An interesting case of small bowel obstruction

Q1: What is the diagnosis?
This is a case of mechanical small bowel obstruction secondary to an enterolith/bezoar the likely source of which is jejunal diverticulosis.

Q2: What is the differential diagnosis?
This includes the various intraluminal causes of small bowel obstruction such as:
- True foreign bodies: metallic, plastic.
- Food bolus.
- Gallstones.
- Concretions.

Q3: What are the other possible complications of the primary disease of the small bowel?
The possible complications include:
- Diverticulitis
- Haemorrhage
- Obstruction: 1. True obstruction due to enterolith, diverticulitis, adhesions associated with inflammation, volvulus about the adhesions, and intussusception. 2. Pseudo-obstruction or motility disorder.
- Malabsorption due to stasis and bacterial overgrowth.
- Fistulac.
- Asymptomatic pneumoperitoneum.
- Malignant tumours.

Q4: What are the possible treatment options?
The simplest surgical option for small or crushable enteroliths is to milk them distally into the cecum and allow them to pass naturally. If this is not possible, the treatment of this condition is an enterotomy to remove the enterolith with or without resection of the segment of small bowel involved with diverticulosis. Resection may be advocated if the diverticulosis is localised and is recommended for the other forms of obstruction, haemorrhage, and patients with malabsorption who do not respond to conservative management.

Discussion
The findings at laparotomy were as follows: dilated loops of small bowel seen to mid-ileum. Obstruction at this point was due to an enterolith with collapsed distal bowel (see fig 3 in questions p 626) and fig 1 below). Two large jejunal diverticulae 12 and 24 inches from the duodenojejunal flexure seen which were palpably empty. The gallbladder was normal with no palpable gallstones. The enterolith was milked proximally and removed via a longitudinal antimesenteric enterotomy which was closed transversely, without resection of the diverticular segment. At laparotomy it is essential to rule out the other causes of enteroliths such as gallstones (as evidenced by a cholecyst-jejunoduodenal fistula) and to carefully palpate the entire length of the small bowel including the diverticula for further enteroliths.

Bezoars are masses of solidified organic or non-biological material commonly found in the stomach or small bowel. Four types have been described based on their composition: phytobezoars (containing fibre and cellulose), trichobezoar, lactobezoars, and miscellaneous. The last group includes medications (hydroscopic bulk laxatives, cholestyramine, non-absorbable antacids, vitamin C tablets, and isocal tube feeds), parasites (Ascaris lumbricoides or roundworm), and synthetic fibre.1 A case of carpet fibre bezoar forming at the site of a stapled intestinal anastomosis in a child with pica has been described. In general, the formation of bezoars in the small intestine appears to be at sites of stasis such as blind loops, tumours, and diverticulae (duodenal, jejunal, and Meckel’s).2,3

The incidence of acquired jejunal diverticulosis varies from 0.2% to 1.3% on necropsy studies to 2.3% on enteroclysis.4 It is associated in 33% to 75% of cases with diverticula elsewhere in the gastrointestinal tract. Enteroliths that form in the proximal small bowel contain bile salts and are frequently radiolucent whereas as many as a third of those that form in the ileum are radio-opaque because of precipitation of mineral salts in an alkaline environment.5

The diagnosis is therefore rarely made on the preoperative plain abdominal radiograph.Computed tomography is the modality of choice for investigating patients with higher grades of small bowel obstruction where early surgical intervention is contemplated.6 There is an increasing tendency to utilise computed tomography to help define the cause, severity, and complications of small bowel obstruction due to the unreliability of clinical signs to predict accurately those patients requiring early intervention.7

This is an unusual cause of small bowel obstruction that needs prompt diagnosis and operative treatment.

Final diagnosis
Enterolith causing small bowel obstruction.

References

Iatrogenic groin pain

Q1: What is the likely clinical diagnosis?
The triad of groin pain, hip flexion, and femoral neuropathy indicates iliospas sheath haemorrhage. This condition occurs in patients with inherited coagulation disorders, particularly haemophilia A, and in patients on oral anticoagulants.1,2 Spontaneous haemorrhage occurs deep to the iliacus fascia from the iliacus or psoas muscles, blood tracking from the retroperitoneal space into the pelvic extraperitoneal space. Occasionally massive bleeding can lead to signs of volume deficit.

