for more training and widespread availability of gastrostomy replacement tubes. Replacement gastrostomy tubes are expensive and it is impractical to have every size and make available. In addition, attending staff need to know what size of PEG tube was removed / dislodged in order to replace a similar size and unfortunately this information is not always available. All attending professionals caring for patients in the community following PEG tube insertion have a responsibility to be fully informed and competent.

It is our current practice that all patients being discharged with new PEG tubes are given a replacement gastrostomy tube and instructions as to what to do if the original tube becomes dislodged. This advice includes bringing it with them to Accident and Emergency unit if a hospital visit is required. Nutrition Nurse specialists in our trust send a letter to the District nursing services which also includes the requisition numbers for replacement gastrostomy tubes and the request that these are ordered and available in the patients' homes. Contact details of the Nutrition Nurse specialist are also contained in the documentation.

We would dispute that our comments regarding the ability of District Nurses to replace gastrostomy tubes are inappropriate since this "needs to be performed in a hospital environment". Ideally, trained professionals in the community are suitably situated to replace gastrostomy tubes to avoid unnecessary trips to Accident and Emergency units.

The letters have identified several areas for possible service development. We greatly appreciate the comprehensive service provided in the community by our District Nursing colleagues and others.

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REFERENCE:

1. Lowry S, Johnston SD. *Who Follows Up Patients After PEG Tube Insertion? Ulster Med J* 2007;**76(2):** 88-90.

SYMPTOMATIC HYPERPHOSPHATAEMIA FOLLOWING PHOSPHATE ENEMA IN A HEALTHY ADULT

Editor,

Adequate colonic cleansing is essential for accurate and safe colonic procedures[1]. Common preparations for cleansing include diet in combination with a cathartic agent (stimulants), gut lavage, and phosphate preparations (osmotics). Phosphate preparations offer an attractive alternative due to smaller volumes required for ingestion. We report an unusual case of acute hyperphosphataemia following the administration of a phosphate enema.

Case report: A 79 year lady with a six month history of lower abdominal cramps and diarrhoea including mucous per rectum underwent flexible sigmoidoscopy. She had taken one sachet of picolax (10mg sodium picosulfate) as bowel preparation the night before and reported minimal effect. As such she received a single phosphate enema at 09.30. This contained 30.8g of sodium phosphate in 118ml delivered by a standard rectal tube. She became unwell within 10-15 minutes with severe nausea and dizziness. Observations demonstrated a heart rate of 86 beats per minute and a blood pressure of 80/34mmHg. Bloods were taken for urea and electrolytes and a normal saline infusion was started. Over the subsequent 90 minutes her blood pressure improved to a systolic of 100mmHg and her heart rate fell to 60 beats per minute. Her blood results were normal with the exception of a phosphate of 2.65 mmol/L (0.8 – 1.55). Her symptoms and clinical observations continued to improve and by 11.30 she was able to undergo flexible sigmoidoscopy which was normal. Repeat blood tests two days later were normal (phosphate 1.31mmol/L). At subsequent outpatient review a small bowel series and ultrasound scan of abdomen were normal. Barium enema demonstrated mild sigmoid diverticular disease. Eight months later her gastro-intestinal symptoms had settled.

Discussion: Asymptomatic hyperphosphataemia with levels 2-3 times above normal has been reported in nearly 25% of individuals with normal renal function after administration of oral phosphate-based laxatives[2]. Current recommendations[3] simply suggest caution in the elderly and those with renal impairment. Multiple case reports exist warning of the dangers of oral phosphate-based laxatives in patients with renal disease and in paediatrics and only a handful of accounts of hyperphosphataemia have been reported in patients receiving phosphate-based enemas in similar patient groups[4,5].

The mechanism of hyperphosphataemia in renal impairment is felt to be secondary to decreased excretion of phosphate by the kidneys. In paediatrics it is believed to occur due to large volumes of phosphate containing solution, relative to the child's size. Other recognised causes following oral phosphate based laxatives include Hirschsprung's disease, faecal impaction, or functional intestinal obstruction where increased gastrointestinal phosphate absorption may occur, elderly age because of the diminished intestinal motility, and increased intestinal permeability in the presence of inflammatory intestinal disorders[6].

There are no cases in the literature of hyperphosphataemia arising due to diverticular disease following phosphate-based enema. However one could postulate that, for the reasons mentioned above, it could be an aetiological factor albeit unlikely in this instance due to the absence of significant disease or active inflammation. In summary this case report highlights the need for vigilance even in patients deemed low risk of developing hyperphosphataemia following a phosphate-based enema.

The authors have no conflict of interest.

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REFERENCES:

1. McCoubrey AS. The use of mechanical bowel preparation in elective colorectal surgery. *Ulster Med J* 2007;**76(3):** 127-130.
2. Ainley EJ. Hyperphosphataemia after bowel preparation with oral sodium phosphate. *Endoscopy* 2006;**38(7)**:759.
3. Levin TR, Farraye FA, Schoen RE, Hoff G, Atkin W, Bond JH, *et al.* Quality in the technical performance of screening flexible sigmoidoscopy: recommendations of an international multi-society task group. *Gut* 2005;**54(6)**:807-13.
4. Fine A, Patterson J. Severe hyperphosphatemia following phosphate administration for bowel preparation in patients with renal failure: two cases and a review of the literature. *Am J Kidney Dis* 1997;**29(1)**:103–5.
5. Craig JC, Hodson EM, Martin HC. Phosphate enema poisoning in children. *Med J Aust* 1994;**160(6)**:347-51.
6. Vukasin P, Weston LA, Beart RW. Oral Fleet Phosphor-Soda laxative-induced hyperphosphatemia and hypocalcemic tetany in an adult: report of a case. *Dis Colon Rectum* 1997;**40(4)**: 497–9.

