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Super Speciality Hospital

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Right Upper and Middle Lobe Lobectomy on ECMO – a technical challenge

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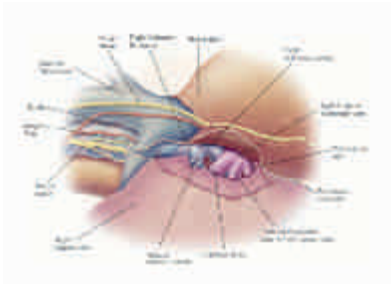
Extracorporeal membrane oxygenation (ECMO) is a rescue therapy for critically ill patients with reversible cardio-respiratory pathology and those who have probability of death around 80% despite maximal conventional treatment.Here we describe a unique case with multiple bronchopleural fistula in right lung. He underwent right upper and middle lobe lobectomy while on ECMO support a young craftsman from East Delhi got a new lease of life after he was put on ECMO and operated for Lobectomy procedure.



A 41 yr old diabetic male was suffering from kleibsiella pneumonia. He went into adult respiratory distress syndrome due to severe pneumonia and was taken on ventilator in another hospital. He was on 100% FiO2 for the last 7 days and deteriorating when Max Saket ECMO team was called to see him if he can be a candidate for ECMO insertion. He suffered from Acute Pneumonia due to which he endured complete lung failure. His right lung was damaged to the extent that he could not manage to survive even on ventilator with 100% oxygen. He was almost on the death bed and the family had lost all hope.

We instituted venovenous ECMO onsite and shifted on ECMO to our center. After four days on ECMO other organs recovered and hewas well alert. His air leak was so massive that 90% of the tidal volume was leaking through broncho pleural fistula. While he was on ECMO, his upper and middle lobes in the right

lung were successfully removed, which is a very difficult procedure to do while the patient is on ECMO. Over a period of a week, rest of the right lung expanded in the cavity and acted as the lung with perfect functioning. On day 4 after lobectomy he suddenly started bleeding from chest tubes.He was taken for re-exploration and it was only oozing from the chest wall.No active bleeder was found.On day 5 after the lobectomy his ECMO was separated once the lung functions recovered.He was given factor 7 after ECMO separation. His bleeding was controlled. After few days his ventilator was removed and he was brought back to dependence on his natural lungs.

In summary, ECMO is salvage therapy in patients with life threating refractory severe ARDS. Lobectomies can be safely performed on ECMO. Meticulous hemostasis is required to avoid complications of heparinization. ECMO should be separated after thoracotomy as soon as lung functions recoverto avoid spontaneous bleeding from raw surfaces. This is a successful case of Lobectomy procedure done on an adult patient while on ECMO. A rare case in medical literature where the patient's life was saved when he was almost on the death bed. The families who are into a small scale business of handicrafts are delighted with the patient's recovery and grateful to the ECMO team for giving a new lease of life to him. He has been doing well after that.

CHOLERA
– an ancient but silent killer

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CLINICAL VIGNETTE

A 7 year old girl presented in the Emergency with watery diarrhea and vomiting. She had developed the symptoms just one day previously, and the quantity of stools were not known. However she could not tolerate any oral liquids due to repeated vomiting. The parents didn't know when she had last passed urine. Other family members were unaffected.

She was carried by her father, and appeared pale and stuporous, with only minimal response to pain. She had sunken eyes, reduced skin turgor, and acidotic breathing, with a heart rate of 135/min, respiratory rate 34/min, BP 69/45mmHg; the pulse was thready with only central pulses being felt, cold clammy peripheries, and a scaphoid abdomen with normal bowel sounds. The mucous membranes were dry, and she had no fever. The chest was clear, and she had no meningeal signs.

She was admitted to the PICU for resuscitation: rapid fluid administration of up to 1000mL (60mL/kg) was required before hypotension was corrected. The venous blood gas confirmed severe metabolic acidosis (pH 7.1, HCO3 11mmol/L, BE -18mmol/L), blood glucose was 86mg/dL, there was prerenal azotemia (urea 53.5mg/dL, creatinine 1.5mg/dL), extreme hemoconcentration (hematocrit 53.5%), and leucocytosis (25,800 with 86% neutrophils). Fluid therapy was continued to replace ongoing fluid losses, and vomiting was controlled with antiemetics. She voided urine within 6 hours. She required a total of 160mL/kg of fluids in the first 24 hours.

Stool purge rate remained high and it was noted that the stools were watery with a pale whitish appearance, like rice water. Cholera was suspected and stool examination was requested. She was placed in isolation, and appropriate barrier precautions were instituted. Darting motility was confirmed by light microscopy, and stool was put up for culture. Oral ciprofloxacin was added at 15mg / kg / dose q12h. After enrichment with alkaline peptone water, stool culture returned positive for Vibrio cholerae. It was resistant to cotrimoxazole, but sensitive to fluoroquinolones, ampicillin, and chloramphenicol.

