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Slipping Rib Syndrome

Slipping rib syndrome (SRS) is a rare cause of lower rib and abdominal pain in children and adults. SRS accounts for approximately 5% of all musculoskeletal chest pain in primary care. SRS is caused by a hypermobility of the anterior false ribs that allows the 8th to 10th ribs to slip or click as the cartilaginous rib tip abuts under the rib above. The costal cartilage slips out of its normal anatomical position, the anterior false ribs (8th through 10th) slide out of orientation and become pinned underneath their adjacent superior ribs. This displacement is caused by a congenital anomaly, damage to the fibrinous articulation, or hypermobility of unknown origin. Impingement of the intercostal nerve along the adjacent surface of the adjacent ribs during this slip occurs causing acute pain. Failure to diagnose this condition might lead to unnecessary tests in the child. The pain is caused when the lower costal cartilages 8th to 10th which are not connected to the sternum lose their fibrous or cartilaginous attachments to each other. Pain can extend to the floating ribs (11th and 12th). Movements in the child such as twisting, simple lumbar flexion, bending, deep breathing, laughing, sitting, sneezing, or coughing results in irritation of the intercostal nerve hence pain. Pain is sharp, intermittent, stabbing lasting for a few minutes followed by a dull or burning sensation for several hours. Sometimes associated with nausea and vomiting. Slippage may produce a clicking or popping sensation. Pain can range anywhere from the midline to the lateral flank and from the xiphoid process to as inferiorly as the umbilical line. Due to interconnections between intercostal and somatic visceral nerves the pain might be interpreted as upper abdominal (subcostal). The lack of significant radiographic findings makes the diagnosis of SRS difficult using imaging. Time to diagnosis is often years. Differential diagnosis includes rib fracture, chondritis and pleuritic pain. There is usually one dominant affected side, though the syndrome can occur bilaterally. SRS can occur in any age group and females athletes (swimmers) are more commonly affected than males. Many of these patients have hypermobile joints with laxity and subluxation without symptoms. Diagnosis is established through history and physical examination as imaging are not very useful. Dynamic US can be of help in the diagnosis by demonstrating an overlapping movement of the lower rib above the upper rib. Less thickness of the ipsilateral rectus abdominis muscle has also been found. Pain is intermittent and localized to the lower ribcage with some trigger point of tenderness palpable. Popping or grinding sensation with movement can be elicited. At physical exam the hook maneuver, the examiner slides his finger under the costal margin and lift anteriorly and superiorly, reproducing a click and pain and diagnosing the condition. Careful palpation can identify the disconnected cartilage, or the cartilage curling beneath the overlying ribcage causing point tenderness. Compressing on the sides of the ribs simultaneously may reproduce the pain at the affected

costal margin. As a temporary and localizing measure, a local anesthetic rib block with Bupivacaine can be performed which provides temporary or complete symptoms relief. Repeated anesthetics and steroid rib block, manipulative techniques, acupuncture, Botox injections, prolotherapy, and topical anesthetics produce long-lasting results. Should conservative measures fail, the management should consist of removing the slipping, disconnected cartilages through a small incision at the affected lower subcostal margin. The hypermobile cartilage is removed all the way to the costochondral junction in most cases including the perichondrium to avoid regrowth of the cartilage. During this maneuver, the intercostal neurovascular bundle is preserved. Injury to the neurovascular bundle can cause acute blood loss or chronic neuropathic pain. The use of a vertical bioabsorbable plate to stabilize the hyperflexible subluxing bony ribs used to stabilize fractures is also highly recommended to be incorporated with resection of the cartilages as recurrence rate are decreased significantly. If the patient develops recurrent symptoms after surgery, this might be due to missed slipping cartilages during the initial procedure, regrowth of the cartilage, or new symptoms on the contralateral side. Most patients are satisfied after surgical resection of the slipping ribs.

