the breathing circuit was attached across the surgical field. Endotracheal tube cuff was optimally inflated to avoid air leak. A second flexometallic endotracheal tube of 7.5 no. was introduced through right nostril positioned just beyond the larynx by direct laryngoscopy.

Maintenance of anaesthesia was done with an air:oxygen mixture (0.5% to 1% FiO2) with intermittent Sevoflurane. Also a dexmedetomidine (0.5mic/kg/hr) and atracurium (0.5mg/kg/hr) infusion was maintained. Ventilation and oxygenation was maintained with IPPV with intermittent apnoea to facilitate the surgical procedure. The longest apnoea duration was of 92 seconds. At no point during the surgery did we encounter any fall in the oxygen saturation. We had kept a higher rate of ventilation (20 breaths/min with 500ml tidal volume) to prevent hypercapnia. All vital parameters were maintained well within normal limits throughout the procedure.

Trachea was surgically exposed through Sorenson's incision and by retraction of strap muscles in the neck. A laryngeal drop procedure was done and muscle of superior border of hyoid bone was cut to mobilize the same. Oesophagus was separated off posteriorly. The stenosed segment of trachea including two tracheal rings was excised around the tracheostomy site with intermittent apnoea. The two cut ends were brought together and the posterior wall of the two cut ends of trachea were anastomosed with intermittent prolene sutures. On the anterior wall stay sutures were taken. [fig. No.2- posterior tracheal wall sutured and stay sutures on anterior wall]. The nasally passed flexometallic ET was then guided into the lower end of the trachea making sure that the cuff of the tube is beyond the suture line under vision. The anaesthesia circuit was now connected to the nasally passed flexometallic ET. Anterior wall was now sutured to complete the end to end anastomosis. As there was no leak we did not inflate the ET cuff. At the end of the surgery bronchoscopy was done through the nasal endotracheal tube to ensure that the ET cuff does not lie on the suture line and also the position of tracheal end of ET was confirmed to be just above the carina.

Postoperatively the patient was kept in a position of head flexion by taking a sternomental stitch (called the guardian suture) to avoid tension on the suture line of tracheal anastomosis. [fig. No 3- sternomental stitch] .Neuromuscular blockade was reversed and spontaneous respiration was achieved. Patient was kept on T-piece with 6l/min oxygen flow in the recovery room. After two hours of observation patient was extubated to avoid any strain on suture line due to coughing. Nebulization with saline and steroid therapy was given in the recovery room. Patient maintained adequate ventilation and good vocalization confirmed that there was no injury to the vocal cords or the laryngeal nerve. Patient was then shifted to the intensive care unit for postoperative care and was discharged from hospital on 7th postoperative day.

ANAESTHETIC MANAGEMENT FOR TRACHEAL RESECTION AND RECONSTRUCTION

ANAESTHETIC MANAGEMENT FOR TRACHEAL RESECTION AND RECONSTRUCTION

DISCUSSION:

Tracheal stenosis may occur secondary to trauma, chronic inflammatory disease, benign or neoplastic lesion and collagen vascular diseases. However the commonest cause for tracheal stenosis is internal trauma like prolonged endotracheal intubation, tracheostomy or external like blunt or penetrating neck trauma.[1]

Various treatment modalities including balloon dilatation, stenting, laser ablation and surgical resection are currently available for the management of tracheal stenosis. Treatment of choice depends upon the site and degree of obstruction. For short (<1 cm), membranous stenosis without damage to the cartilages, laser incisions followed by gentle dilatation or tracheal stent is the safe and complete treatment. But for complex tracheal stenosis, with circumferential hourglass-like contraction, surgical resection and end-to-end anastomosis are considered the standard curative treatment.[2]

Anaesthesia for tracheal resection is often challenging due to narrowing or interruption in the continuity of the airway, potential for complete obstruction of an already stenosing lesion and problems of ventilation during induction, bronchoscopy and surgical repair. As our patient had a high tracheal stenosis with distal tracheostomy we replaced the tracheostomy tube with a flexometallic ET and the patient maintained both oxygenation and ventilation despite intermittent apnoea. In patients with critical low tracheal stenosis ventilation may be difficult through ET due to high airway pressures and hypercarbia may develop. Extracorporeal circulation may be initiated in some such cases to maintain oxygenation.[3]

Several ventilation techniques have been used in tracheal surgeries. In our case we used IPPV with intermittent apnoea. There is risk of hypoxia and hypercarbia with this technique. To prevent this we used a higher rate of ventilation with 1.0 FiO2. Apnoea durations were also small the longest being 92 secs. High-frequency jet ventilation (HFJV) & superimposed high-frequency jet ventilation (SHFJV) use small catheters and provide an improved view of an immobile operative field. However there is risk of barotauma due to intrinsic PEEP, hypercarbia and also tracheobronchitis with the cold unhumidified gases.[4]

Percutaneous transtracheal jet ventilation (PTJV) via a catheter placed through the cricothyroid membrane or the trachea has also been used to avoid tracheostomy. It is simple, safe and its most important benefit is immediate oxygenation. The catheter can be left in place postoperatively in the event that the patient needs further respiratory support. Potential complications of PTJV include a kinked cannula, catheter misplacement, haemorrhage at the site insertion, barotrauma, oesophageal injury, surgical emphysema, and gas embolism.[5]

We used pulse oximeter as it is essentially a reliable monitor for early detection of hypoxia. Arterial blood gas analysis is not much useful due to time delay involved. As the edema at the site of anatsomosis may lead to respiratory insufficiency intravenous steroids are useful.

Spontaneous ventilation should be resumed after the procedure to minimize trauma to the tracheal suture line. Most patients may be safely extubated, but in those for whom difficult anatomy or copious secretions make this undesirable, a small

ANAESTHETIC MANAGEMENT FOR TRACHEAL RESECTION AND RECONSTRUCTION

tracheostomy may be placed below the tracheal repair. The patient should be awake enough to maintain spontaneous ventilation and avoid aspiration but should be extubated before excessive head movement can damage the surgical repair. If tracheal collapse, airway edema, or secretions cause respiratory distress after extubation, the patient should be reintubated fiberoptically with a small uncuffed endotracheal tube, preferably with the head maintained in forward flexion. Small amounts of opioids are needed to treat the mild pain for neck excision; given after the patient is wide awake and responsive while monitoring for undesirable respiratory depression.

In conclusion, successful outcome is possible with safe and efficient intraoperative measures, intensive monitoring and good postoperative care.