Stool output rapidly reduced after ciprofloxacin, and had stopped by 24 hours. Hydration had been restored by this time, and the child began feeding orally. Acidosis resolved, and hemoconcentration and azotemia were completely reversed, with a return to baseline hematocrit of 43% and creatinine 0.6mg/dL. She was discharged in a stable condition after two days with advice to complete ciprofloxacin therapy for three days.

This case highlights the timely recognition of hypovolemic shock and successful treatment of cholera with appropriate fluid and antibiotic therapy. It is important to suspect cholera in every child especially over 5 years of age with profuse watery diarrhea and repeated vomiting. Rapid diagnosis can be achieved by examining a fresh stool sample for darting motility characteristic of Vibrio cholerae.

BRIEF REVIEW

Cholera is an ancient diarrheal disease that probably originated in India around the Gangetic delta and adjoining Bay of Bengal. It affects nearly 3-5 million people annually, and causes 100,000-130,000 deaths every year, as per the World Health Organization (WHO). Beginning in the 19th century, cholera spread from India to cause worldwide pandemics periodically. Currently the seventh of these pandemics is prevalent. Of the several serotypes of cholera only O1 and O139 cause epidemic disease; O1 exists in two biotypes, classical form and El Tor. Most isolates in the last decade belong to the O1 El Tor biotype.

Cholera is caused by intestinal infection with the Gram-negative bacterium, Vibrio cholerae, and is transmitted through the feco-oral route. These bacteria occur naturally in water of high salinity like sea coasts, but can survive with low salt

content if the water is rich in organic nutrients like contaminated urban water supplies in areas with low sanitation. They are also found attached to zooplankton and shellfish, and consuming such shellfish is a common route of infection. Contaminated water as a source of infection was recognized by the pioneering epidemiological work of Dr John Snow, who traced the source of an outbreak of cholera in London in 1854 to a hand pump contaminated with sewage. Robert Koch identified Vibrio Cholerae as the causative organism in 1883.

The infectious dose required is 105-108 bacteria, but can be as low as 103 if gastric acid is depressed. Most of the ingested bacteria are killed by gastric acid. Persons with blood group O are especially susceptible to symptomatic cholera infection. About 75% of infected persons do not develop any symptoms, and shed the bacteria in stools for a few days. Even among symptomatic individuals, 80% have only mild to moderate diarrhea. Vomiting is a prominent feature unlike other causes of childhood diarrhea. Fever is rare and usually indicates a secondary infection. Symptomatic patients may shed the bacteria for up to 2 weeks in their stool, and these have a peculiar hyperinfective property. Infecting dose from such bacteria is 10-100 times lower than environmental forms, and this enhances person-to-person transmission and causes explosive outbreaks.

In severe forms, infection can lead to fluid losses of up to 1L per hour, and patients may die within a few hours (cholera gravis). Another unusual form is the rapid accumulation of fluid within the bowel lumen with shock and death occurring even before the first loose stool has been passed (cholera sicca). Severe cholera has a mortality rate of up to 70%.

Vibrio attach to the intestinal epithelium where they produce the cholera toxin (CTX). This consists of two subunits: the B subunit attaches to the GM1 ganglioside receptor on the epithelial cell, through which the A subunit enters the cell. This is an adenylate cyclase enzyme which increases c-AMP within the cell, causing chloride secretion through the apical chloride channel and profuse secretory diarrhea. It is interesting to know that the cystic fibrosis mutation in the epithelial chloride channel (CFTR) is protective against cholera: heterozygous carriers of the CFTR mutation are resistant to cholera infection, and it has been postulated that this has provided a selective evolutionary advantage to such individuals.

The secretory diarrhea leads to profound fluid and electrolyte losses. In children, these losses are markedly different from adults; to illustrate, typical cholera stool losses of sodium, chloride, bicarbonate in children are 100, 90, and 30mmol/L of stool respectively, whereas in non-cholera diarrheas they are 50, 25, and 20mmol/L respectively. Effective treatment relies on rapid replacement of these fluid and electrolyte losses. Oral rehydration with the low-osmolality ORS is the mainstay of treatment. However if stool volume is more than 10ml/kg/hour, then intravenous fluid therapy is recommended.

Antibiotic therapy is adjunctive to fluid replacement. It reduces the duration of diarrhea, decreases stool volume by 50%, and restricts bacterial shedding to 1-2 days. In children, ciprofloxacin (15mg/kg BD for 3 days) or doxycycline 4-6mg/kg as a single dose is effective. Single dose azithromycin (20mg/kg) is also recommended in areas with fluoroquinolone resistance.