PERIANAL LEIOMYOMA INVOLVING THE ANAL SPHINCTER.

Editor,

Leiomyomas are benign soft tissue tumours of mesenchymal origin and can develop wherever smooth muscle is present. Their pathogenesis remains obscure. Deep soft tissue leiomyomas are rare and are further classified as somatic and retroperitoneal. Whereas the former have a predilection to occur in extremities (usually in the thigh) the latter usually occur in the pelvic retroperitoneum[1]. We report a case of perianal leiomyoma stretching the muscle fibres of the external sphincter. Reports of perianal leiomyomas are sparse in the literature. Features of deep soft tissue leiomyomas, anal leiomyomas and their management are discussed.

Clinical background: A 45-year-old female presented with a history of a painless swelling in the perianal region for 18 months, gradually increasing in size. Clinical examination revealed a 30mm diameter extrasphincteric swelling in the rectovaginal septum. Endoanal ultrasonography showed a soft tissue mass related to the anterior and lateral wall of the anal canal over its entire length. Although the mass appeared to be entirely outside the external sphincter complex there was a suspicion of sphincter involvement anteriorly. The lesion was well defined and homogeneous in texture with an intermediate to low signal intensity on T2 weighed magnetic resonance imaging (Figure 1). Fat saturation (FAT SAT) & Short Tau Inversion Recovery (STIR) sequences suggested that the lesion displaced rather than infiltrated the sphincter. There was loss of visualisation of the lower subcutaneous and superficial components of the external sphincter with a suspicion of extension to the deeper component of the anal sphincter.

An elective excision was performed with a circumanal incision. Sphincter fibres were stretched over the surface of the lesion. Complete extra capsular dissection of the lesion was performed in continuity. Sphincter fibres were divided and repaired with 2'0 PDS.

Macroscopically, the tumour was solid and well circumscribed with a whorled white cut surface without gross cystic

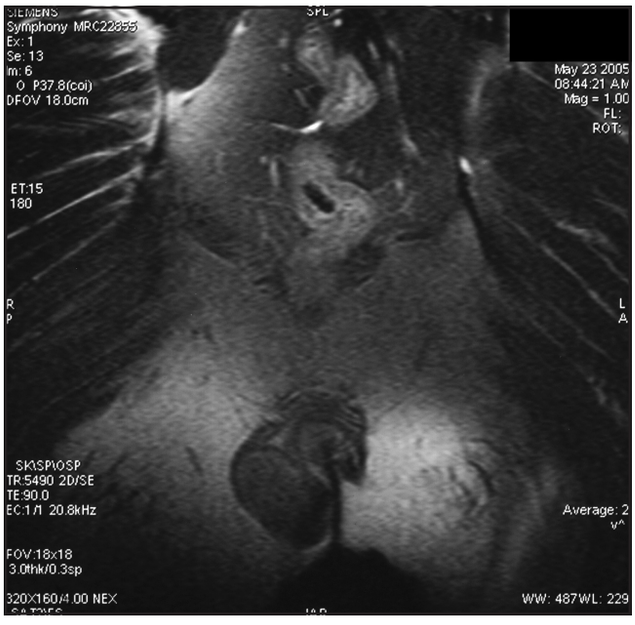

Fig 1. MR sequence with T2 weighting with fat saturation demonstrating an ovoid shaped low signal mass in relation to the right side of anal canal displacing the fibres of external sphincter.

degeneration or necrosis. The tumour measured 65mm in diameter. Histological examination revealed a circumscribed smooth muscle tumour consisting of interlacing fascicles of bland spindle cells admixed with focal areas of myxohyaline stroma. There was no cytological atypia, abnormal mitotic activity or necrosis. Only one or two mitoses were identified in the sections examined. Immunohistochemistry demonstrated strong positivity for smooth muscle markers desmin (Figure 2) and actin. Positivity for estrogen and progesterone receptors was also noted. CD117 was negative. Two months after the surgery, the patient has no incontinence with good sphincter tone.

Discussion: First described by Virchow in 1854, leiomyomas are benign soft tissue tumours that arise from smooth muscle accounting for 3.8% of all benign soft tissue tumours[1]. Kloepfer originally noted a hereditary syndrome characterised by multiple leiomyomas in 1958. Leiomyomata can develop

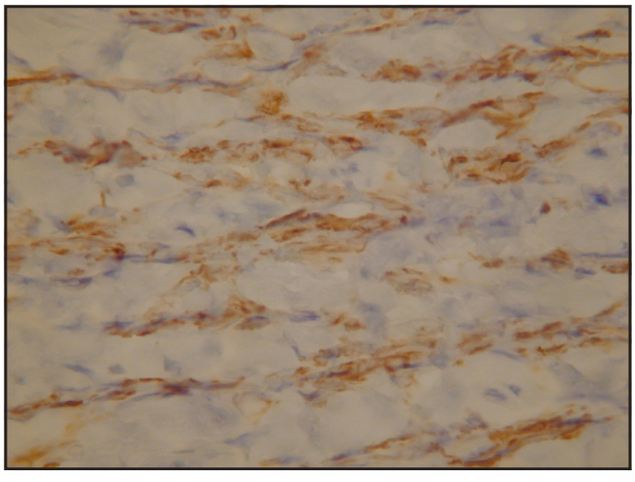

Fig 2. Immunohistochemical staining for Desmin (immunoperoxidase, x 250)