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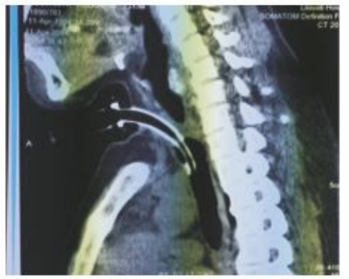
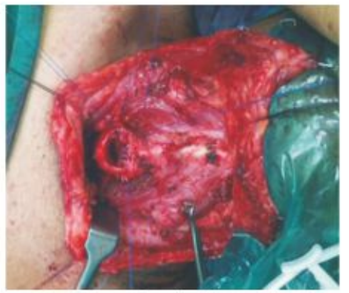
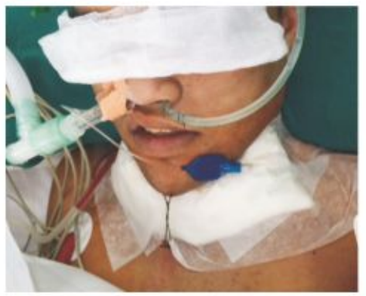


CT Scan- Tracheal Stenosis just above tracheostomy



Posterior tracheal wall sutured and stay sutures on anterior wall



Sternomental stitch

CASE REPORT: CAELIAC ARTERY COMPRESSION SYNDROME

Dr. Rohan Reddy, *(2nd year DNB General Surgery),* **Dr. Raman Garg,** *M.S,*
Dr. Raina Tembey, *DNB,* **Dr. Aneeta Bajaj,** *M.D,* **Dr. Abdul S Ansari,** *M.D,*
Dr. Prasad K Wagle, *M.S*

INTRODUCTION

Celiac artery compression syndrome (also referred to as celiac axis syndrome, median arcuate ligament syndrome (MALS) and Dunbar syndrome) is defined as abdominal pain related to compression of the celiac artery by fibers of the median arcuate ligament. MALS was first described by Lipshultz as an anatomic structure that caused celiac artery compression in 1917. Thereafter, Harjola and Dunbar et al. described it as a clinical syndrome causing nausea, vomiting and postprandial pain in 1963 and 1965. We reported a case of a 27 year-old female patient who presented to us with a perplexing set of symptoms. Several Imaging techniques were employed, to rule out the common etiologies and to arrive at the diagnosis. Surgical division of the compressive 'band' was done to achieve relief of symptoms.

CASE REPORT

A 27-year-old, unmarried female presented to us with the chief complaints of excrutiating pain in epigastric region, typically aggravated after food intake, without any radiation. She had several episodes of vomiting over past few weeks, containing the ingested food material, non-bilious and non-blood stained. She never had any altered bowel habits, change in appetite or urinary symptoms at present or in the past; no symptoms of jaundice, nor any fever, giddiness or trauma to head. Her menstrual periods were regular (4-5 / 28-30), with normal flow. She was not on any regular medications except over-the-counter analgesics during the present episode of pain.

On admission, she was in severe discomfort due to pain. She was afebrile (98.8 F), pulse rate - 86/min, good volume, blood pressure of 110/60 mmHg in supine position, with a normal respiratory rate, and adequately hydrated. Abdominal examination revealed moderate to severe tenderness in the epigastric region, without guarding or rigidity. There was no evidence of free fluid in the abdomen and peristalsis was normal. Rest of the systemic examination was within normal limits.

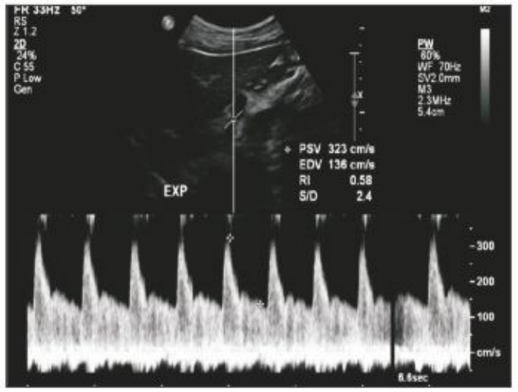
Routine haemogram, urine routine and microscopy was done; was found to be within normal range. Liver function tests were essentially normal, but for a small elevation of transferase enzymes (AST-52U/L, ALT-54U/L). Urine for porphobilinogen was negative. An ultrasonography and CECT of the abdomen and pelvis was done, which was reported to be normal. An Upper GI Scopy revealed granular reflux oesophagitis, and no other significant findings were noted. She was being treated symptomatically for pain in abdomen and head aches, her symptoms were persistent despite therapy. A Computerized Tomography with Mesenteric Angiography was performed, which revealed a Compression of the Celiac Artery on its superior aspect, at its origin from aorta. It was followed by a Colour Doppler Examination of the Abdomen which showed luminal narrowing of celiac trunk, that aggravated in expiratory phase; hence reaffirming the suspicion of Median Arcuate Ligament.

CAELIAC ARTERY COMPRESSION SYNDROME

On supine position luminal narrowing with aliasing flow was noted. On Spectral Doppler study increased velocity was noted which further accentuated on expiration. On erect position luminal narrowing reduced as compared to supine position. Aliasing artifact was absent on colour Doppler. On spectral, velocity in the inspiratory and expiratory phases were within the normal range. These findings confimed the suspicion of median arcuate ligament syndrome. An Exploratory Laparotomy was undertaken on this patient; a thick fibrous median arcuate ligament was noted compressing the Coeliac trunk, just at its origin from the Aorta. Following excision of the 'band' there was immediate relief in the compression. The patient showed gradual improvement in her presenting symptoms post-operatively.

DISCUSSION:

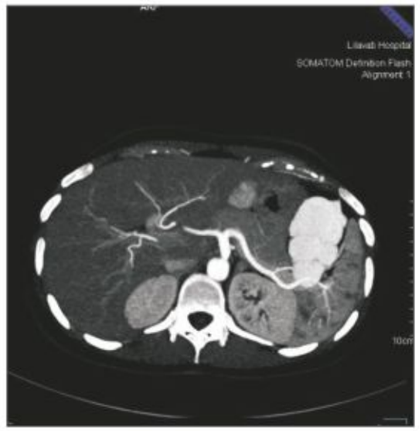
The median arcuate ligament (MAL) is located at the T12–L1 level and bridges the crura of the diaphragm, just anterior to the aorta. Dunbar's syndrome, median arcuate ligament syndrome (MALS) or celiac artery compression syndrome (CACS) is caused by external compression of the celiac trunk by the MAL, and is characterized by postprandial abdominal pain, nausea, vomiting, and weight loss. In the general population, 10-24% of people may have indentation caused by an abnormally low ligament. Few of these patients have hemodynamically significant stenosis that would cause symptoms.



There are 2 main theories used to explain the pathogenesis of the symptoms. The first theory is mesenteric ischemia due to celiac artery compression. Mesenteric ischemia arises either from direct foregut ischemia or, alternatively, through postprandial steal via collaterals from the superior mesenteric to the celiac bed, leading to midgut ischemia. The second theory has it that neurogenic stimulation is caused by compression of the celiac ganglion and plexus. Neurogenic stimulation-related pain, can be caused either from celiac plexus stimulation leading to splanchnic vasoconstriction or via direct sympathetic pain fiber irritation. Although the syndrome has been described in the 1960s, controversy continues as to whether celiac compression leads to the clinical picture or not. The controversy stems from an undefined pathophysiological mechanism and the existence of celiac compression in asymptomatic patients. It has been reported that 13%-50% of healthy individuals may exhibit, to a variable degree, angiographic features of compression especially during expiration. While patients are in an erect position, with inspiration, the

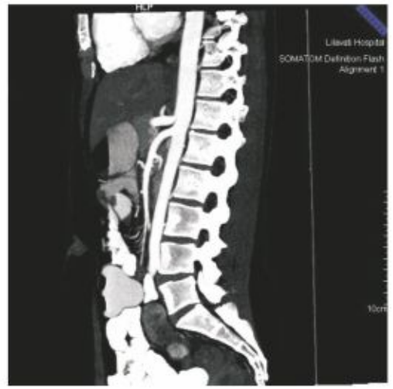
CAELIAC ARTERY COMPRESSION SYNDROME

celiac artery descends to the abdominal cavity, and compression is often relieved with a more vertical orientation of the celiac artery.