The iliacus fascia invests the psoas major and iliacus muscles and continues inferiorly to the posterior wall of the femoral sheath. This explains the association with femoral neuropathy, the nerve lying in the groove between the iliacus and psoas muscles. The predilection for the iliacus muscle is unclear.

Q2: What lesion is shown on the computed tomograms (see p 627)?
The computed tomograms shows a collection behind the left iliacus muscle which displaces this anteriorly and separates it from the iliac blade. The left iliospas muscle appears enlarged with heterogeneous attenuation internally.

Q3: How should this condition be managed?
In our patient, warfarin was temporarily stopped. He was administered vitamin K, and thereafter started on heparin. The international normalised ratio came down from 7.2 to 2.0 within 24 hours. The pain resolved. There was weak residual non-disabling thigh weakness at the time of discharge.

Discussion
Haemorrhage into the iliacus and or psoas muscle is a well recognised complication of overanticoagulation, as well as in haemophiliac disorders. The precise incidence and initiating mechanism of this condition is unclear. Two anatomical syndromes have been described.

Spontaneous haemorrhage may commence either in the iliacus muscle, in which case bleeding occurs deep to the iliacus fascia and a femoral neuropathy may be seen. Alternatively bleeding may commence in the psoas major muscle initially or spread from the iliacus muscle to the psoas. In this case involvement of other components of the lumbar spine, including the obturator nerve and the lateral femoral cutaneous nerve of the
thigh, is likely. A similar clinical picture may be produced by neoplastic infiltration of the lumbosacral plexus.

Pain is the presenting feature, involving the groin, and radiating to the thigh and leg. This is followed by gradually increasing paraesthesiae and limb weakness. A flexion contracture of the hip occurs. In the present case, the patient has not been on any medication for the disease. The hip extension is restricted and painful. Delayed development of bruxing in the groin may occur. The pain may resolve in a week with slower and often incomplete recovery of neurological function. In 10%–15% of cases there may be no significant improvement.

There is little definitive guidance on management. As the literature is largely anecdotal, based on case reports or small case series. Overanticoagulation needs to be recognised and corrected. Computed tomography or ultrasound guided aspiration may be helpful, especially when a pseudocyst is suspected.

The prognosis must remain guarded, as residual neurological sequelae are possible even where surgical treatment has been undertaken. With the increasing usage of therapeutic anticoagulation, doctors dealing with anticoagulated patients need to be aware of this clinical presentation.

**Final diagnosis**

Iliopsoas sheath haemorrhage.

**Acknowledgement**

We wish to thank Dr David Grant for selecting and commenting on the radiographs.

**References**


**Terminal ileal stricture**

**Q1: What does the small bowel enema show (see p 627)?**

The small bowel enema shows normal jejunum. The ileum is shortened in its distal portion and uniformly narrowed with a smooth outline; the ileocecal junction is well delineated and the caecum is normal.

**Q2: What is the differential diagnosis?**

The differential diagnosis of ileal stricture includes tuberculosis, Crohn’s disease, pelvic inflammation, ischaemia, radiation enteritis, carcinoid infiltration, lymphoma, and diffuse enteritis—that is, disorders where there is inflammation, infiltration, or oedema of the small bowel. History and clinical findings in this case did not contribute to the diagnosis.

**Q3: How can you confirm the diagnosis?**

Enteroscopy/terminal ileoscopy is the investigation of choice. In active Crohn’s disease, the terminal ileum shows patchy asymmetrical and heterogeneous mucosal lesions. Ulcers which may be aphthoid, superficial, or deep are seen surrounded by normal mucosa. Tuberculoid granuloma is the most specific finding on histology apart from infiltration of lamina propria by lymphocytes and plasma cells with agglutates of lymphocytes near the base of the ulcers. In the present case, the smooth and featureless mucosa, and inflammatory cellular infiltrate of lamina propria suggests Crohn’s disease in remission. The patient has not been on any medication for over four years.