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Encapsulating Peritoneal Sclerosis

Peritoneal dialysis is the preferred chronic dialysis modality in the pediatric age. Encapsulating peritoneal sclerosis (EPS) is a rare but most serious complications of continuous ambulatory peritoneal dialysis (CAPD) in end-stage renal patients. The incidence of EPS in children receiving CAPD is 2-15% depending on the long-term use of CAPD, while the mortality exceeds 30% with most survivors requiring long-term parenteral nutrition. EPS is characterized by progressive fibrosis of the peritoneum resulting in reduced ultrafiltration capacity, dysfunctional peristalsis of the bowel, and partial or complete bowel luminal obstruction. EPS involves both the visceral and parietal peritoneum. EPS is not specific to CAPD children and can be seen secondary to drug therapy (Beta-blockers), sarcoidosis, systemic lupus erythematosus, abdominal tuberculosis, gastrointestinal malignancies, protein S deficiency and ovarian luteinized thecomas. EPS is a clinical syndrome that continuously, intermittently, or repeatedly presents with symptoms of intestinal obstruction due to adhesions of diffusely thickened peritoneum. The diagnosis of EPS is based on clinical, histologic, and radiographic

findings. The pathophysiological event in the development of EPS is an inflammatory process resulting in loss of the mesothelial layer of the peritoneum and fibroconnective tissue proliferation. Fibrin deposition and fibrinolysis with hyalinization of the superficial stromal collagen possibly tanned through nonenzymatic glycosylation by the dialysate glucose plays an important role in causing excessive fibrogenesis in children with CAPD. Contributing events include duration of CAPD (single most significant risk factor), recurrent episodes of bacterial or fungal peritonitis, the acetate dialysis solution, chlorhexidine, plasticizers, and cumulative exposure to hypertonic glucose-based dialysis solutions. The rate of developing EPS increases with the years receiving CAPD. If a child receives CAPD for more than five years and shows poor ultrafiltration with peritoneal calcifications on CT-Scan, a peritoneal biopsy should be performed to rule out the presence of severe EPS. Peritoneal biopsies of EPS show loss of normal mesothelial cells, massive expansion of the submesothelial compact zone and increased vascularization, mononuclear cell infiltration, calcifications, and low peritoneal mast cell number. EPS occurs insidiously with vague presenting symptoms initially. Symptoms of EPS include weight loss, malnutrition, low-grade fever, nausea, vomiting, hemorrhagic effluent, ultrafiltration failure, ascites, and recurrent bouts of abdominal pain to subacute or acute intestinal obstruction with bowel necrosis. Imaging might show dilated small bowel loops, air-fluid levels, and calcific plaques. Ultrasound is the most sensitive modality to detect EPS demonstrating thickened bowel wall with a trilaminar appearance and adhesion of bowel loops to the anterior abdominal wall. CT-Scan demonstrates peritoneal thickened, bowel teetering, thickened bowel wall, loculated ascites, peritoneal calcification, and clouding of mesenteric fat. The cocoon appearance is secondary to the presence of a thick fibrous layer encapsulating the small bowel. Some recommendations to stop peritoneal dialysis in cases with EPS include ultrafiltration failure, bloody deacylate with calcifications of the peritoneum, duration longer than eight years of dialysis, persistency elevated C-reactive protein and recurrent peritonitis. EPS can occur up to five years after withdrawal of CAPD, when on hemodialysis, or after transplanted. These cases are all characterized by an acute presentation with a rapid clinical course. Management of EPS includes termination of peritoneal dialysis, immunosuppression, steroids (anti-inflammatory), tamoxifen (antifibrotic agent), and surgical debridement. Surgical therapy is required when the child does not respond to medical therapy or presents with complete bowel obstruction, bowel perforation or hemoperitoneum. Total enterolysis and Noble plication are the methods suggested in the literature. Preventing EPS includes minimizing dialysate glucose exposure, preventing acute peritoneal dialysis peritonitis and using a neutral-pH solution.