Particular attention must be paid to nutritional management in children. During the acute stage, hypoglycemia and hypokalemia can be life-threatening. Vitamin A supplementation is required for malnourished children. Zinc is also recommended for children below 5 years of age (10mg/day for children <6 months age, 20mg/day for those >6 months age, for 10 days).

Prevention by ensuring safe water and public sanitation remains the only measure to control cholera as a public health strategy. Hand hygiene, prevention of contamination of drinking water with sewage, and proper disposal of infected stools, are essential. A simple but effective method for water safety was developed in Bangladesh (sari method): filtering drinking water through a sari folded 4-8 times, reduced cholera transmission by >50% probably by eliminating zooplankton.

Two effective vaccines are available, Dukoral and Shanchol: both are oral, killed vaccines, induce mucosal immunity, and are currently recommended for high-risk and endemic areas only. Shanchol was indigenously developed by Shantha Biotechnics and Sanofi Pasteur, and field studies are being completed. It contains several biotypes and serotypes of both Vibrio Cholerae 01 and 0139 without supplemental B subunit of the toxin. Children above one year of age are eligible. Two doses, administered 14 days apart, confer 60-70% protection lasting 24-36 months. Boosters are recommended every 2 years in endemic areas. The vaccine also has an important herd protective effect of >90% when at least half the susceptible population in an endemic area is immunized. Nevertheless, the vaccine is only effective when integrated into a control programme with sanitation and water safety.

KEY MESSAGES

- Suspect cholera in any child >5 years with acute watery diarrhea and severe dehydration, even in an area where cholera is not presnt; or in any child >2 years with similar symptoms in an endemic area

- Vomiting is a prominent symptom unlike other causes of watery diarrhea in children

- Profound fluid losses can be fatal within a few hours of onset of symptoms

- Diagnosis can be confirmed by microscopy for darting motility or culture of a fresh stool sample

- Rapid fluid administration is the mainstay of treatment

- Children are especially susceptible to hypoglycemia and hypokalemia

- Antibiotic therapy is indicated to reduce duration of diarrhea and fecal shedding

- Hand hygiene, water safety, and sanitation must be emphasized

Detection of Intra Cardiac Thrombus on Cardiac MRI – a case study

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29 years young male came with history of stroke and sudden onset right hemiparesis. He visited a Neurologist in Max Super Speciality Hospital, Saket and he was advised for a MRI Brain. (month of February 2014.)

Brain MRI done which showed Left Parietal Infarct (Figure 1).

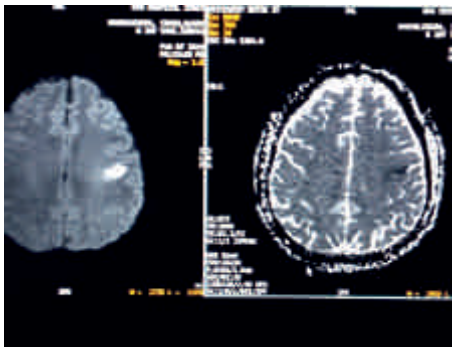

Figure 1 showing left parietal infarct.

Then patient was then referred to Cardiologist for Echocardiography to r/o if any cardiac tumor or thrombus. In Echocardiography there were multiple echogenic lesions in leftventricular cavity seems to arising from apex and septal wall of left ventricle (figure 2).

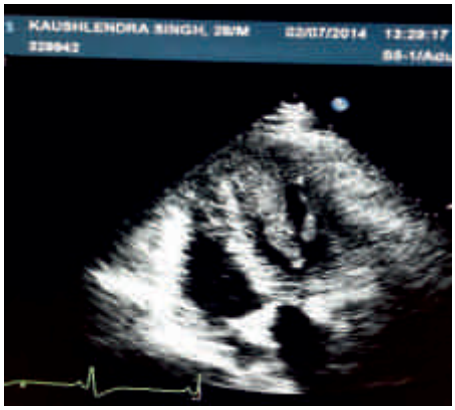

Figure 2 showing echogenic mass like lesions arising fromapico-septal region.

Provisional diagnosis on echocardiography was made as left ventricular mass or clots. In view of young age with no predisposing factor like diabetes, hypertension or any h/o chest pain again cardiactumor was considered and differential diagnosis was thrombus. To confirm thisthe patient was referred to Radiology again for Cardiac MRI. In MRI all routine sequences were done under viability protocol like Axial Fiesta, 2 and 4 Chamber Cine, Short Axis Cine, First Pass Perfusion and Late Gadolinium enhancement (LGE).

On Cine sequences LV regional function demonstrated hypokinesia of septum and apex of left ventricle.

