



## **PEDIATRIC SURGERY *Update*\***

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#### **Mucous Fistula Refeeding**

Neonates require enterostomy for a variety of conditions such as congenital atresia, meconium ileus, midgut volvulus, necrotizing enterocolitis, spontaneous bowel perforation and rarely gastroschisis. Substantial surgical resection is associated with a short residual length of bowel, often with a proximal stoma and distal mucous fistula. Stomas located in the jejunum or proximal ileum are classified as high output stomas resulting in production of large ostomy losses, fluid and electrolytes imbalances, metabolic acidosis, impaired absorption of fat, protein and other nutrients. In neonates with jejunal enterostomy and mucous fistula a significant length of bowel may be defunctionalized and not be able to be used for absorption of nutrients and electrolytes. Though they anatomically have near normal length of bowel, the higher the enterostomy, the higher the complications associated with a functional small bowel syndrome. These infants rely on total parenteral nutrition (TPN) for growth and development. Mucous fistula refeeding (MFR) is the practice of collecting proximal ostomy effluent and reinfusing it into the distal mucous fistula. Refeeding the distal defunctionalized small bowel through the mucous fistula using the proximal succus entericus secretions can reduce the complications associated with a short bowel syndrome. The clinical benefits of MFR include simplified control of fluids and electrolytes balance in patients with high stoma output, optimal utilization of the remaining absorptive capacity for enteral nutrition, and reduction of gastrointestinal proximal stoma secretions up to 30%. MFR can be used with and without TPN preventing the atrophy of the distal bowel while preparing it for reanastomosis. Refeeding the proximal stoma effluent through the distal mucous fistula uses the absorptive surface of the distal bowel for nutrient absorption, stimulates mucosal growth and intestinal adaptation and prevents atrophy of the villi of the defunctionalized bowel. The increase absorptive function from the added length of intestine may reduce the requirement for parenteral nutrition, promote better weight gain and help eliminate cholestasis by stimulating the enterohepatic circulation. The aim of the MFR technique in infants who have undergone bowel resection is to prime the bowel with luminal feeding promoting intestinal adaptation such as cell hyperplasia, bowel hypertrophy, lengthening and heightening of villi, improved peristalsis and mucosal growth. Strong intestinal growth stimulants including peptides and nutrient substances present in high concentration in the proximal enterostomy effluent induce substantial bowel lengthening and hypertrophy. Disuse atrophy of distal loop can be prevented. A further advantage of the MFR technique is simplification of the control of fluids and electrolytes balance in neonates with a high stoma that has a large output. Indications for refeeding of stoma effluent into the mucous fistula include the presence of a proximal stoma, a high output enterostomy, electrolytes disturbance or failure to achieve adequate weight gain.

Prior to initiation of MFR, patency of the distal bowel is ensured by means of a contrast fluoroscopy study through the mucous fistula. Infuse rate should not exceed 6-10 ml/hr with output refeeding performed every 3 hours to avoid bacterial overgrowth of the effluent to be used. Enteral refeeding technique is safe, reduce hospital stay, improves weight gain and potentially reduces TPN use and related complications in infants with small bowel syndrome and high output enterostomies. Complications associated with MFR include bowel perforation with the use of the catheter, bleeding, bacterial overgrowth if there is a delay between collection and refeeding of the stoma effluent.

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## **Atypical Mycobacterias**

Atypical mycobacteria infection refers to disease produce by Nontuberculous mycobacteria (NTM). They are environmental acid-fast organisms isolated from soil, water, milk, eggs, vegetables and animals transmitted to humans through the respiratory system. More than 130 species have been identified, many of which cause human disease. In children, infection with NTM can result in cervical lymphadenitis, skin and osteoarticular infections, lung disease (predominantly in children with chronic lung disease) and disseminated disease infection in immune-compromised children. Mycobacterium Avium-Intracellulare complex (MAC) is usually the most frequently NTM isolated in children. Cystic fibrosis and Mendelian susceptibility to mycobacterial disease are two distinct inborn genetic disorders associated with NTM disease. Of the acquired disorders associated with NTM, HIV infection predominates. The most common NTM-associated disease in healthy children is chronic cervicofacial lymphadenitis most frequently caused by mycobacterium avium/Intracellulare complex. The oropharyngeal mucosa is the typical portal of entry as toddlers place contaminated objects in their mouths. NTM lymphadenitis occur early in childhood with 80% in children younger than five years, with a mean age of diagnosis of 2.5 years. Children present with history of unilateral lymph node swelling usually affecting the jugulodigastric, parotid or preauricular, submandibular and posterior triangle lymph node persisting for weeks to months despite antibiotic therapy. The infection is not associated