CT Angiogram (cross-sectional image) - Narrowing of Coeliac trunk with post-stenotic dilatation.

Celiac artery compression may be investigated with Doppler US, spiral CT angiography, selective catheter angiography, and magnetic resonance angiography. The gold standard diagnostic method is selective angiography, which should be performed during both inspiration and expiration, in the lateral position. Doppler US has been reported to have a high sensitivity for the diagnosis of CACS and was proposed to be the modality of choice for diagnosing CACS. The classical CT Angiographic picture is of focal narrowing of the vessel with a characteristic "hooked" appearance and post-stenotic dilataion. In our patient the CT Angiogram showed similar compression of the Coeliac axis at its origin with post-stenotic dilatation in both lateral and cross sectional views. The classical hooked appearance was not evident. The Colour Doppler revealed an angle of 52.5° between the celiac trunk and aorta. Luminal narrowing was confirmed with increased 'aliasing' of flow during the expiratory phase in supine position; the aliasing artifact disappeared in erect posture in both inspiratory and expiratory phases. Similarly, the Spectral Velocity in the vessel reduced in erect posture when compared to supine.



CT Angiogram (lateral image) - 'Constricted' appearance of Coeliac Artery at its origin.

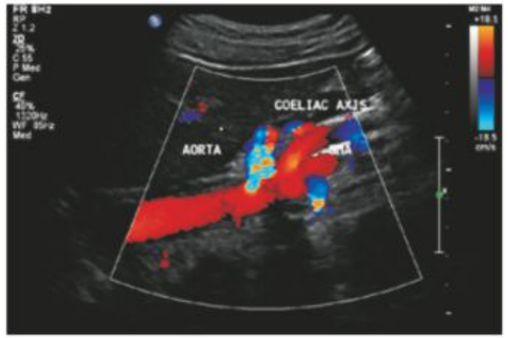
SUPINE	PSV	EDV
INSPIRATORY	342	149
EXPIRATORY	201	61
ERECT	SV	EDV
EXPIRATORY	99	35

(PSV- Peak Systolic Velocity; EDV - End Diastolic Velocity,in cm/s.)

CAELIAC ARTERY COMPRESSION SYNDROME

RATIO OF PSV OF COELIAC TRUNK : AORTA WAS MORE THAN 3.

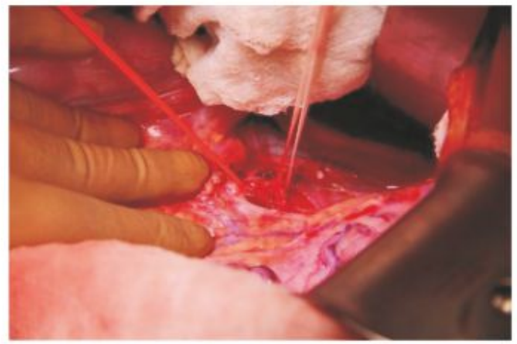
The above findings confirmed the fact that the compression is not due to a fixed lesion such as atherosclerosis but due to a dynamic extrinsic compression, that showed postural variation.



'Aliasing artifact in coeliac trunk insupine position - Colour Doppler

Surgical median arcuate ligament release has been the mainstay of treatment. Patient characteristics that predicted symptoms of relief after surgery were postprandial pain, age 40-60 years, female gender, and weight loss greater than 20 pounds. Typically, pain relief is immediate but because postoperative pain can mimic preoperative symptoms and may take up to 6 weeks to resolve, it may take that long to determine if the procedure was successful.

Our patient underwent an exploratory laparotomy that confirmed the imaging findings - a thick fibrous tract was seen bridging across the crurae of diaphragm, at the origin of celiac artery, compressing it. The division of arcuate ligament relieved the compression over the artery. Post operatively the patient had one bout of severe abdominal pain that likened presenting symptoms, but following analgesics it settled down. The patient has been symptom-free over a 3-month follow-up.



Red Tape - around Coeliac axis
Umbilical Tape (White) - Arcuate ligament compressing the former

A CASE OF HYPER IG E SYNDROME WITH LUNG ABSCESS

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Dr. Janani, *DNB Pediatric Surgery,* **Dr. Vinod,** *2nd year* **and**
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Dr. Redkar *MS, MCH, Pediatric Surgery,*

ABSTRACT:

The hyper-immunoglobulin E syndrome (HIES) is a rare primary immunodeficiency disorder characterized by high serum levels of immunoglobulin E (IgE), recurrent skin and lung infections, chronic dermatitis and a variety of connective tissue and skeletal abnormalities. The bacteria that commonly infect these patients are Staphylococcus aureus and Haemophilus influenzae. These patients share some characteristic facial appearance and many oral manifestations. Therapy should include prolonged antibiotic therapy. We report the clinical presentation of one such patient and a literature review of HIES.

Key words: Hyper Ig E, eosinophilia, recurrent infections

CASE HISTORY:

A 12 year female child first product of a third degree consanguinous marriage was admitted to our center with the complaints of fever and cough from 2 weeks. The child had cough with muco-purulent sputum, moderate in quantity not associated with blood in the sputum or positional variation. Coughing was associated with intermittent chest pain on the left side which reduced in intensity by lying on the same side. Fever was high grade associated with chills and rigors and was relived with medication. She had decreased breath sounds on the left side with no evidence of respiratory distress.

The child was diagnosed as a case of hyper IgE syndrome at 9 years of age with persistent eosinophilia and elevated serum Ig E levels (78566 IU/ml). She used to present with repeated episodes of infections in the form of boils over the scalp, neck and buttocks, repeated cold and cough with fungal sinusitis (Aspergillus flavus) requiring FESS. The child also has bilateral sensorineural deafness and uses hearing aids. She has hypertelorism with squint and mild proptosis with a depressed nasal bridge and findings suggestive of seborrheic dermatitis.

There was no family history of similar complaints in siblings or other members, no history of tuberculosis contact.

The child was treated with oral antibiotics but the symptoms did not resolve. She was further investigated and a chest x ray showed a left sided pleural effusion. A pleural tap was done which grew Haemophillus influenza and an inter-costal drain was placed on the left side under anesthesia. In spite of treating the child with IV antibiotics and a chest drain her symptoms did not resolve. A repeat x ray showed air fluid levels in the left chest and a CT scan chest showed an infected sequestered left lobe with minimal pleural effusion.