**Discussion**

Segmental areas of luminal narrowing of ileum referred to as ileal stricture is due to rigid thickening and fibrosis of its wall resulting in obstruction. It is a common complication of Crohn’s disease, tuberculosis, and intestinal ischaemia. A flare-up of inflammatory process causes temporary intestinal narrowing; when healing occurs with a scar or fibrous tissue formation the obstruction is complete. This may be circumferential and concentric or eccentric and irregular in nature. On barium contrast examination, these strictures typically appear as segmental narrowing without normal mucosal pattern and with smooth tapered ends—referred to as the “string sign”. Strictures themselves are painful and may not require treatment. But sometimes, these areas become so narrow and result in a partial or total obstruction.

What is the pathophysiology of stricture formation? The intestine can propel the luminal contents only when the lumen remains fairly wide enough. When there is damage to the intestinal crypts, the process due to inflammation, the smooth muscle cells of the intestine activate a complex chain of events, involving a host of immune system components, for example IFN-gamma. There is production and deposition of more than normal collagen at the site of injury. Scarring occurs, the layers of intestinal muscle thicken, and the muscles no longer move smoothly and easily. In short, a stricture, either by circumferential narrowing or eccentric or irregular in nature. On barium contrast examination, these strictures typically appear as segmental narrowing without normal mucosal pattern and with smooth tapered ends—referred to as the “string sign”. Strictures themselves are painful and may not require treatment. But sometimes, these areas become so narrow and result in a partial or total obstruction.

An important differential diagnosis of ileal stricture in the present case is Crohn’s disease, based on the peroperative findings of mucosal constrictions and smooth tapered ends. In the series reported, the biopsy is non-specific and simulates tuberculosis. Hypotensive drugs can occasionally produce intestinal ulceration and stricture formation.

The barium infusion technique (enteroclysis) is an ideal investigation for study of the ileal lumen, both for functional obstruction and for extensive mucosal disease. In the best of hands, the procedure gives an optimal radiological-gross pathological correlation and satisfactory evaluation of the extent, depth of the disease, and complications.

A histological difference between Crohn’s disease and tuberculosis is not always possible. Supportive information only helps in making a diagnosis. In the case reported, the peroperative findings of ileal stricture resembled Crohn’s disease and ileal tuberculosis; terminal ileoscopy and histology was not helpful. The possible diagnosis is Crohn’s disease in “remission”.

The patient under study has been asymptomatic for four years and is not on any medication. Intervention in ileal strictures is not necessary when an individual is asymptomatic. Steroids, aminosalicylic acid preparations, immunomodulators, and verapamil have been used during the inflammatory phase; the latter is considered the spasmolytic drug of choice to intestinal injury and prevents collagen deposition. When the affected segment is fibrous and scarred, endoscopic balloon dilatation can relieve the obstruction. Surgical options include stricturoplasty and resection of the affected segment.

**Final diagnosis**

Crohn’s disease in “remission”.

**References**


**Lung nodules in a silver polisher**

**Q1: What do the chest radiograph and HRCT of the lung show (see p 628)?**

The chest radiograph shows nodular opacities, predominantly in the upper lobe. The HRCT lung images show diffusely distributed centrilobular nodules without any evidence of fibrosis.

**Q2: What is the differential diagnosis of the HRCT scan appearance and the likely diagnosis in this case?**

The differential diagnosis of centrilobular nodules with a diffuse distribution is HRCT
Box 1: Differential diagnosis of centrilobular nodules on HRCT lung scan

- Infectious bronchiolitis including tuberculosis.
- Pneumoconiosis (coal worker’s pneumoconiosis and siderosis).
- Diffuse panbronchiolitis.
- Vasculitis and vascular metastases.
- Sarcoidosis.
- Respiratory bronchiolitis-interstitial lung disease.
- Hypersensitivity pneumonitis

Discussion
Siderosis (synonyms: welder’s lung, buffer’s lung, or silver polisher’s lung) is a non-fibrogenic or a “benign” form of pneumoconiosis due to the inhalation of iron particles. Iron dust is an inorganic, inert, mineral dust with high radiodensity, which neither causes substantial proliferation of reticulin fibres nor gives rise to collagenous fibrosis when re- sulting in the lungs. 1 Occupations leading to siderosis involve exposure to iron oxide dust or fumes and include steel and silver polishing, iron and steel rolling, steel grinding, electric arc welding, fettling, stripping and dress- ing castings in iron foundries, boilermaking, and mining iron ores. 2

Patients are usually asymptomatic unless there is concurrent smoking or contamination of air with other chemicals such as silica or asbestos. 3 They may have a reddish coloured sputum due to exposure to these dusts. 4 Siderosis is therefore essentially a “radiolog- ical disorder”, due to the presence of very radiodense opacities, but with no functional impairment. 5 Iron oxide exposure may be carcinogenic for the human lung. 6 7 The emis- sion of polycyclic aromatic hydrocarbons as pyrolysis products of organic materials used may be responsible, but requires further con- firmation.