with systemic symptoms or signs. Involvement of submandibular lymph nodes represents the most frequent localization (80%). The affected lymph node can go from a painless firm mass with increased vascularity, to a more fluctuant mass due to liquefaction. Next the skin over the lymph node takes a violaceous discoloration which might lead to fistulization that may discharge for months. Spontaneous healing usually occurs within six months. Pulmonary NTM disease is indistinguishable from pulmonary tuberculosis and is usually associated with cystic fibrosis. Diagnosis of NTM disease requires clinical, radiological and microbiological assessment. A tuberculin (PPD) induration greater than 5 mm at 48 hours suggest a diagnosis of NTM infection. Microbiological diagnosis of NTM disease is achieved by detection of the causative organisms by PCR (more sensitive; more rapid), or bacterial culture (slow growth). Molecular detection of NTM in lymph node biopsy samples is more sensitive than bacterial culture. Histopathology reveals necrotizing granulomatous inflammation associated with caseous necrotic areas. Interferon gamma release assay (IGRA) is positive in 70-80% of tuberculosis lymphangitis cases and generally negative in NTM. The characteristic radiological feature of NTM infection is the presence of central cavitating lesions represented by low-density necrotic material. Management of NTM disease relies on combination of several antibiotics, with macrolide being the cornerstone of treatment. Treatment of NTM adenitis depends on disease stage and severity. Lack of response to three months of antibiotic therapy is considered a treatment failure. Surgery remains an option for lesions that show evidence of progression to cutaneous involvement. Complete surgical excision of the affected lymph node, as soon as possible, is regarded as the best curative option. Secondary wound infection and permanent injury to the facial nerve is a major concern with surgical excision of affected lymph nodes. In cases of incomplete excision of the infected lymph node a macrolide-containing drug regimen should be given. Fluctuant lesions are managed more frequently with antibiotics, while a firm lesion can be observed for spontaneous resolution.

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#### Accessory Cardiac Bronchus

Accessory cardiac bronchus (ACB) is a very rare, poorly recognized, usually asymptomatic congenital anomaly of the tracheo-bronchial tree. ACB is a supernumerary bronchus usually arising from the inner wall of the right main or intermediate bronchus, opposite to the origin of the right upper lobe bronchus. Most cases are incidental findings in asymptomatic adult patients. ACB is a true bronchus, with normal epithelial lining and cartilage walls. The ACB is thought to be a remnant of the cardiac bronchial bud that failed to regress during embryogenesis. Three anatomic variations of ACB have been described: a short, blind ending type, an accessory lobed type which branches into rudimentary ventilated lobules, and a long diverticular type lacking any further arborization. The configuration may range from a short diverticulum where no lung tissue is observed and it appears as a stump, to a longer structure where surrounding lung tissue is present. 70% of ACB are of the diverticulum type ending blindly. Usually, ACB arises from the medial wall of the bronchus intermedius (75%), has a mean diameter of 8.7 mm and a mean length of 12 mm. It is lined by a normal bronchial mucosa, has cartilage within its wall and is usually demarcated by a spur at its origin from the normal bronchus. Though most cases are asymptomatic, ACB may be a site of chronic inflammation and produce several complications including recurrent secondary lung infection, hemoptysis, chronic cough, and rarely malignant transformation. Diagnosis is established with chest CT-Scan. Bronchoscopy might miss the accessory bronchus due to constriction by repeated inflammation. The recognition of an ACB is important since it should be differentiated from acquired bronchial fistula, diverticulum, or adenoid recess. Surgical excision of ACB is recommended when symptomatic, or in asymptomatic patients with the lobed or long diverticular type because of the high probability of long-term complications. This can be accomplished using either minimal invasive thoracoscopy or open thoracotomy.

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**\*Edited by: Humberto Lugo-Vicente, MD, FACS, FAAP**

**Professor of Pediatric Surgery, UPR - School of Medicine, UCC School of Medicine & Ponce School of Medicine.**

**Director - Pediatric Surgery, San Jorge Children's & Woman Hospital.**

**Postal Address: P.O. Box 10426, San Juan, Puerto Rico USA 00922-0426.**

**Tel (787) 340-1868 E-mail: [peditricurgerypr@gmail.net](mailto:peditricurgerypr@gmail.net)**

**Internet: [pedsurgeryupdate.com](http://pedsurgeryupdate.com)**

