Iatrogenic groin pain

Q1: What is the likely clinical diagnosis?

The triad of groin pain, hip flexion, and femoral neuropathy indicates illoposa sheath haemorrhage. This condition occurs in patients with inherited coagulation disorders, particularly haemophilia A, and in patients on oral anticoagulants.23 Spontaneous haemorrhage occurs deep to the iliacus fascia from the iliacus or psosas 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 psosas 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 psosas 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 illoposa 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 some residual non-disabling thigh weakness at the time of discharge.

Discussion

Haemorrhage into the iliacus and or psosas muscles is a well recognised complication of overanticoagulation, as well as of haemophilic 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 coexist. Alternatively bleeding may commence in the psosas major muscle initially or spread from the iliacus muscle to the psosas. In this case involvement of other components of the lumbo-sacralplexus, including the obturator nerve and the lateral femoral cutaneous nerve of the

Figure 1 Enterolith.
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 flexed and laterally rotated deformity of the hip may ensue. Passive hip extension is restricted and painful. Delayed development of bruising 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 and 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 if the clot 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 embolisation, 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. Tuberculous granuloma is the most specific finding on histology apart from infiltration of lamina propria by lymphocytes and plasma cells with aggregates 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. There may be either 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 painless 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 due to inflammation, the smooth muscle cells of the intestine activate a complex chain of events, causing muscle relaxation to inhibit the smooth muscle cells' response to inhibitory neurotransmitters (NETs) and result in the muscle contractions.

An important differential diagnosis of ileal stricture in the present case is Crohn's disease, based on the peroperative findings of mucosal thickening and ileal stricture at enteroscopy. A featureless outline of a diseased segment, referred to as the "string sign". Strictures themselves are painless and may not require treatment. But sometimes, these areas become so narrow and result in a partial or total obstruction.

A rare clinical example of ileal stricture is found in patients with Crohn's disease, where the stricture is due to a longitudinal ulceration of the intestine. This is followed by gradual increase in paraesthesiae of the leg, this was seen in 29% of cases.

Khwaja and Subbuswamy reported that ischaemic strictures of the small intestine from northern Nigeria. This is an ideal investigation for study of the small intestine, both for focal lesions 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 the diagnosis. In the case reported, the postoperative 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 necessary when an individual is symptomatic. Steroids, aminosalicylic acid preparations, immunomodulators, and verapamil have been used during the inflammatory phase; the latter is used to reduce the smooth muscle response to intestinal injury and prevents collagen deposition. When the affected segment is fibrosed 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 lobes. 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 subtle 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
- Sarcoïdosis
- 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 fibrogenic or a “benign” form of pneumoconiosis. The pathology of siderosis is characterized by perivascular and peribronchiolar aggregations of dark pigmented iron oxide particles present extracellularly in alveolar spaces and walls as well as in macrophages. 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.

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 symptoms may be present in smokers or if there is concurrent exposure to silica or asbestos.
- Centrilobular nodules with diffuse distribution are seen on a HRCT lung scan.
- Diagnosis is made on radiological features and occupational exposure.

Final diagnosis

Siderosis.

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 lesions.
- Neurinobromatosis.
- 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 xanthochromic with an opening pressure of 30 cm H2O. CSF protein was 30 g/l, glucose 5.4 mmol/l, white blood cell count 40 × 10^3/l, and red blood cell count 160 × 10^3/l. This was suggestive of a CSF block in the spinal canal.

Magnetic resonance imaging of the spine showed a intradural, extramedullary heterogeneously enhancing mass lesion 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 include:

- Spinal tumour with raised intracranial pressure
- Siderosis
- Hypersensitivity pneumonitis
- Respiratory bronchiolitis
- Diffuse panbronchiolitis
- Infectious bronchiolitis
- Sarcoïdosis
- Metastases
- Tuberculosis
- Malignant meningitis
- Neurinobromatosis

Figure T1 T1 weighted gado enhanced sagittal magnetic resonance image of the thoracolumbar spine showing a well circumscribed 5 × 2 cm mass at D11–L2 level.
Viscocity could delay CSF absorption and in clear.

For the raised intracranial pressure in these youn, female patient.

Diplopia.

Raised CSF pressure causes increased pressure are ependymoma, schwannoma, meningioma, neurofibroma, and glioma. Common clinical features seen among the patients with spinal cord tumours have been reported in the past. Raised CSF protein (causing delayed absorption of CSF due to increased viscosity) and leptomeningeal inflammation (probably due to the toxic effect of protein secreted by the spinal cord tumours) are the proposed mechanisms for the raised intracranial pressure in patients with spinal cord tumours.

Discussion

This patient underwent excision of the tumour and the biopsy was reported as a myxopapillary ependymoma. After this, raised intracranial pressure and lateral rectus palsy resolved. Bilateral papilloedema and diplopia without an expanding intracranial mass lesion in a patient with spinal cord tumours have been reported in the past. Raised CSF protein (causing delayed absorption of CSF due to increased viscosity) and leptomeningeal inflammation (probably due to the toxic effect of protein secreted by the spinal cord tumours) are the proposed mechanisms for the raised intracranial pressure in patients with spinal cord tumours.

Q1: Describe the abnormalities shown in figs 2 and 3 (see p 629)

The chest radiograph demonstrates a dumbbell shaped neoplasm measuring 40 × 30 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 × 30 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 canal, which is minimally displaced.

Q2: What is the differential diagnosis?

Mediastinal neoplasms encompass a long list of histologically diverse lesions that can arise from a wide variety of mediastinal structures. In adults, most primary mediastinal neoplasms can be classified in one of four categories: thymus-derived neoplasms, neurogenic tumours, lymphomas or germ cell neoplasms. The differential diagnosis of masses in the posterior mediastinum includes neurogenic neoplasms (often neurofibromas and schwannomas), aneurysms of the descending aorta, oesophageal tumours, infectious processes including abscesses, and disorders of the thoracic spine, such as spondylolisthesis.

In adults, most posterior mediastinal tumours are of nerve sheath origin and are often benign and asymptomatic.

Q3: What was the definitive procedure?

Given the likelihood of this lesion being a benign neurogenic neoplasm, the patient proceeded to a right posterolateral thoraectomy with excision of the lesion.

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

The 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.

Outcome

In view of the proximity of the lesion to the spinal cord, thoraectomy rather than thoracoscopic resection was considered the preferred surgical approach. The patient proceeded to a right posterolateral thoraectomy in the fourth intercostal space, and a soft yellow dumbbell shaped mass measuring 30 × 30 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 loosely 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 paragangliotic origin and are generally located in the posterior mediastinum. Neurogenic tumours of the mediastinum with an
intrapulmonary route, or the vertebral venous plexus without pulmonary resection. The specimen is an excision biopsy of the rib?

Q2: What are the different types of rib tumours?
The bone metaseses is either osteoclastic (often squamous cell carcinoma), osteolytic (prostate carcinoma, poorly differentiated adenocarcinoma, and breast carcinoma in young patients), intratrabeclular, 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.

Final diagnosis
Adenocarcinoma of the rib.

References