The child was taken up for a thoracotomy and lobectomy in view of her present clinical condition. A large pus containing cavity nearly replacing the entire left lower lobe was seen with thick concretions within the cavity. The pleura was thickened with multiple adhesions. The entire left lower lobe was excised and the bronchus ligated in two layers. A large bore inter-costal drain was placed post-operatively. The patient developed a bronchial leak on the 6th post-operative day which was managed conservatively. The leak stopped and the drain was removed on the 12th post-operative day. The child had an uneventful recovery after that and was put on long term antibiotic prophylaxis.

A CASE OF HYPER IG E SYNDROME WITH LUNG ABSCESS

DISCUSSION:

Hyper IgE syndrome (HIES) originally known as Job's syndrome was first reported in 1966 when 2 patients were reported with eczematous dermatitis, recurrent boils, recurrent bone fractures, and distinctive coarse facies.[1] Males & females are affected equally. All ethnic groups are equally affected. Inheritance is both Autosomal dominant, which is common, as well as Autosomal recessive.

The underlying pathophysiology in HIES is impaired T helper cell function leading to abnormal regulation of IgE production and greatly reduced production of interferon gamma and tumor necrosis factor.[4,5] There may also be chemotactic defects in these patients.

HIES is disorder of cytokine signaling. Autosomal dominant is associated with mutations in STAT 3 gene 9 (signal transducer and the activator of transcription 3). STAT 3 signaling causes cytokine signaling by the IL-6 and IL-10

pathways.[6] IL-6 is responsible for inflammatory response, hence defective signaling is associated with minimal inflammatory response and IL-10 is a negative regulator, hence defective signaling is associated with dysregulated immune response and enhanced IgE production.

Autosomal dominant pattern present with immunologic manifestations such as staphylococcal furuncles on the head and neck, as well as chronic dermatitis. Cold abscesses are typically present in HIES due to lack of inflammatory response. Children with AD-HIES present with recurrent pneumonias, pneumatocoeles, lung abscesses and bronchiectasis. The most frequent bacterial isolates are Staphylococcus aureus, Streptococcus pneumonia, Haemophilus influenzae or fungal infections like Aspergillus. Children also have recurrent sinusitis and otitis.[2]

Pneumonias may present with fewer symptoms than would be expected in a person with intact immunity. This leads to a subsequent delay in clinical presentation and may contribute to advanced disease and significant tissue damage before identification and initiation of appropriate therapy. The severity of lung tissue damage and the subsequent emergence of chronic lung disease are higher in patients with AD-HIES as compared to those with AR-HIES.[9] These patients, therefore, require surgical interventions in the form of inter-costal drain insertions, decortications and lobectomies as they present with progressive disease which is usually not controlled by medical management. Complications of surgery like broncho-plueral fistula, bronchial stump blow out after lobectomy, poor expansion of the remaining lung after surgery are not uncommon due to extensive damage of the tissues.[10]

Broncho-pleural fistula, which developed in our patient, can be managed conservatively with an inter-costal drain, dependent drainage, antibiotics and nutritional supplementation. Surgical interventions in the form of suturing the bronchial stump with or without a vascularized muscle flap, ligating the fistula or a limited thoracoplasty to obliterate the pleural space can be attempted. Bronchoscopic interventions can be attempted for fistulae less than 5 mm using sealant agents like cryoprecipitate fibrin glue, oxidized regenerated cellulose, gel foam or autologous blood patch.[11]

Somatic manifestations include coarse facial features characterized by a broad nasal bridge and nose with a bulbous tip and hypertelorism which is seen in our patient. Dental abnormalities present as retained primary teeth. Skeletal abnormalities include hyperextensible joints, minimal trauma fractures and scoliosis more than 10 degrees.[3]

A CASE OF HYPER IG E SYNDROME WITH LUNG ABSCESS

Autosomal recessive pattern is associated with recurrent viral infections for e.g. Molluscum contagiosum, herpes simplex virus and varicella. There may be associated with neurological symptoms ranging from facial paralysis to hemiplegia. There is absence of pneumatocoeles, dental problems, skeletal abnormalities or characteristic facies.[2]

Diagnosis is by clinical history, pronounced blood eosinophilia, high serum IgE concentration >2000 IU/ml with normal concentration of IgG, IgM & IgA.

Treatment of HIES is supportive.[7,8] Prophylactic antibiotics such as cloxacillin or cotrimoxazole are used which helps prevent staphylococcal infections. Pneumonia and deep seated infections should be aggressively treated with parenteral antibiotics with surgical drainage of deep seated infections. Surgical treatment can either be via a thoracoscopic approach or by open surgery. The procedures can involve placing an inter-costal drain to drain effusion, decortication of the pleura, removal of the affected lung segment or the lobe which has formed an abscess.

Monitoring for scoliosis & fractures is essential. Dermatitis should be treated with topical steroids. Retained primary teeth may necessitate extraction.

Plasmapheresis, cyclosporine, Intravenous IgG and Recombinant human IFN-gamma are also known treatment modalities which are used depending upon individual patient requirements.[7]

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CASE REPORT: MUCKLE WELLS SYNDROME

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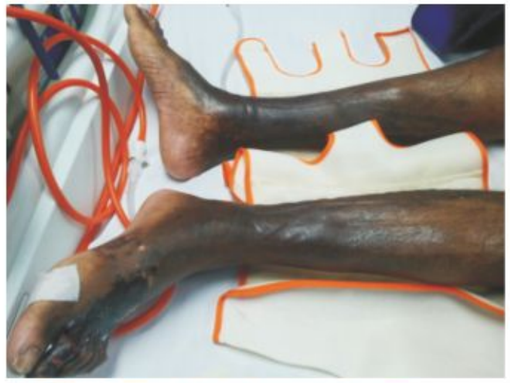
BACKGROUND

Muckle-Wells syndrome, a rare disorder, has been reported in many regions of the world, but its prevalence is unknown. It is characterized by periodic episodes of skin rash, fever, and joint pain. Progressive hearing loss and kidney damage also occur. It is uncommon in India, and this is probably first report of such case from India.

CASE:

62 yrs old dentist, presented with loose motions for 1 month. He had hearing impairment since early childhood, and progressively increasing blackening of both legs for past several years.



SCLERODERMOID LESIONS WITH HYPERPIGMENTATION AND SCLEROSIS

He had normal S. Creatinine to begin with but 1 week back, it was 9mg/dl and 17mg/dl on admission. He was admitted with lethargy, loss of appetite, breathlessness and decreased urine output. He had high anion gap metabolic acidosis, hyponatremia, hyperkalemia and TLC of 32,000. He was initiated on dialysis.

FAMILY HISTORY:

He is a father of 2 sons, the elder son is normal but the younger son, studying in USA, developed meningitis, no cause was found (Serositis). He has deafness and now quadriplegia.

COURSE:

In view of h/o deafness, audiogram was done which showed bilateral sensory neural deafness. However, ophthalmic examination did not show spherophakia, lenticonus or retinal changes, thus ruling out Alport's syndrome (A type of hereditary nephritis with SN deafness and ophthalmic changes). He was continued on dialysis and supportive care. We decided to do skin biopsy suspecting amyloidosis as a cause and also review the colonic biopsy done earlier for his loose motions. Both these showed presence of AA type of amyloidosis. The question was: What is the reason for this type of amyloidosis?

