

Letters

GIANT CELL TUMOUR OF THE TENDON SHEATH - AN UNUSUAL CAUSE FOR LOCKING OF THE KNEE JOINT.

Editor,

Locking of the knee is a common presentation at orthopaedic outpatient departments. These patients normally require magnetic resonance imaging (MRI) where there is a clinical suspicion of a soft tissue lesion. The most common lesions are meniscal tears and typically present with associated pain. We present an unusual case of a giant cell tumour arising from the tendon sheath causing painless locking of the knee joint.

Case report: A 39-year-old male presented with a twelve-month history of painless locking in his left knee. He worked as a roofer and found his symptoms worse when climbing ladders. He was referred by his general practitioner who suspected a meniscal tear. There was no history of any trauma to the knee. Clinical examination revealed no swelling, effusion or joint line tenderness around the knee, and a full range of movement. A mass arising from the medial aspect of his patella, which was mobile within the knee joint, was palpable. The lesion was firm but not bony in nature, and was not visible on X-ray. The mass was presumed to be a soft tissue lesion and, because of the hazards associated with his occupation, the patient proceeded directly to arthroscopy rather than MRI. At arthroscopy a large intra-articular lesion was identified originating posterior to the medial patella. The size of the lesion prohibited removal during arthroscopy, and was therefore excised in its entirety via a medial parapatellar incision. Histology showed the specimen to be a giant cell tumour of the tendon sheath measuring 40 x 35 x 15mm (Fig 1). Postoperative recovery was uneventful and at two-month review the patients symptoms had resolved.

Discussion: Giant cell tumours of the tendon sheath (GCTTS) are benign soft tissue masses, typically found on the flexor surface of the hand and wrist¹. They are more common in males with an average age of presentation of 30-50 years². These tumours are classified in two types; the common localized type and the rare diffuse type. The more localised form accounts for 88% of cases effecting the hands and feet arising from the synovium of the tendon sheath². It is unusual for giant cell tumours to involve larger joints and to be intra-articular. In large joints diagnosis is difficult because the signs and symptoms can be non-specific³. The rare diffuse form, occurring in joints such as the knee and ankle, is considered to be an extra-articular extension of a primary intra-articular pigmented villonodular synovitis (PVNS). PVNS and GCTTS share similar histological characteristics and are regarded as different manifestations of synovial proliferations².

As in this case, plain X-rays are often of limited benefit. MRI is an important diagnostic modality. T1 and T2 weighted images show a low intensity homogenous signal for a GCTTS due to the presence of dense fibrous tissue³.

Ideally GCTTS should be completely excised, but may have to be incomplete due to the nature of spread into the surrounding synovium⁴. Zhang *et al.* reviewed 12 cases of intra-articular

GCTTS within the knee, and reported that nine cases were misdiagnosed with meniscal injuries or chronic synovitis. Only three cases had the diagnosis confirmed by MRI prior to surgery⁵. They found no incidence of recurrence in any of the cases. Further studies have looked at recurrence and quote figures around 10-20% rising to 44% if excision was inadequate².



Fig 1. Giant cell tumour of the tendon sheath measuring 40 x 35 x 15mm being excised through a medial parapatellar incision.

Conclusion: This case highlights that GCTTS, although rare, can be an unusual cause for locking of the knee joint. Its presentation may mimic a meniscal tear, but a history of no previous trauma to the knee and painless locking are important discriminating symptoms.

Conflicts of Interest: None declared.

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CLIMATE CHANGE AND ITS IMPACT ON HEALTH

Editor,

On the 29th January 2008, a landmark conference entitled 'Climate Change and its Impact on Health' took place at the Royal College of Physicians in London. Although the

audience was from a medical background, most of the speakers were not – the former Government Chief Scientist, a City financier, a climatologist, Professors of Palaeontology and Peace Studies, and the former Director General of the Confederation of British Industry all talked about different aspects and they were joined by the Editors of the *Lancet* and *British Medical Journal*, and a Professor of Public Health.

There were some very clear messages. None of the speakers had any doubt that climate change – a rise in average temperature and a rise in atmospheric carbon dioxide levels – first was real, second was due to the activities of humans and third was likely to have serious consequences for human health because of an increase in infectious diseases, heat-related diseases and malnutrition. There was also no doubt that the only solution in town was to reduce carbon emissions and that this needed to be done by concerted government and inter-governmental action. There was also agreement that currently-available technologies were able to do this. Those cited were fuel-efficient vehicles, reduced vehicle use, more nuclear energy, substitution of coal by gas for electricity generation, carbon capture and storage and more use of wind, sun, hydrogen and biomass to generate power. Used together these could stop any rise in carbon emission. The cost arguments were set out in the Stern Report published in 2006¹. The cost of failing to deal with climate change would be at least 20% of global gross domestic product (GDP) whereas it would cost only 1% of GDP to act now on global warming.