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Burnout Syndrome

Burnout syndrome is a psychological syndrome arising from the continued response to chronic interpersonal stressors while at work. Defined as a state of physical and mental exhaustion related to caregiving activities or work is a severe problem affecting medical personal and healthcare organizations. Emotional exhaustion and irritability in the work environment leads to development of psychiatric problems characterized by emotional exhaustion, depersonalization, and diminished personal accomplishment. The three main components of burnout are overwhelming exhaustion, feeling of cynicism or depersonalization, and a sense of ineffectiveness and lower efficacy. Burnout is the most common mental issue faced by residents and junior attending. The prevalence among residents is over 50% comparable between medical and surgical specialties. Older age and male prevalence, especially married male residents, are associated with higher prevalence. Systematic review studies have revealed that women, young surgeons, single status, increased workload, and conflict with other colleagues is associated with a higher risk of burnout. In either case, men manifested more depersonalization symptoms, whereas women report more emotional exhaustion symptoms. Women are more likely to adopt emotionally based strategies (self-blame, seeking comfort, getting help and advice from others), while men often resort to ego-defense mechanisms like depersonalization which might impact the ability to provide care. The consequences of developing burnout syndrome between physicians and residents in-training include physical illness, increase feelings of hopeless, irritability, impatience poor interpersonal relationships, risk of medical errors, reduced medical service, depression, and adverse effect in patient safety. In the case of residents, they are subjected to sleep deprivation, high workload, unsatisfactory salaries, and high responsibility. Symptoms of burnout can originate from causes such as: bureaucratic requirements, continued changing work environment, micro-management by administrations, poor clinical supervision, sensationalist media reports of medical errors, limited healthcare resources, litigious environment, and poor work-life balance. Residents and interns in urgency and surgical specialties such as general surgery, anesthesiology, obstetrics & gynecology, and orthopedics have the highest prevalence of burnout syndrome. A plausible explanation is that residents in these specialties deal with emergency routines dealing directly with life-threatening situations with overload shifts. Specialties with less burnout syndrome include otolaryngology, plastic surgery, and neurology. To deal with the problem of burnout in residencies it is necessary to recognize that the problem exists and describe its impact. By addressing burnout, you can increase your personal wellness, improve patient safety, satisfaction, and quality of life. Burnout syndrome needs evaluations of the prevalence and intensity using validated instruments such as the Maslach Burnout inventory and apply these to students, interns, residents and

attending periodically. The Maslach burnout inventory toolkit asses the level of burnout by measuring: 1) emotional exhaustion caused by work, 2) depersonalization, translated into unfeeling and impersonal response toward recipients of our service, care or treatment, and 3) personal accomplishment, measure by the feeling of competence and successful achievement in our work toward patients. In addition, the work environments should be asses by using the Areas of Worklife Survey which measures workload (amount of work to be done), control (opportunity to make choices and decisions to solve problems), reward (recognition, financial and social you receive for contributing into the job), community (quality of the social context in which you work), fairness (extent to which organizations has consistency and equitable rules for everyone) and values (what matters to you in your work). Burnout is prevalent in more than half of US physicians, makes us less happy with our professional choice and less effective in our roles as clinicians, teachers, mentors, and role models. Neglected burnout leads to alcohol and substance misuse, anxiety, depression, discontinuation of residency, fatigue, impaired interpersonal and marital relationship, insomnia and even suicide. Working more than 60-80 hours per week and taking two or more nights on call per week promote burnout. Managing burnout syndrome can be divided into preventive and therapeutic measures. Actions must be concentrated on risk factors, improvement in the relationship between professionals and the promotion of healthy behavior in physicians. Therapeutic strategies consist in management of negative emotions and relaxation technique. To alleviate and prevent burnout we should fix what we control and advocate for the rest. These include aligning with patients providing high-quality care, develop expertise in disease and not technique, redesign the practice, developing progressive employment policies, and respect toward our own choices.

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