We were concerned about deafness in father and son. With diagnosis of amyloidosis confirmed, the only other possible diagnosis we thought was of Familial Mediterranean Fever (FMF). But does FMF occur in India? And how common is deafness in FMF? Or, are we dealing with a group of disorders with common or overlapping features??

BACKGROUND:

Cryopyrin-Associated Periodic Syndromes (CAPS) are a group of rare, inherited, auto inflammatory diseases with the same genetic basis and overlapping symptomatology. There are 3 subtypes of CAPS:

MUCKLE WELLS SYNDROME

- Familial Cold Autoinflammatory Syndrome (FCAS)
- Muckle-Wells Syndrome (MWS)
- Neonatal-Onset Multisystem Inflammatory Disease (NOMID) (also called Chronic Infantile Neurologic Cutaneous Articular, or CINCA, Syndrome)

HEREDITARY PERIODIC FEVER SYNDROMES (HPFS):

Six periodic fever diseases have been well characterized over the last few years, and considerable recent progress has been made in identifying causative genes and developing treatment options

- Familial Mediterranean fever (FMF)
- Hyperimmunoglobulinemia D with periodic fever syndrome (HIDS)
- Tumor necrosis factor (TNF) receptor - associated periodic syndrome (TRAPS)
- Muckle-Wells syndrome (MWS)
- Familial cold autoinflammatory syndrome (FCAS)
- Chronic infantile neurologic cutaneous articular syndrome (CINCA), also known as neonatal-onset multisystem inflammatory disease (NOMID)

MWS, FCAS, and CINCA are also known as cryopyrin-associated periodic syndromes (CAPS).

FMF:

The familial Mediterranean fever (FMF) gene (MEFV) encodes pyrin, a major regulator of the inflammasome platform controlling caspase-1 activation and IL-1β processing. Pyrin has been shown to interact with the gene product of NLRP3, NALP3/cryopyrin, also an important active member of the inflammasome. Mutations in the MEFV gene have been identified in most patients. These include 4 conservative missense mutations (M680I, M694V, M694I, and V726A) clustered in exon 10, which, together with mutation E148Q in exon 2, account for the vast majority of familial Mediterranean fever chromosomes identified in patients with the disease.

MWS:

Muckle-Wells syndrome, a rare disorder, has been reported in many regions of the world, but its prevalence is unknown. It is characterized by periodic episodes of skin rash, fever, and joint pain. Progressive hearing loss and kidney damage may also occur. It is uncommon in India, and this is probably first report of such case from India. MWS has inflammatory symptoms similar to those of FCAS, but they may be more chronic and have random, unknown triggers. Stress, exercise, or cool temperatures may also be triggers. Episodes of rash, fever/chills, arthralgia, conjunctivitis, and fatigue can last 24 to 48 hours. Patients may develop progressive, adolescent onset sensorineural hearing loss.MWS poses a risk for reactive amyloidosis (deposition of amyloid fibrils in the kidneys, heart, and other organs), with renal dysfunction as a potential consequence (our patient did not have features of amyloidosis on 2DE).

Summary of the Genes and Proteins of the Hereditary Periodic Fever Syndromes

Syndrome	Gene and Locus	Protein	Mode of Inheritance
FMF	*MEFV*, 16P13.3	Pyrin, marenostrin	Autosomal recessive
HIDS	*MVK*, 12q24	Mevalonate kinase (MK)	Autosomal recessive
TRAPS	*TNFRSF1*, 12p13	TNF-receptor type 1	Autosomal dominant
MWS	*NLRP3 (CIAS1)*, 1q44	Cryopyrin (NALP3/PYPAF1)	Autosomal dominant
FCAS	*NLRP3 (CIAS1)*, 1q44	Cryopyrin (NALP3/PYPAF1)	Autosomal dominant
CINCA	*NLRP3 (CIAS1)*, 1q44	Cryopyrin (NALP3/PYPAF1)	Autosomal dominant

MUCKLE WELLS SYNDROME

MEFV GENE MUTATION:

For patients with no identifiable MEFV gene mutation, genetic diagnosis helps early diagnosis, novel NLPR3 mutations are found related to typical CAPS phenotype.

GENETIC TESTING - OUR PATIENT:

Analysis of all coding exons with flanking intronic regions of MEFV gene by bidirectional sequencing showed compound heterozygote (including heterozygous missense variants c.443G>C/p.E148Q and c.605G>A/p.R202Q in exon2). Heterozygous mutations p.E148Q and p.R202Q are individually reported as pathological variants, consistent with the diagnosis of Familial Mediterranean Fever. This case has clinical features of Muckle Wells syndrome. We should have done genetic mutation study for NLRP3 (CIAS1). However, patient succumbed to severe sepsis before doing further tests.

TREATMENT:

Patient was started on colchicine without response. We were trying to get kineret (anakinra), recombinant, non-glycosylated synthetic form of human Interleukin-1ß receptor antagonist. But the patient succumbed to septicemia.

CONCLUSION:

This patient had clinical features suggestive of MWS. There is a mutation in MEFV gene, but SN deafness favors diagnosis of MWS. Finding of novel NLPR3 mutations would have confirmed the diagnosis of MWS

Note: The genes MEFV and NLRP3 (CIAS1) belong to the pyrin gene family based on their nucleotides sequences and predicted protein structures.

This case has been selected for publication in the American Journal of Nephrology, which will publish abstracts accepted during Annual conference of American Society of Nephrology, 2014.

The case was also selected for oral presentation during 30th Annual conference of Indian Society of Nephrology - West Zone chapter, 2014 and published in the souvenir.

We are thankful to Dr Thampi, our histopathologist for correct diagnosis of the disease, Dr Parasramani for doing skin biopsy and Dr Mrs Dhanjeebhoy for helping us get genetic testing done. We are also thankful to the patient's family who were very keen at getting the diagnosis and further treatment.

ALL CASES OF "GRANULOMATOUS LYMPHADENITIS" ARE NOT TUBERCULOSIS

Dr. Asha George *MD (Path),* **Dr. Chandralekha Tampi** *MD (Path)*

INTRODUCTION

Tuberculosis is one of the major causes of morbidity and mortality in India.

While investigating a patient, there are several investigations and clinical features that favour the possibility of tubercular infection but it is the finding of granulomas, on microscopy, that strongly supports such a diagnosis. Frequently patients are started on anti-tubercular treatment on the basis of an FNA report of granulomatous lymphadenitis.

However, one should be aware that though Tuberculosis is the cause of granulomas in most cases, similar granulomas can also be found in several other infectious and sometimes even in neoplastic conditions. Microbiological culture of Mycobacterium tuberculosis remains the confirmatory test for tuberculosis

We present two such cases where patients, who showed granulomas on FNAC, did not respond to anti tubercular treatment, and a lymphnode biopsy subsequently done, revealed a different disease.