There was also general agreement that the medical profession could act both personally and politically. Personal contributions included using energy-saving light bulbs, reducing travel by car, using public transport and cycling or walking to work. Politically the medical profession should be at the forefront of lobbying for effective government action on climate change and to this end the Climate and Health Council has been established (www.climateandhealth.org). The obvious analogy here is the leading role of the profession against cigarette smoking.

The closing address of the conference was given by Dr James Hanson, Director of the NASA Goddard Institute of Space Studies who suggested a moratorium on further coal-fired power stations and urged individuals to influence those who are elected to Governments. This address was delivered by videolink so Dr Hanson did not have to increase his carbon footprint by flying to London. (The possibility of reducing the NHS carbon footprint by practising more medicine this way was barely mentioned at the conference.)

Although this conference may have scared many in attendance about the future of our planet it also conveyed a message of optimism. This optimism though, was tempered by the proviso that corrective action needs to be taken now rather than later, and that to do that we all need to emerge from our states of denial.

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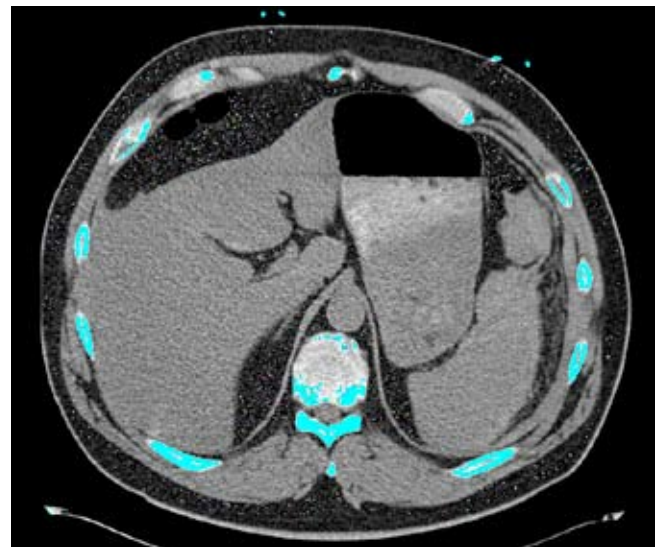
IATROGENIC SPLENIC INJURY IN PERCUTANEOUS PROCEDURES

The potential for splenic injury during left sided thoracentesis or percutaneous renal biopsy is well known, its occurrence has been rarely reported¹. In a series of 244 incidental splenectomies, only one was secondary to left thoracentesis². Each year about 600 new cases of extrinsic allergic alveolitis are diagnosed in the UK³. Lung biopsy is an important diagnostic tool for diffuse lung disease. Others include bronchoalveolar lavage and high resolution computed tomography³. We present a case of delayed splenic rupture following percutaneous lung biopsy, which required urgent laparotomy.

Case Report: A 48-year-old gentleman presented with left upper quadrant pain and shortness of breath for eight hours prior to admission. There was no history of trauma, haematological or storage diseases. The patient underwent a left lung biopsy two months earlier which had led to the diagnosis of extrinsic allergic alveolitis.

On examination, he was comfortable and haemodynamically stable. Respiratory and cardiovascular examinations were unremarkable, abdomen was soft and non-tender. Initial haematological investigations, cardiac enzymes and electrocardiogram were normal. A provisional diagnosis of inferior wall myocardial infarction was made.

He became progressively hypotensive and developed abdominal distension with left upper quadrant tenderness. Two scars were noted, one over 6th and the other over 8th intercostal spaces.



Urgent computed tomography of abdomen and pelvis revealed complex fluid collection around the spleen and free intraperitoneal fluid (fig 1). At emergency laparotomy, 2.5 litres of intraperitoneal blood was removed. A large clot was identified under left dome of diaphragm. A small-healed wound over the lateral surface of the spleen was identified. No

active bleeding was evident and the findings were consistent with those of a ruptured subcapsular splenic haematoma. Postoperative recovery was uneventful and the patient was discharged on day seven.

Discussion: This case highlights the delay with which an iatrogenic splenic injury can present. The most important indicator in this case, which could relate to splenic injury, was a scar near the splenic region following lung biopsy. Clinical problems after splenic rupture have been classified into three groups characterized by the delay in presentation and type of symptoms⁴. Group one: acute ruptured spleen, Group two: delayed ruptured spleen, and Group three: occult ruptured spleen. Our case was group two, with delayed presentation two months after injury.

Computed tomography is the gold standard for investigating splenic injuries. Grading scales based on computed tomography findings can predict the likelihood of successful non-operative management, which is often possible if the splenic hilum is intact (even when capsular disruption is present)⁵. However, due to haemodynamic instability in this case, percutaneous drainage was not performed. As new cases of diffuse lung disease are being investigated, physicians should be increasingly aware of the possibility of splenic injury after lung biopsy. Splenic injury should be considered if haemodynamic instability occurs even after two months of lung biopsy.