Short axis (SAX) and 4 Chamber (4CH) images showed hypo intense mass like lesion seems to arising from apex (figure 3 and 4).

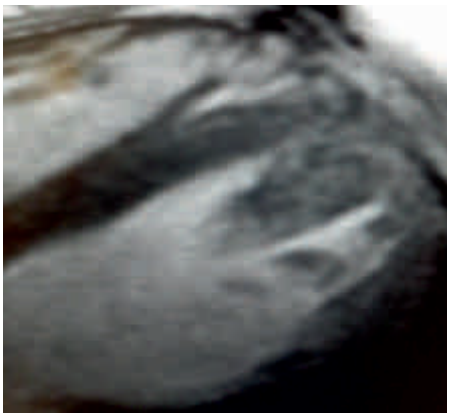

Figure 3. A 4 CH image showed hypo intense mass like lesion arising fromapex.

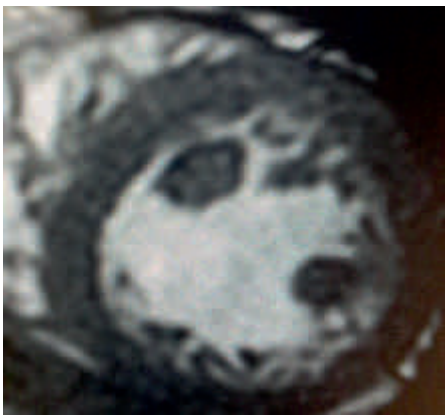

Figure 4. A SAX image showed hypo intense mass like lesion arising from apex.

In first pass perfusion delayed arrival of the enhancement is demonstrated in anterio-septal wall of left ventricle suggesting perfusion defect (figure 5).

Figure 5. (First pass perfusion) A SAX imageshowing delayed arrival of the enhancement in anterio-septal wall of left ventricle.

Then Late Gadolinium Enhancement (LGE) image showed areas of late gadolinium enhancement in the septal mid-apical segments (figure 6, 7, 8,), papillary muscles and in the sub endocardium of the lateral and inferior apical segments.

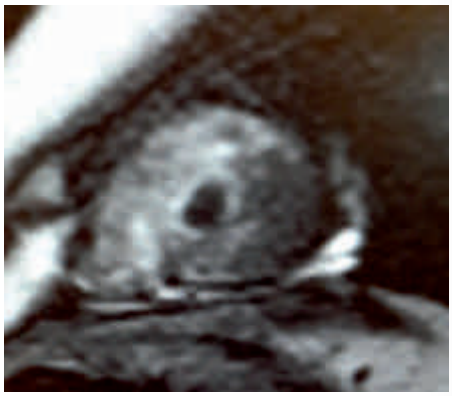

Figure 6 (sax image) showed areas of late gadolinium enhancement in the septal mid-apicalsegments.

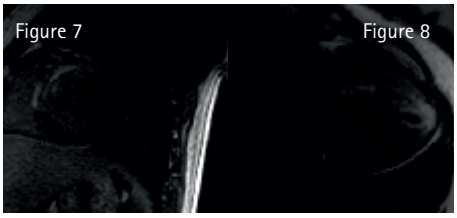

Figure 7,8 (LGE IMAGE) showed areas of late gadolinium enhancement in the septal mid-apicalsegments.

RESULTS

MRI findings were:

1. Hypokinesia of septum and apex of left ventricle.
2. In first pass perfusion delayed arrival of the enhancement is demonstrated in anterio-septal wall of left ventricle suggesting perfusion defect.
3. Late Gadolinium Enhancement (LGE) image showed areasof late gadolinium enhancement in the septal mid-apical segments papillary muscles and in the sub endocardium of the lateral and inferior apical segments.
4. Over all features were suggestive for myocardial infarct involving the territory of the LAD.

Conventional Angiography was done which showed consistent findings.

DISCUSSION

Cardiac MRI is a valuable modality for confirming and evaluating intracardiac thrombi. CMR identifies 'Mural Thrombus Formation' as an indication of medical necessity for CMR. In patients with a history of ischemic heart disease or myocardial infarction, ventricular thrombi frequently occur as complications. These thrombi can lead to stroke, pulmonary embolism, or peripheral arterial embolism. Contrast-enhanced MRI provides the highest sensitivity and specificity for LV thrombus when compared to other imaging modalities, and should be considered in the care of patients at high risk of LV thrombus formation. CMR complements other imaging modalities to detect thrombi which are hard to visualize. For example, Cardiac MRI is significantly more sensitive than echocardiography for detecting ventricular thrombi. Studies have demonstrated an approximately twofold increase in sensitivity for the detection of ventricular thrombi when comparison is made with echocardiography. Cardiac MRI is exquisitely sensitive for the detection of even small ventricular thrombi when certain techniques are conducted with intravenous contrast.