Case No. 1

A 36 year old male patient presented with fever, weight loss and axillary lymphadenopathy

An FNAC done elsewhere had shown caseating granulomatous inflammation and he had been started on AKT. However the response was poor.

He was then referred to our Institute, and an enlarged axillary node was then excised.

The histology of the lymphnode showed caseating granulomas (Fig-1) and several Langhan's giant cells. However, on careful examination, small yeast like micro-organisms were observed within the giant cells.

PAS stain Confirmed numerous Histoplasma capsulatum organisms (Fig-2) in the cytoplasm of the giant cells, and he was diagnosed to have Histoplasmosis and not Tuberculosis.

His AKT was stopped, and he was put on specific therapy with good response.

DISCUSSION

Fungal infections of lymphnodes can present as chronic suppurative lesions, as granulomatous processes, or a combination of the two.

Caseation, a sine qua non of Tuberculosis, is also sometimes seen in fungal infection - Histoplasmosis as demonstrated in this case .

Langhans giant cells are a non specific feature of granulomas and do not imply that the granuloma is Tubercular.

Histoplasmosis is caused by a fungus - Histoplasma capsulatum. The infection occurs when small spores - microconidia are inhaled from soil contaminated with bat or bird droppings.

It has many similarities to Tuberculosis.[1] 90% of human infections are latent and remain asymptomatic.

The remaining 10% cases which develop symptomatic infections, closely mimic tuberculosis clinically and radiologically.

The chronic form can produce unilateral cavities in the upper lung lobes and the disseminated forms[2] can involve various organs like lungs, lymph nodes, spleen, liver, bone marrow, GI tract and adrenal glands.

The treatment is Amphotericin B.

Studies have shown that histoplasmosis is endemic in parts of India, is often under recognized, and mistaken as Tuberculosis[3].

ALL CASES OF "GRANULOMATOUS LYMPHADENITIS" ARE NOT TUBERCULOSIS

Case No 2 :

A 12 year old girl underwent cervical lymph node biopsy. A previous FNAC done elsewhere had shown granulomas based on which she was being treated for Tuberculosis. Since she did not respond, she was referred to Lilavati hospital. A lymph node biopsy was done

Microscopic examination showed a background of non caseating granulomas (Fig-3), histiocytes, lymphocytes, eosinophils and few plasma cells,within which scattered binucleate and mononuclear R.S. Cells (Fig-4) were present. A diagnosis of Hodgkins Lymphoma was made[4]

IHC studies done confirmed the diagnosis of Classical Hodgkin's Lymphoma, Mixed Cellularity Type.

DISCUSSION :

Hodgkin's lymphoma (HL) is a malignancy of the lymph nodes.

HL can simulate tuberculosis clinically[5] as most often (75% cases), it involves lymph nodes of the cervical region followed by the mediastinal region and is sometimes accompanied by fever and weight loss.

Majority of the cases manifest clinically in young adults.

A lymph node biopsy with immunohistochemistry (IHC) studies is necessary to diagnose the condition.

Though FNAC is a cost effective diagnostic test, and presence of granulomas in most instances is a marker of Tuberculosis, yet it is important to keep in mind especially in non responding patients, that other than resistant tuberculosis, the granulomas may actually represent a different disease altogether.

Therefore in all non responders, excision of an intact enlarged node and histopathological examination, and microbiological examination is mandatory .

COMMON CAUSES OF GRANULOMATOUS LYMPHADENITIS.

1) **Suppurative / Necrotizing granulomas (in order of incidence)**
   - Tuberculosis.
   - Atypical mycobacteriosis
   - Fungal infections.
   - Cat scratch disease.
   - Brucellosis.
   - Lymphogranuloma venereum.
2) **Non necrotizing granulomas**
   - Tuberculosis
   - Sarcoidoisis
   - Toxoplasmosis.
3) **Malignancies associated with granulomatous inflammation**
   - Hodgkin's Lymphoma
   - NHL esp. T cell lymphomas
   - Seminoma / Dysgerminoma.
   - Nasopharyngeal carcinoma.

ALL CASES OF "GRANULOMATOUS LYMPHADENITIS" ARE NOT TUBERCULOSIS

Granulomas are also often seen in the draining nodes around tumor cells.

Thus though tuberculosis is the usual cause in a case of granulomatous lymphadenitis, there are other conditions, also, to be kept in mind, especially if the patient is not responding to therapy. Fungal stains, routinely performed, on all granulomatous inflammations, as is done in our Institute, is a simple way, to pick up some of these unusual cases. The actual visualization of AFB by special stain or culture remains the confirmatory test for Tuberculosis.

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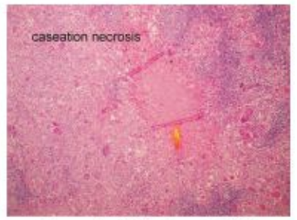

Figure 1: *(H & E stain 100X)*
Caseating granulomas in Histoplasmosis.

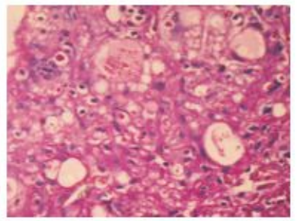

Figure 2: *(PAS stain 400X).*
PAS stain showing yeast forms of Histoplasma capsulatum

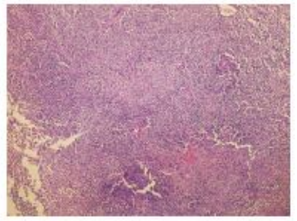

Figure 3: *(H & E stain 100X)*
Non caseating granulomas in Hodgkin's Lymphoma.

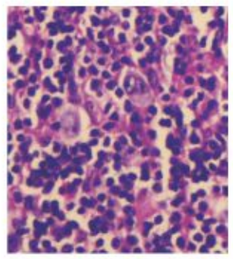

Figure 4:*(H & E stain 400X)*
Reed Sternberg (RS) cells in Hodgkin's lymphoma.

SEWA

The social service wing of the hospital-**SEWA**-serves to the health requirements of the needy people. This department seeks to bridge the gap between the needy patients and the fast evolving medical technology. Various social activities such as Free OPD, services to senior citizen, sending mobile vans to Adivasi areas to organizing free health checkup camps are undertaken as an on-going process. The Roshni Eye bank managed by Lilavati Hospital is a well equipped comprehensive centre for cornea removal, supplying, processing, storing, and corneal transplantation.

BENEFICIARIES

Year	Free OPD	Sewa Mobile Clinic
2011-2012	16327	23010
2012-2013	14965	24211
2013-2014	14301	30232

Free OPD

Sewa Roshni Eye Bank

Sewa Nana Nani

Mobile Medical Clinic

SEWA ORGANIZES FREE HEALTHCHECKUP CAMPS

SEWA
Service Equality For The Welfare Of All

Free Healthcheckup Camps



Lonawala Nagar Parishad



Bharat English School, Thane *Ashram Shala Sanegaon, Roha* *Anjuman Islam School, Kurla*


National English School, Virar (W) *Carmalite English School, Vasai (W)*

Rural Adivasi Area, Dahanu *Slum Area, Dharavi*

EDUCATIONAL ACTIVITIES

Lilavati Hospital and Research Centre doctors share their intellectual capital and expertise with others through CMEs using means like workshops, seminars, conferences, live telecast of procedures and surgeries, which they are performing. Lilavati Hospital and Research Centre has been accredited by Maharashtra Medical Council for conducting CMEs.