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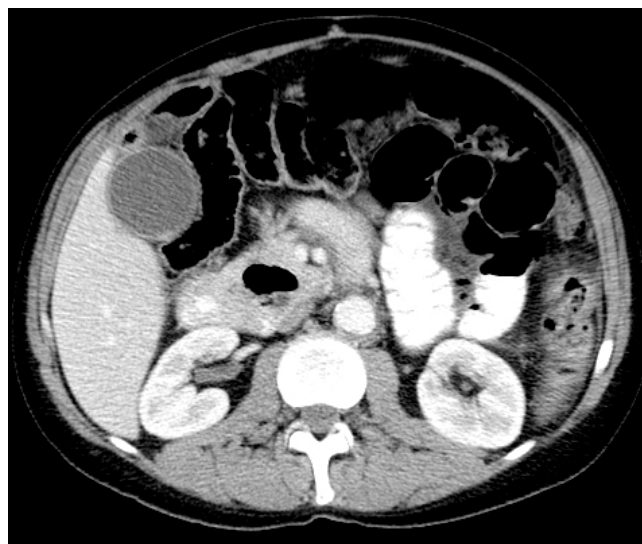
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CHYLOUS PERITONITIS WITH SMALL BOWEL OBSTRUCTION

Chylous ascites may present with a number of abdominal

complications^{1,2}. We present a case of chylous peritonitis presenting as small bowel obstruction.

Case Report: A 44 year old male presented to the accident and emergency unit with crampy abdominal pain of a two days duration, worse over the previous day. He had a background history of a partial oesophago-gastric resection for Boerhaave's syndrome some months earlier. On examination there was marked abdominal distension with epigastric tenderness. Plain abdominal X-ray showed a large fluid filled loop suggestive of a volvulus. CT scan (fig 1) confirmed a volvulus of the small bowel with a twist of mesentery root and likely venous obstruction.



At laparotomy there was a volvulus around a band from the apex of the anti-mesenteric border of the small bowel to the fourth part of the duodenum. The entire small bowel was dusky with venous engorgement but viable. There was striking engorgement of the lymphatics in the wall of the small bowel and 400ml of milky chylous fluid free in the peritoneal cavity. The patient made an uneventful recovery after surgery.

TABLE I

| Causes of Chylous fluid |
|--|
| 1. Abdominal surgery |
| 2. Blunt abdominal trauma |
| 3. Malignant neoplasm's - Hepatoma, small bowel lymphoma, Small bowel angiosarcoma, and retroperitoneal lymphoma |
| 4. Spontaneous bacterial peritonitis |
| 5. Cirrhosis - Up to 0.5% of patients with ascites from cirrhosis may have chylous ascites. |
| 6. Pelvic irradiation |
| 7. Peritoneal dialysis |
| 8. Abdominal tuberculosis |
| 9. Carcinoid syndrome |
| 10. Congenital defects of lacteal formation |

Discussion: Chylous peritonitis is the extravasation of milky chyle into the peritoneal cavity. This can occur de novo as a result of trauma or obstruction of the lymphatic system. An existing clear ascitic fluid can turn chylous as a secondary event. A true chylous effusion is defined as the presence of

ascitic fluid with high fat (triglyceride) content, usually higher than 110 mg/dL.

Chylous fluid in the peritoneal cavity is a rare clinical condition that occurs as a result of disruption of the abdominal lymphatics. Multiple causes have been described (table I). Congenital chylous ascites is the commonest cause of chyloperitoneum in young children. Other causes in children include idiopathic or obstructive lesions caused by malrotation, intussusception, incarcerated hernia, lymphangioma, blunt trauma, liver disease, and tuberculosis. In children, malrotation and volvulus contribute to chylous ascites³. Volvulus of the midgut may result in several manifestations. Venous and lymphatic obstructions occur first because of lower intravascular pressures. Vascular congestion leads to bowel oedema and possible oozing of blood, causing melaena. Lymphatic congestion causes the formation of a mesenteric cyst and chylous ascites.

Milky ascites is subdivided into three groups: True chylous ascites - Fluid with high triglyceride content, Chyliform ascites - Fluid with a lecithin-globulin complex due to fatty degeneration of cells, and Pseudo-chylous ascites - Fluid that is milky in appearance due to the presence of pus. Dietary chylomicrons are absorbed in the small intestines and gradually pass along larger omental lymphatics to the cisterna chyli located anterior to the second lumbar vertebra. The cisterna is joined by the descending thoracic, right and left lumbar, and liver lymphatic trunks, and, collectively, these form the thoracic duct, which passes through the aortic hiatus and courses through the right posterior mediastinum and eventually enters the venous system. The thoracic duct carries lymphatic drainage from the entire body, except for the right side of the head and neck, right arm, and right side of thorax. Chylous effusions develop when these channels are injured or obstructed. Abdominal distension is the most common symptom, and rarely, it may present as acute peritonitis.