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Foreign Body in a Child's Airway

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A small foreign body lodged in a child's airway can be a catastroph. It is unimaginable, as to what a child can swallow and choke on it, lodging it in their tiny airway. The human body has many mechanisns to keep the airway clean. These include cartilages, vocal cords and cough reflex. But none of these are perfect; more so in children and foreign bodies get lodged in the airway of children.

There are many ways a child can present with a foreign body lodged in his airway. It could be a sudden catastroph where the obstruction is total and rapidly results in cardo-respirtory arrest and death. But a more common presentation is a sudden episode of coughing or choking with wheezing or stridor later. But many times the episodes are not witnessed or recalled by parents or caregiver. Sometimes the child is brought very late with symptoms of lung infections.

The only treatment is to remove the foreign body if suspected or do a endoscopic examination in a child with high degree of suspicion.

Medications are not necessary before removal; surgical therapy for an airway foreign body involves endoscopic removal, usually with a rigid bronchoscope. It's a highly specialized procedure and should only be done at a centre equipped to handle such cases and should be done by a pediatric surgeon with enough experience in this procedure.

OUR EXPERIENCE

We at Max Healthcare have had a number of referrals for foreign body in children. Since 2009, we have had 3-4 children with foreign body in the airway, referred each year. A 2 year old child was referred to us from Gurgaon in a sick state with complains of sudden coughing while eating almonds. The child was very sick with laboured breathing. On the basis of history given

by the family and urgent x-ray, we took the child for emergency endoscopy. We found pieces of almonds blocking both her main bronchi which were successfully removed. Only the timely referral and emergent bronchoscopy could avoid what could have been a catastroph. The child was started orally within 2 hrs and was sent home the next day.

KEY MESSAGES

No child less than 15 months old should be offered foods such as popcorn, hard lollies, raw carrot or apples. Children under the age of 4 years should not be offered peanuts, almonds and other dry fruits.

Encourage the child to sit quietly while eating and offer food one piece at a time. Avoid toys with small parts for children under the age of 3 years.

Recovery of Dysphagia in Lateral Medullary Stroke

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ABSTRACT

Lateral Medullary stroke is typically associated with increased likelihood of occurrence of dysphagia and exhibits the most severe and persistent form.

Worldwide little research exists on dysphagia in brainstem stroke. An estimated 15% of all patients admitted to stroke rehabilitation units experience a brainstem stroke out of which about 47% suffer from dysphagia. In India, a study showed that 22.3% of posterior circulation stroke patients develop dysphagia. Dearth of literature on dysphagia and its outcome in brainstem stroke particularly lateral medullary stroke motivated the author to present an actual case study of a patient who had dysphagia following a lateral medullary infarct.

This article documents the severity and management approach of dysphagia in brainstem stroke, with traditional dysphagia therapy and Vitalstim therapy. Despite being diagnosed with a severe form of dysphagia followed by late treatment intervention, the patient had complete recovery of the swallowing function.
Key words: Dysphagia, Vitalstim, Stroke, Lateral Medullary syndrome, Deglutition, Deglutition Disorders

INTRODUCTION

Swallowing mechanism is a sequential event of oral, pharyngeal and esophageal phases that transports saliva, ingested solids and fluid from mouth to the stomach and protects the airways during swallowing. Pharyngeal function involves numerous interacting control mechanisms that ultimately link pharyngeal contraction patterns to the adjacent oral cavity and esophagus.

Dysphagia (difficulty in eating and swallowing) is extremely common following a brainstem stroke. About 15% of all patients admitted to stroke rehabilitation units experience a brainstem stroke out of which about 40%-47% of the patients suffer from dysphagia.2 Dysphagia following brainstem stroke is characteristically associated with a greater likelihood of occurrence, showing signs of most severe form of dysphagia, compared to hemispheric strokes.1 Worldwide little research exists on dysphagia in brainstem stroke.

Dysphagia has been associated with respiratory complications, increased risk of aspiration pneumonia, nutritional compromise, and dehydration. It is also socially penalizing and affects the patient's quality of life.

A brainstem stroke affects the swallowing function as the major swallowing centers of the nucleus tractus solitarius (NTS), nucleus ambiguus (NA) and the reticular formation are situated in the dorsolateral medulla oblongata.16 As a result; dysphagia following a lateral medullary stroke (LMS) is often more severe and spontaneous recovery may not completely restore the swallowing function. It may persist for life time or may take months or years to resolve.1 Factors such as lesion size and actual stroke location, as detected and correlated with brainstem MRI, can play a significant role in determining dysphagia morbidity. However, MRI results can demonstrate only the area of the infarct and not the swallowing-related structures and, therefore, cannot clarify the variation of swallowing disorders among patients with LMS.16

Treatment for dysphagia involves traditional therapy8, 20 (diet modification, exercises to strengthen the oropharyngeal musculature, compensatory maneuvers to facilitate laryngeal elevation and closure during swallowing, and techniques to stimulate and strengthen the swallow reflex) and advanced Vitalstim therapy.