The pathology of siderosis is characterised by perivascular and peribronchial aggrega- tion of dark pigmented iron oxide particles present extracellularly in alveolar spaces and walls as well as in macrophages. 8 Slight reticulin proliferation may be present in siderosis, but there is no collagenous fibrosis. If fibrosis is present, it is secondary to the presence of crystalline silica.

Radiologically, siderosis presents as centri- lobular opacities on HRCT with a uniform dis- tribution throughout the lung fields with no conglomeration. Centrilobular opacities on HRCT lung can be divided into two types according to size. 9 The larger ones are seen as sharply demarcated, rounded nodules and these correspond to the q and r types of pneu- moconiosis (box 2) seen on the chest radiograph. 9 The smaller ones, more frequent in number, are seen as relatively ill defined nodular or branching opacities, a few closely spaced dots, or areas of low attenuation on HRCT and represent radiographic type p pneumoconiosis. The nodules are present dif- fusely and bilaterally, but with upper lobe and posterior predominance.

The differential diagnosis of centrilobular opacities on HRCT lung includes miliary tuberculosis, metastases, sarcoidosis, hypersensitivity pneumonitis, panbronchiolitis, and respiratory bron- chiolitis-interstitial lung disease. Some radio- graphic features are characteristic of the underlying disorder. In miliary tuberculosis, the size of nodules is relatively uniform throughout the lungs. In haemorrhagic metastases, the metastatic nodules are usually smooth, well defined, and round in shape. They are usually variable in size and do not show ill defined, fine centrilobular nodular or branching opacities. A classical perilymphatic distribution of the nodules is seen in condi- tions like silicosis, sarcoidosis, lymphangitic metastases, and amyloidosis.

Patients with siderosis require no treatment and the radiological changes of siderosis may regress after cessation of exposure. 1 The diag- nosis of siderosis should be considered in rel- evant occupations with characteristic radi- ological abnormalities and absence of respiratory symptoms.

Box 2: International Labor Office classification of radiographs of pneumoconiosis

- Round opacities are classified according to size as: p, q, or r (p, up to 1.5 mm in diameter; q, 1.5 – 3 mm; r, 3 – 10 mm).
- Irregular opacities are classified as: s, t, and u (fine, medium, or coarse respectively).
- Combination of round and irregular as: x, y, and z.

Learning points
- Siderosis, baritosis, and stannosis are types of benign pneumoconiosis with radiographic dense opacities.
- Siderosis, also called welder’s lung, buffer’s lung or silver polisher’s lung is the most common type.
- Usually asymptomatic, respiratory symp- toms may be present in smokers or if there is concurrent exposure to silica or asbestos.
- Centrilobular nodules with diffuse distribu- tion are seen on a HRCT lung scan.
- Diagnosis is made on radiological features and occupational exposure.

References

Spinal tumour with raised intracranial pressure

Q1: Based on history and clinical examination what is the differential diagnosis?
Differential diagnosis for raised intracranial pressure with cauda equina syndrome include entities with lesions at multiple sites such as:
- Malignant meningitis, particularly lymphoma with root lesion.
- Neurofibromatosis.
- Tuberculosis.
- Metastases.

Q2: What are the investigations required?
Imaging of the craniospinal axis and cerebrospinal fluid (CSF) analysis are required for diagnosis. In our case, magnetic resonance imaging of the brain showed no expanding mass lesion, except for a small left parietal arachnoid cyst. CSF analysis was then done by lumbar puncture. The fluid was xanthochro- mic with an opening pressure of 30 cm H2O. CSF protein was 30 g/L, glucose 5.4 mmol/l, white blood cell count 40 × 10⁹/l, and red blood cell count 160 × 10⁹/l. This was suggestive of a CSF block in the spinal canal.