As chylous peritonitis is a manifestation rather than a disease, the prognosis depends on the treatment of the underlying disease or cause. Few cases presenting as chylous peritonitis are reported in literature

Three cases presenting as acute appendicitis have been reported^{4,5}.

The authors have no conflict of interest.

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MESOTHELIOMA – “NOT JUST IN THE CHEST”

Editor,

Malignant deciduoid mesothelioma (MDM) is a rare phenotype of epithelioid mesothelioma, which most commonly occurs in the peritoneal cavity of young females. MDM remains a challenge even to the most astute diagnostician with the differential diagnosis being benign pseudotumoral decidualosis. It carries a dismal prognosis.

Case Report: A previously healthy 31-year-old woman presented with a short history of increasing abdominal girth and shortness of breath without weight loss. She smoked 15 cigarettes per day and had no risk factors for chronic liver disease or prior history of asbestos exposure. There was no family history of neurofibromatosis. Clinical examination revealed ascites in the absence of signs of chronic liver disease, café au lait spots or lymphadenopathy. Diagnostic paracentesis revealed no evidence of bacterial or mycobacterial infection. The serum ascites albumin gradient was 1.1g/dL. Cytology was consistent with benign reactive mesothelial cells although no leucocyte reaction was noted.

Haematological, tumour markers, inflammatory markers and biochemical parameters were in the normal range. A chest radiograph showed no signs of pericarditis, pleural plaques or effusions. Ultrasonographical and CT scanning demonstrated ascites with normal hepatic echotexture and antegrade flow in the portal vein. No thoracic lesions were seen. A diagnostic laparoscopy drained 9L of ascites and numerous small nodules were observed concentrated around the small bowel.

Histological examination of these nodules with conventional stains was consistent with a mesothelial process although it was impossible to differentiate between a benign reactive or neoplastic aetiology. Typical features of epithelioid mesothelioma were not observed. Further expert opinions were sought and immunostaining is shown in Figure 1.

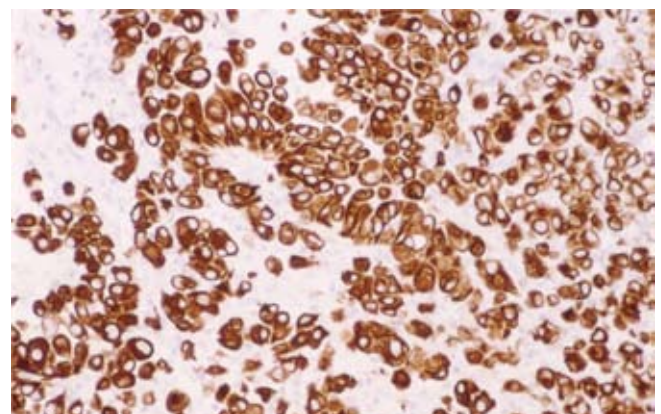


Fig 1. Immunostaining revealed strong positivity for calretinin and cytokeratin which are considered to be sensitive and relatively specific markers for MDM.

These findings were consistent with the diagnosis of MDM. Combination chemotherapy with pemetrexed and cisplatin was initiated but she required multiple hospital admissions

for therapeutic abdominal paracentesis. Our patient died 14 months after diagnosis.

MDM was first characterised in 1994¹ and accounts for approximately 4% of all mesotheliomas². In contrast to “classical” pleural mesothelioma, the most common site of disease is the peritoneum although a pleural form has been described. The most common presenting feature is ascites. There is a female preponderance (F = M ratio, 1.4:1) and younger age at presentation (<40 years). Rates of asbestos exposure are generally lower than “classical” mesothelioma (c.35%)².

MDM is a highly malignant neoplasm with mean survival time reported as 7.33 months (range 1-29.4 months)². Treatment is not curative and the main therapeutic goal is symptomatic palliation. To date, there is no standard treatment for MDM. Using established regimens for peritoneal mesothelioma, limited success has been observed using cytoreductive surgery and intraperitoneal hyperthermic chemotherapy³.

MDM is the example *par excellence* for the difficulties that clinicians face in the differentiation between benign and malignant disease. Moreover, this case highlights the usefulness of diagnostic laparoscopy in investigating unexplained ascites. MDM is, and is likely to remain, a diagnostic challenge for clinicians, even the astute ones.

The authors have no conflict of interest

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Correspondence on articles on this issue of the journal can be sent by email as an attached word file, or by post on CD to the editorial office, and should be less than 300 words and a maximum of three references and one table or figure.