Vitalstim therapy is a special form of neuromuscular electrical stimulation (NMES) device, which has received FDA clearance to be used for treatment of pharyngeal dysphagia. It can only be administered under the direction of certified healthcare professionals (speech language pathologists and occupational therapists) 7. On direct electrical stimulation, contraction of swallowing muscles occurs, which is due to the depolarization of peripheral motor nerve after acetylcholine is released at the end plates.

Vitalstim is a dual-channel electrotherapy system, which uses small calibrated electrical current delivered by specially designed electrodes to stimulate the muscles responsible for swallowing. At the same time, trained specialists help patients "re-educate" their muscles through rehabilitation therapy.
Electrodes are simultaneously activated over the submental and laryngeal regions on the throat with the aim of producing a simultaneous

contraction of the mylohyoid muscle in the submental region (to elevate the hyoid bone) and the thyrohyoid muscle in the neck (to elevate larynx to the hyoid bone).

In Vitalstim therapy, electrode placement varies if there is:

- Insufficient Length of the neck
- Fresh surgical incision (no direct placement)
- Presence of indwelling foreign materials like tracheostomy, staples, sutures etc (avoid placement over them)

Vitalstim therapy is contraindicated if patient is not conscious, uncooperative, has behavioral issues or has any implants like pacemaker etc.7

Vitalstim therapy has been used to re-educate patients to utilize their pharyngeal muscles in the throat for patterned activity to commence or re-establish swallowing.8 It has been theorized that this small electrical stimulation may aid swallowing either by augmenting hyolaryngeal elevation or by escalating sensory input to the central nervous system. The current stimulates motor nerves in the throat instigating the muscles responsible for swallowing to contract. The quality of the swallowing function improves and with repetition, muscles may be re-educated.

This case study aims to document the severity and the advanced technological management of dysphagia in lateral medullary infarct.

CASE STUDY

The patient, a 29 year male, came to the Department of Physiotherapy and Rehabilitation, Max Super Speciality Hospital, Saket, New Delhi with the complaint of inability to swallow. He had to spit out saliva frequently as he was unable to swallow it. The patient had a history of right medullary stroke (infarction) 1 year back. He was treated in a local hospital in Punjab. During the course of his hospitalization, he acquired severe pneumonia due to aspiration while he was feeding orally. Therefore, a nasogastric tube (NGT) was placed for nutritional support. The length of stay in the hospital was two months where all of his functions recovered except swallowing. The patient was discharged with the NGT. He was sent to a neurologist for examination. Clinical dysphagia evaluation20 was done by a dysphagia therapist, which included a detailed history of the subjective complaints and medical status, cranial nerve testing, and an examination of the phases of swallowing. Cervical auscultation and respiratory status examination were also done.18 Food trial was not done as the patient could not swallow. It was observed that the patient could not swallow his saliva.

The dysphagia assessment revealed that the patient had good oral motor functions, was able to chew and propel bolus from the mouth but was unable to swallow. The impairment was found in the pharyngeal phase. No hyolaryngeal movement was elicited in the attempt to swallow voluntarily or on stimulating swallow reflex indicating a complete loss of pharyngeal swallowing phase. This can be explained as when mediated by the swallowing center in the medulla, as any stimulus reaches the pharynx, it causes the food to be pushed further back into pharynx and esophagus by rhythmic but involuntary contractions of several muscles in the back of the mouth, pharynx and esophagus. Laryngeal elevation is essential for airway protection during the pharyngeal phase of swallowing. It aids opposition of the arytenoids to the base of epiglottis for laryngeal vestibule closure and helps shape the aryepiglottic sinuses to divert the bolus laterally around the vestibule. Anterior movement of the hyoid in conjunction with laryngeal elevation helps to pull open the flexed upper esophageal sphincter so that food or liquid may be propelled through it and into the esophagus.7

During phonation, soft palate movement was reduced but protective reflexes like gag, cough, and the ability to clear throat were good. Among the lower cranial nerves, glossopharyngeal (IX nerve) and vagus (X nerve) were found to be relatively more impaired. Disuse atrophy made his chance of recovery even worse as it is believed that post 72 hours of stroke, disuse atrophy starts if any particular muscle does not work.4, 5, and 6 In this case his pharyngeal muscles were not in use for more than a year.