Sr. no.	Topic	Organized Month
1	Newer Aspects in Dermatology	January
2	Peadiatric Endocrinology	January
3	Immuno Histochemistry	January
4	Plastic Surgery	February
5	Pediatric Neurology	April
6	Nuclear Medicine in General Practice	June
7	Andrology	July
8	Constipation in children (Pediatric Surgery)	July
9	Infectious Disease	August
10	Pain Management	October

Pain Management

Newer Aspects in Dermatology

Immuno Histochemistry

LATEST FEATHERS IN CAP...

Efforts and hard work put in by team Lilavati Hospital has resulted in various awards and accolades:

- *Hospital has been recognized as **"India's best Multi Speciality Hospital-Megapolis"** by ICICI Lombard and CNBC TV 18 **in India Healthcare awards 2013.***
- *Hospital has been recognized as **"India's best Multi Speciality Hospital-Metro"** by ICICI Lombard and CNBC TV 18 **in India Healthcare awards 2012.***
- *In 2013, 2014 'THE WEEK' magazine has rated Hospital as **'Number 1 Multispeciality Hospital in Mumbai'.***
- *Hospital has been rated amongst **'Top 10 Hospitals of India'** 2013, 2014 by 'THE WEEK' magazine.*
- *Hospital is **Gold Winner** of **"Reader's Digest Trusted Brand Award 2012** & 2008" in category 'Speciality Hospital'.*
- *Hospital is an official **ESMO (European Society for Medical Oncology) Asia CME Partner** Centre in Colorectal Cancer program in India.*
- *Quality Council of India (QCI) has accredited Lilavati Hospital & Research Centre with **NABH** in February, 2011 and Reaccredited in 2014.*
- *Winner in the category **"Most Popular Maternity Hospital (All-India)"** in the 2nd edition of Child Most Popular Awards, 2014, Child India Magazine.*
- *Hospital emerged as the **Runner-Up** in the category **India's Most Popular Maternity Hospital;** in the inaugural edition of **Child Best Awards 2013** by Child India Magazine.*

SERVICES

MEDICAL
Anesthesiology
Audiology and Speech therapy
Cardiology
Chest Medicine
Chronic Pain management
Dental
Dermo Cosmetology
Diabetology & Endocrinology
Gastroenterology
Haematology
Hair Transplant
Internal Medicine
Infectious diseases
Nephrology
Neurology
Head and Migraine Clinic
Psychiatry / Psychology / Neuropsychology
Medicine Oncology
Pediatrics
Rheumatology
Physiotherapy
Sleep Medicine

SURGICAL
Bariatric Surgery
Cardiothoracic Surgery
Colorectal Surgery
ENT and Head & Neck Surgery
Gastro Intestinal Surgery
General Surgery
Gynecology, Obstetrics & IVF
Transplant: Corneal & Kidney
Minimal Invasive Surgery (Laproscopic Surgery)
Neuro Surgery
Spine Surgery
Onco Surgery
Ophthalmology
Orthopedics, Sports Medicine
Pediatric Surgery
Plastic & Reconstruction Surgery
Urology, Andrology
Vascular Surgery

CRITICAL CARE
Intensive Care Unit (ICU)
Intensive Cardiac Unit (ICCU)
Surgical Intensive Care Unit (SICU)
Paediatric Intensive Care Unit (PICU)
Neo-Natal Intensive Care Unit (NICU)
Paralysis & Stroke Unit

DIAGNOSTICS
Imaging Services
CT
MRI
X-ray
BMD
OPG
Sonography (USG)
Mammography
Nuclear Medicine
Interventional Radiology

LABORATORY SERVICES
Pathology
Microbiology
Histopathology
Blood Bank

24 HRS SERVICES
Ambulance
Emergency
Pharmacy

DOCTORS ASSOCIATED WITH LILAVATI HOSPITAL

Andrologist
Dr. Shah Rupin S.

Anaesthesiologist
Dr. Mascarenhas Oswald
Dr. Barot Hemangini
Dr. Baxi Vaibhavi
Dr. Budhakar Shashank
Dr. Gandhi Nisha
Dr. Gaiwal Sucheta
Dr. Gawankar Prakash
Dr. Kharwadkar Madhuri
Dr. Kulkarni Satish K.
Dr. Mahajan Anjula
Dr. Khatri Bhimsen
Dr. Shah Falguni
Dr. Joshi Kunal

Audiology & Speech Therapists
Dr. Bhan Satyan
Dr. Gorawara Pooja
Dr. Parulkar Bakul
Dr. Patadia Rajesh
Dr. Thakur Zohaa

Cardiovascular & Thoracic Surgeons
Dr. Bhattacharya S.
Dr. Jaiswal O. H.
Dr. Kaushal Pandey
Dr. Kumar Pavan
Dr. Rachmale G. N.
Dr. Nand Kumar
Dr. Mehra Arun P.
Dr. Shetty Mohan
Dr. Joshi Suresh
Dr. Honnekeri Sandeep T.

Cardiologists
Dr. Ballani Prakash H.
Dr. Bang Vijay
Dr. Dargad Ramesh R.
Dr. Gokhale Nitin S.
Dr. Hemant Kumar
Dr. Jhala Darshan
Dr. Kothari Snehal N.
Dr. Lokhandwala Yash
Dr. Mehan Vivek
Dr. Mehta Ashwin B.
Dr. Merchant S. A.
Dr. Menon Ajit R.
Dr. Nabar Ashish
Dr. Punjabi Ashok H.
Dr. Samuel K. Mathew
Dr. Sanzgiri P. S.
Dr. Shah Chetan
Dr. Sharma Anil K.
Dr. Suratkal Vidya
Dr. Vijan Suresh
Dr. Vyas Pradeep R.
Dr. Vora Amit
Dr. Vaishnav Sudhir
Dr. Vajifdar Bhavesh
Dr. Mehta Haresh G.

Chest Medicine
Dr. Mehta Sanjeev K.
Dr. Prabhudesai P. P.
Dr. Parkar Jalil D.
Dr. Rang Suresh V.
Dr. Chhajed Prashant

Colorectal Surgery
Dr. Chulani H. L.

Dentistry / Dental Surgeons
Dr. Bhavsar Jaydeep P.
Dr. Deshpande Dilip
Dr. Gala Dhimant
Dr. Joshi P. D.
Dr. Khatavkar Arun
Dr. Kamdar Rajesh J.
Dr. Parulkar B. P.
Dr. Parulkar Darshan
Dr. Sanghvi Sameer

Department of Imaging
Dr. Bajaj Anita
Dr. Deshmukh Manoj
Dr. Kulkarni Makrand
Dr. Mehta Mona
Dr. Ingule Amol
Dr. Chauhan Sonal
Dr. Sobti Shyam K.