Magnetic resonance imaging of the spine showed a intradural, extramedullary hetero- geneously enhancing mass at the D11–L2 level, which was hypointense on a T1 weighted image and hyperintense on a T2 weighted image (figs 1 and 2).

Q3: What are the causes of bilateral papilloedema without an expanding intracranial mass lesion?
The causes of bilateral papilloedema without an intracranial expanding mass lesion in- clude:

Figure 1

T1 weighted gado enhanced sagittal magnetic resonance image of the thoracolumbar spine showing a well circumscribed 5 × 2 cm mass at D11–L2 level.
Raised CSF protein.  

Diplopia.  

Raised CSF pressure causes increased pressure.  

The chest radiograph suggests that the lesion extends medially to the lateral border of the spinal cord and is causing minimal displacement of the cord. If the tumour were to extend further medially, neurological symptoms resulting from cord compression might arise. Similarly, if the lesion was to further deform the structure of the vertebral body, vertebral collapse might arise.

Outcome  
In view of the proximity of the lesion to the spinal cord, thoracotomy rather than thoracoscopic excision was considered the preferred surgical approach. The patient proceeded to a right posterolateral thoracotomy in the fourth intercostal space, and a soft yellow dumbbell shaped mass measuring 30 \times 30 \text{mm} was discovered in the right third intercostal space, extending into the transverse foramen. The lesion was excised and histological examination showed a partially encapsulated tumour composed of spindle cells in a myxoid background. Antoni type A and B components were present with focal Verocay body formation. The histopathologist concluded that the lesion was a benign intercostal schwannoma. The patient had an uneventful postoperative recovery. Four months after her operation, she remains well, her symptoms have resolved, and there is no radiological evidence of recurrence of the lesion.

Discussion  
Neurogenic mediastinal tumours account for approximately 20% of all mediastinal tumours in adults and 40% in children. These tumours may be of nerve sheath, autonomic ganglia, or paranganglionic origin and are generally located in the posterior mediastinum. Neurogenic tumours of the mediastinum with an

Learning point  
Consider a spinal tumour in a patient with normal cranial imaging and a diagnosis of presumed benign intracranial pressure.

Q4: Based on the radiological appearances, what potential complication might arise if the lesion was left untreated?  

MRI scan (see p 629) demonstrates that the tumour extends medially to the lateral border of the spinal cord and is causing minimal displacement of the cord. If the tumour were to extend further medially, neurological symptoms resulting from cord compression might arise. Similarly, if the lesion was to further deform the structure of the vertebral body, vertebral collapse might arise.

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Venous stasis by the tumour compression of spinal or medullary venous plexuses causing an unfavourable transarachnoid villous hydrostatic pressure, another proposed mechanism.\(^3\) Leptomeningeal inflammation due to the toxic effect of CSF protein causing compromise of CSF absorption is another possible mechanism for raised intracranial pressure.\(^4\)

The spinal canal acts as an elastic reservoir for CSF. With normal changes in physiological cerebral blood flow, the ability of the CSF to flow in and out of the spinal canal is thought to be important in maintenance of a constant intracranial volume. By compromising this system spinal tumours may in fact reduce the capacity of this reservoir and cause papilloedema.\(^5\)