American Speech-language-Hearing Association (ASHA) National Outcome Measurement System (NOMS) swallowing level scale was used as a parameter for evaluating the swallowing function on which the patient was rated level 1. The ASHA level is a measurement of both the level of supervision required and the diet level that intuitively reflects a patient's functional status.13 A FEES (Fibreoptic Endoscopy evaluation of swallowing) was not done.

COURSE OF TREATMENT PLANNING & RECOVERY

After detailed assessment and counseling of the patient, a treatment program of traditional therapy combined with Vitalstim therapy was planned in the Outpatient Department of Physiotherapy and Rehabilitation to be followed five days a week. Studies have shown the effectiveness of combined traditional therapy and Vitalstim therapy in stroke patients.[7, 21] The treatment was started and for convenience, the management was divided into two phases.

PHASE ONE

The initial goal was to elicit swallow reflex to initiate hyolaryngeal movement. Documented laryngeal elevation in normal adults is approximately 0-2.50cm or 20mm.11, 12 Therapy commenced after familiarizing the patient with the treatment protocol and Vitalstim device.

The sensory threshold of the patient was identified as the lowest current level at which he reported a 'tingling sensation' on the skin. Following this, the current intensity was raised to the level reported as 'grabbing sensation' by the patient, which in our case ranged between 12 and 14 mA.

Each Vitalstim therapy session lasted for 60 minutes and five sessions were taken per week. In conjunction with Vitalstim therapy, traditional therapy was provided which involved sensory stimulatory activities to elicit swallow reflex with the use of lemon drops, salt, honey, and ice under the supervision of the dysphagia therapist. Sensory input from the gut has a major influence on the activity of brainstem swallowing centers, cortical sensory and motor areas.14

A flicker in the hyolaryngeal excursion on stimulating swallowing reflex was noticed on clinical palpations after completion of 34 sessions of Vitalstim therapy. Subjectively the patient was sensitive to voluntary swallowing attempts, though after every attempt, it was seen that there was clinical laryngeal penetration signs like coughing, wet and gurgling voice which required throat clearing followed by spitting of saliva. Pharyngeal strengthening exercises like Shaker exercise, Mendelssohn's maneuver, Effortful swallow, and Masako exercise were also incorporated.15, 20

PHASE TWO

After 12 weeks, approximately more than 50% of hyolaryngeal excursion, not functional (MMT) 12, was observed on clinical palpation. Food trials were introduced along with Vitalstim therapy. Among the eight types of consistencies of food groups20, initially group 4 (pudding thick consistency) foods like ice-cream, curd, and jelly were given as they have minimal risk of aspiration. Patients with neurogenic dysphagia experience more difficulty with fluids than with thicker or solid consistencies.10

The patient experienced weakness of pharyngeal peristalsis while swallowing, resulting in food residue in the pharyngeal pockets followed by throat clearing and coughing. The patient had coughs during food trials, but was gradually able to swallow the food bolus partially along with the saliva by attempting to swallow repeatedly

after throat clearing instead of spitting out. The quantity and frequency of spitting reduced significantly. Chewable food was introduced to reinforce the physiological oropharyngeal coordination of swallowing. The patient was advised a home regime of pharyngeal exercises and practice of swallowing with recommended safe consistencies of food, like group 4(pudding thick) and 5(mechanical soft chewable), three to four times a day.

After 17 weeks and 76 sessions of Vitalstim therapy, hyolaryngeal excursion became approximately 80%, which was functionally weak (MMT) 12, and the patient was able to swallow foods of group 3(honey thick liquids), 4(pudding thick), 5(mechanical soft chewable), and 6(chewy food) slowly and comfortably without any signs of laryngeal penetration. On completion of 79 sessions of treatment in a period of 18weeks, the patient attained fully functional (MMT) 12 hyolaryngeal excursion and started swallowing foods of group 1(thin liquids), 2(nectar thick liquids), 7(food that fall apart) and 8(mixed textures) without any clinical signs and symptoms of laryngeal penetration and aspiration.

It was noted that the patient was able to swallow all consistencies (group 1 to 8) in a safe manner and rated level 7 on ASHA scale. He was suggested to continue Shaker and Mendelssohn's maneuver for pharyngeal strengthening as well as follow the general aspiration precautions. The NGT was subsequently removed. The patient had a follow up in the Department of Physiotherapy and Rehabilitation after 6 months. Re-assessment showed no deficit in swallowing function.