Dermatologists
Dr. Goyal Nilesh
Dr. Oberai Chetan
Dr. Mehta Nimesh
Dr. Parasramani S. G.

Diabetologists
Dr. Joshi Shashank R.
Dr. Panikar Vijay

ENT Surgeons
Dr. Chaturvedy Gaurav
Dr. D'souza Chris E.
Dr. Kapadia Sanjay P.
Dr. Pusalkar A.
Dr. Parasram Kamal S.

Gastro Surgeons
Dr. Bharucha Manoj
Dr. Kulkarni D. R.
Dr. Mehta Hitesh
Dr. Varty Paresh
Dr. Wagle Prasad K.
Dr. Zaveri Jayesh P.

Gastroenterologists
Dr. Barve Jayant S.
Dr. Gupta Ravi
Dr. Kanakia Raju R.
Dr. Khanna Sanjeev
Dr. Phadke Aniruddha Y.
Dr. Parikh Samir S.
Dr. Shah Saumil K.

General Surgeons
Dr. Garud T. V.
Dr. Mehta Narendra
Dr. Shastri Satyanand B.
Dr. Shetty Sadanand V.
Dr. Trivedi Narendra

Gynaecologist
Dr. Agarwal Rekha
Dr. Coelho Kiran S.
Dr. Dhanu Ranjana V.
Dr. Dhanu Vilas R.
Dr. Nanavati Murari S.
Dr. Pai Rishma D.
Dr. Palshetkar Nandita
Dr. Pai Hrishikesh
Dr. Shah Cherry C.
Dr. Goyal Swarna

Haematology Clinical
Dr. Agarwal M. B.
Dr. Bhave Abhay

Headache & Migraine Clinic
Dr. Ravishankar K.

Infectious Diseases Consultant
Dr. Nagvekar Vasant C.

Intensivist
Dr. Vas Conrad Rui
Dr. Ansari Abdul

DOCTORS ASSOCIATED WITH LILAVATI HOSPITAL

Interventional Radiologists
Dr. Sheth Rahul
Dr. Warawdekar Girish
Dr. Limaye Uday S.

Joint Replacement Surgeons
Dr. Maniar Rajesh N.

Nephrologists
Dr. Mehta Hemant J.
Dr. Shah Arun
Dr. Suratkal L. H.
Dr. Upadhyaya Kirti L.

Neurologists
Dr. Chauhan Vinay
Dr. Sirsat Ashok M.
Dr. D'souza Cheryl
Dr. Dalal P. M.
Dr. Vyas Ajay

Neuropsychologist
Dr. Panjwani Siddika

Neuro Surgeons
Dr. Ramani P. S.
Dr. Goel Atul
Dr. Dange Nitin

Nuclear Medicine
Dr. Lele R. D.
Dr. Luthra Karuna

Oncologists
Dr. R. Gopal
Dr. Smruti B. K.
Dr. Pendharkar Dinesh

Oncosurgeons
Dr. Deshpande Ramakant K.
Dr. Chabra Deepak
Dr. Jagannath P.
Dr. Parikh Deepak
Dr. Sharma Sanjay
Dr. Shah Rajiv C.

Ophthalmology
Dr. Agrawal Vinay
Dr. D'souza Ryan
Dr. Mehta Salil
Dr. Nadkarni Shivram
Dr. Nagvekar Sandip S.
Dr. Shah Manish
Dr. Vaidya Ashish R.
Dr. Mehta Himanshu

Orthopaedic Surgeons
Dr. Agrawal Vinod
Dr. Archik Shreedhar
Dr. Chaddha Ram
Dr. D'silva Domnic F.
Dr. Desai Sanjay S.
Dr. Deshmukh Niranjan
Dr. Garude Sanjay
Dr. Joshi Anant
Dr. Kohli Amit
Dr. Mukhi Shyam R.
Dr. Nadkarni Dilip
Dr. Padgaonkar Milind
Dr. Panjwani Jawahar S.
Dr. Vengsarkar Nirad S.
Dr. Vatchha Sharookh P.
Dr. Warrier Sudhir
Dr. Thakkar C. J.

Pathologists
Dr. Chavan Nitin
Dr. Dhunjibhoy Ketayun R.
Dr. George Asha Mary
Dr. Rangwalla Fatema
Dr. Mehta Kashvi
Dr. Saraswat Shubhangi (Blood Bank)
Dr. Tampi Chandralekha

Paediatric Surgeons
Dr. Karmarkar Santosh J.
Dr. Redkar Rajeev G.
Dr. Nathani Rajesh

Paediatricians
Dr. Ali Uma
Dr. Avasthi Bhupendra
Dr. Chittal Ravindra
Dr. Gupta Priyam
Dr. Kanakia Swati R.
Dr. Lokeshwar M. R.
Dr. Mehta Kamini
Dr. Shah Krishnakumar N.
Dr. Sharma Shobha
Dr. Ugra Deepak

Paediatric Cardiology
Dr. Changlani Deepak K.

Chronic Pain Management
Dr. Baheti Dwarkadas
Dr. Jain Jitendra

Physicians / Internal Medicine
Dr. Ballani A. G.
Dr Bandukwala S. M.
Dr. Nair C. C.
Dr. Dalvi Sunil G.
Dr. Jadwani J. P.
Dr. Gidwani Vinod N.
Dr. Medhekar Tushar P.
Dr. Shimpi Shrikant

Plastic Surgeons
Dr. Kumta Samir
Dr. Pandya Narendra
Dr. Purohit Shrirang

Psychiatrist
Dr. Deshmukh D. K.
Dr. Shah Bharat R.
Dr. Vahia Vihang N.

Psychologist
Dr. Chulani Varkha

Physician / Rheumatologist
Dr. Sangha Milan
Dr. Kalke Shubhada

Physiotherapist
Dr. Garude Heena

Spine Surgeon
Dr. Bhojraj Shekhar
Dr. Mohite Sheetal

Urologists
Dr. Pathak Hemant R.
Dr. Raina Shailesh
Dr. Sanghvi Nayan
Dr. Shah Sharad R.
Dr. Vaze Ajit M.
Dr. Raja Dilip

Urological Laparoscopic Surgeon
Dr. Ramani Anup

Vascular Surgeons
Dr. Patel Pankaj
Dr. Pai Paresh

In Remembrance of Late Dr Samir Desai

Ode To An Illusionist

One Hundred And Fifty Pairs Of Eyes,
Remained Transfixed,
On That Houdini Like Figure,
Mesmerised, By His Hypnotic Eyes,
And Deep Baritone Voice,
Waiting For That Sleight Of Hand;
But They Were Disarmed....
By His Easy Smile,
And Charm..
And They Relaxed....
That Is When He Struck...
With His Disappearing Act.
His Body Remained Limp..
But He Was Far Away....!
They Knocked...
They Called...
They Pleaded....
They Cajoled....
But He Had Transported Himself
.....to Another World.
(adieu My Friend)

Dr. L. H. Suratkal