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Transanal Endoscopic Microsurgery

Transanal endoscopic microsurgery (TEM) refers to a minimally invasive technique using an operating proctoscope, a laparoscopic camera and modified laparoscopic instruments to accomplish local removal of low level rectal tumors. The technique is ideal to use to remove anorectal benign adenomas, early stage rectal carcinomas, carcinoids of the rectum, for palliative resection of advanced rectal cancers, and surgically correct rectal prolapse and rectal diverticular disease. Recently TEM has been used to resect presacral tumors in adults. In children TEM has been utilized to resect a presacral rectal duplication cyst. The proctoscope is 4 cm in diameter, has four operating ports, maintains an airtight seal at the anus, with a port for CO2 inflow and outflow facilitating smoke evacuation during cauterization. Due to the restricted space to work the operating instruments are angulated to improve the operator range of motion. Most patients who undergo TEM experience short hospital stay, lower rate of postoperative complications and early return to routine activities even after resection of very large lesions with full thickness excision. Anorectal function short of causing decreased anal sphincter pressures and compliance after surgery seldom causes permanent damage such as fecal incontinence. Compared with preoperative levels there is no significant change in anal squeeze pressure after surgery. Tumors larger than 4 cm caused significant decline in rectal sensitivity, urge to defecate threshold and maximum tolerated volume. TEM excision of adenomas results in low adenoma recurrence rates due to higher negative margin rates. Major complications though low include perforation, bleeding and fistulas. The need for diversion before the procedure has been questioned since the oncologic or infectious morbidity associated with opening the peritoneal cavity is low. The use of TEM in managing more advanced rectal must be tested within formal clinical trials before adopting as routine practice.

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Fitz-Hugh-Curtis Syndrome

Fitz-Hugh-Curtis syndrome (FHCS) is a rare complication of pelvic inflammatory disease. It results from ascending pelvic infection causing liver capsule inflammation and right upper quadrant pain mostly in reproductive-aged female with sexual activity characterized by violin-string like perihepatic adhesions between the liver capsule and diaphragm or anterior peritoneal surface. The incidence ranges from 4% to 27% in women with pelvic inflammatory disease. FHCS can be seen in 4% of adolescent females with pelvic inflammatory disease. Predominant symptoms are pain in the right upper quadrant, tenderness, fever and pleuritic right-sided pain. Symptoms can be confused with biliary disease. On physical exam patients can be positive for cervical motion tenderness and adnexal tenderness. Neisseria gonorrhoeae and Chlamydia trachomatis are the primary causative agents with this last one as a more common causative organism. In women, the inflammation of the liver capsule has been attributed to the direct bacterial spread from an infected fallopian tube via the right paracolic gutter. In men, hematogenous and lymphatic spread to liver has been postulated as the underlying mechanism of spread. CT scan may show subcapsular fluid collection, thickening of hepatic capsule in the arterial phase, and wedging enhancement of the involved liver parenchyma in more than 50% of patients. Definitive diagnosis needs invasive procedure like laparoscopy or laparotomy. Most cases of FHCS are managed with antibiotics against the principal offending bacteria. If symptoms persist then surgical lysis of adhesions should be considered. Laparoscopy has both diagnostic and therapeutic benefits.

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Spontaneous Pneumomediastinum

Pneumomediastinum can have diverse etiology in the pediatric population such as chronic lung disease (asthma), rapid scuba diver ascent, illicit drug use, foreign body ingestion and blast injury. There is a significant group of children with spontaneous pneumomediastinum (SPM) and no apparent etiology. The SPM develops after alveolar rupture following an acute rise in intrathoracic pressure. Air escapes from the alveoli to the interstitium and then tracks along the tracheobronchial tree after a pressure gradient between the lung periphery and mediastinum. Clinically the child develops chest pain

radiating to neck or back, shortness of breath, hoarseness, coughing or subcutaneous emphysema. Asthmatics have a higher incidence of spontaneous pneumomediastinum caused by inspiratory effort against a blocked airway (Mueller Maneuver). Weight lifting and sport related are some other instigating factor. Other times inhalation of illicit drugs is a predisposing factor. In such situation the workup imposed in patients with SPM has included CT-Scan, esophagogram, bronchoscopy, serial chest films, etc., when most cases resolve spontaneously without significant intervention. Most of this cases can be initially managed with a short period of observation in the emergency department followed by rest under the care at home of a responsible guardian. Invasive studies or esophagography should be performed on an individual and not systematic basis such as history of emesis, dysphagia/odynophagia, radiographic findings of pleural effusion or atelectasis, etc. The hospital course is usually benign in cases of SPM and most patients require only supportive measures. The risk of recurrence is very low except in cases with chronic lung disease.

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