DISCUSSION

Evidence of combined traditional therapy and Vitalstim therapy to treat dysphagia due to stroke is documented.7, 21 numerous researches exist on dysphagia management in patients with cerebral stroke but less work has been reported worldwide on the management of dysphagia in brainstem stroke, esp. in cases of lateral medullary infarct. One of the case studies on dysphagia following lateral medullary infarct reported resolution of dysphagia 18 months post stroke.1 The peculiarity of this case was that the patient did not receive any kind of attention to swallowing or intervention until after an year. Also, it signifies that the chances of spontaneous recovery of dysphagia followed by lateral medullary infarct are lesser compared to dysphagia following a hemispheric stroke.2
After almost 16 months of dysphagia, the patient started intensive swallow rehabilitation therapy under the guidance of a Dysphagia therapist at Max Healthcare, Saket, New Delhi, India and, in only four months, regardless of severe dysphagia and late intervention, the patient recovered and achieved complete ability to swallow, eat and drink by mouth.

Technologic advances like Vitalstim therapy, Transcranial Magnetic Stimulation (TMS), Functional Magnetic Stimulation (FMS) have enhanced the assessment and treatment of patients with dysphagia by permitting better quantification of impairment and treatment effectiveness.[17]

Dysphagia due to brainstem stroke can be managed efficiently and early intervention may help in reducing recovery period and dysphagia related complications. An important aspect in patient care is improving the recognition and management of dysphagia, or the difficulty or inability to swallow.

Fiberoptic Endoscopic Evaluation of Swallowing (FEES) is a useful tool in the assessment of swallowing. The purpose of the examination is to determine if there is aspiration (food or liquid going into the airway) during or after swallowing, or if the food or liquid remains in the throat after the swallow. It provides information regarding the structure and functions of the pharyngeal phase of swallowing and swallowing safety.19

The patient's refusal for undergoing Fiberoptic Endoscopic Evaluation of Swallowing (FEES) has been the limitation of this study.

Future study is needed in a larger population of patients with lateral medullary stroke to understand the recovery pattern and effectiveness of combined traditional therapy and Vitalstim therapy for improving swallowing function.

CONFLICTS OF INTEREST

There is no conflict of interest in this study.

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Funny Bone

Funny Definitions

Respiration - When you breathe, you inspire.
When you do not breathe, you expire!

Cardiovascular - The three kinds of blood vessels are arteries, veins and caterpillars

Gastrointestinal - The alimentary canal is located in the northern part of Alabama

Dentistry - A permanent set of teeth consists of eight canines, eight cuspids, two molars, and eight cuspidors

Haematology - Before giving a blood transfusion, find out if the blood is affirmative or negative

Humour in Scrubs

- A doctor and a nurse were called to the scene of an accident.
  Doctor: We need to get these people to a hospital now!
  Nurse: What is it?
  Doctor: It's a big building with a lot of doctors, but that's not important now!

- A patient came to his dentist with problems with his teeth.
  Patient: Doctor, I have yellow teeth, what do I do?
  Dentist: Wear a brown tie!

- Patient: Doctor, what does the X-ray of my head show?
  Doctor: Absolutely nothing!

Obesity doesn't run in family.
The main problem is
nobody runs
in family.

*Source - Social Media

WELCOME TO THE TEAM

Dr. Adarsh Koppula
Associate Director - CTVS
Max Super Speciality Hospital, Saket

EDUCATION

- MBBS and MS (General Surgery) from B.J. Medical College, Ahmedabad
- M.Ch (Cardiovascular & Thoracic Surgery) from All India Institute of Medical Sciences

EXPERIENCE

- More than 30 years of rich experience in the field of Cardiovascular & Thoracic Surgery
- Worked as Consultant & Chief - Cardiovascular & Thoracic Surgeon, Fortis Escorts Heart Insitute, Noida
- Worked as Sr. Consultant & Chief - Cardiovascular & Thoracic Surgeon, Metro Hospital & Heart Institute, Noida
- Worked as Cardiac Surgeon & Divisional Medical Officer, Perambur Railway Hospital, Chennai
- Worked as Sr. Resident – CTVS, All India Institute of Medical Sciences

ACCOMPLISHMENTS / AWARDS

- Harjibhai Prize (B.J. Medical College) for anatomy
- Janardhan Prize (B.J. Medical College) for anatomy
- Honor student of the year (St. Xavier's College)
- National Merit Scholarship
- Listed in Marquis' 'Who is who in the World', 14th edition
- More than 50 publications/presentations in national and international journals and conferences

AREAS OF INTEREST

- Cardiac surgeries - more than 8000 to his credit
- Special emphasis on high-risk Coronary bypass operations, Valvular surgeries & Congenital heart operations
- Expert in quality initiatives in healthcare like QCI/NABH
- Experienced senior manager of streamlined surgical departments

MEMBERSHIPS

- Fellow Member of Indian Association of Cardiovascular & Thoracic Surgery (IACTS)
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