A smoker with an apical mass  

Q1: Describe the abnormalities shown in figs 2 and 3 (see p 629)  
The chest radiograph suggests that the lesion arises in the posterior mediastinum as it is visualised above the clavicle. Figure 2 is a computed tomogram of the thorax that demonstrates a well marginated lesion extending from the neural canal at right T2/T3 level, with significant widening of the neural foramen at this level. There is no evidence of mediastinal lymphadenopathy or lung parenchymal involvement. Figure 3 is a MRI scan that demonstrates a dumbbell shaped neoplasm measuring 40 \times 30 \text{mm} arising from the right T2/T3 nerve root. It is of high signal intensity on T2-weighted images with a heterogeneous area of lower attenuation centrally. The lesion measures 15 mm within the canal and 21 \times 30 \text{mm} within the thorax and is expanding the neural canal at this level, consistent with a neoplasm of neural origin. The medial aspect of the tumour extends to the lateral border of the spinal cord, which is minimally displaced.
intraspinal component connected by a narrowed segment in the intervertebral canal are generally described as dumbbell or hourglass tumours. Tumours of nerve sheath origin classically present as paraspinal masses. These tumours may be neurofibromas, schwannomas, granular cell tumours, melanotic schwannomas, or malignant schwannomas. Schwannomas are most often benign tumours originating from the sheath of peripheral nerves and usually present as solitary and well encapsulated tumours. Most are asymptomatic and are detected incidentally on a routine chest radiograph. Schwannomas arising from the intercostal nerve are well described and may be multiple. Infrathoracic schwannomas may also arise from other nerves, such as the phrenic and glossopharyngeal nerves. Schwannomas are usually benign but may occasionally be malignant. Malignant schwannomas have been described in patients with neurofibromatosis.

Of special concern in the management of neurogenic tumours arising in the thorax is spinal cord compression resulting from either intraspinal lesions or vertebral body destruction and collapse. Predictably, this complication makes for a more difficult surgical excision. Intercostal schwannomas have been successfully resected thoracoscopically. This technique may reduce hospital stay and minimise postoperative complications, but is best reserved for lesions in which there is no evidence of intraspinal extension.

The diagnosis of mediastinal tumours has been aided by recent advances in computed tomography, MRI, ultrasonography, radionuclide scanning, and fine needle aspiration. MRI is the preferred modality for imaging neurogenic tumours, because of its multiplanar capability and high contrast resolution. MRI can best demonstrate the number and nature of the lesions (differentiating cysts from neoplasms) and the intraspinal and crano-caudal extent of the lesion, and thus determine the optimal surgical approach for patients. This technique is also especially useful for evaluation of the mediastinum of patients for whom the administration of iodinated contrast material is contraindicated. In cases where intraspinal extension is demonstrated, open thoracotomy is the more suitable approach, as the likelihood of conversion to open thoracotomy during thorascopic resection is high. However, the definitive diagnosis of schwannoma requires histological examination.

**Final diagnosis**

Benign schwannoma, fourth intercostal space.

**References**


**A man with a chest mass**

**Q1: What is the interpretation of the biopsy specimen (see p 629)?**

The specimen is an excision biopsy of the tumour. Cut section of the mass shows a variated appearance with areas of haemorrhage, cystic changes, and necrosis. The histological findings are those of metastatic adenocarcinoma.

**Q2: What are the different types of rib tumours?**

The bone metastases is either osteoclastic (often squamous cell carcinoma), osteolytic (prostate carcinoma, poorly differentiated adenocarcinoma, and breast carcinoma in young patients), intratrabecular, or of mixed type.

**Q3: What is the mode of spread to the rib?**

Bone marrow metastases is either via the transpulmonary route, or the vertebral venous system—the latter results in metastases to the spine. The frequency of bone metastases via the vertebral venous plexus without pulmonary metastases is 30% for carcinoma of the prostate, 10.4% for uterus, 7.4% for breast, and 3.5% for stomach.

**Discussion**

The patient has a rib secondary from an obscure primary. Detailed gastrointestinal tract evaluation and chest evaluation were non-contributory.

Moriwaki has described different types of rib tumours. These can be further classified as benign or malignant—the latter are often secondaries from a primary in the lung (35%).

Micrometastases from gastro-oesophageal malignancy has been reported. Oesophagogastric malignancy results in micrometastases of ribs in 88% and in the iliac crest in 15%. These are independent of the histological type, nodal status, and neoadjuvant therapy. Macrometastases is less common.

Yang et al described three sonographic patterns on cross section of abnormal ribs; ultrasound guided biopsy yields a 100% result without any complications and is an ideal method of confirming the diagnosis.

Management depends on the histopathology and is often directed to the primary site. Large tumours, as in our case, require surgical resection followed by chemotherapy. The patient is doing well six months after the resection. Failure to find the primary tumour poses problems in management. Yet it remains controversial whether the prognosis improves when the primary tumour is identified by intensive diagnostic search as in the present case. Overall the prognosis is poor.

**Final diagnosis**

Adenocarcinoma of the rib.

**References**

A smoker with an apical mass

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