A man with swollen calf and discolouration of the foot

Q1: What is the likely diagnosis?
The differential diagnosis of calf swelling includes deep vein thrombophlebitis, a popliteal cyst, popliteal varices or artery aneurysm, ganglia, neural tumours, sarcoma, and haemangioma.

When the swelling is associated with tenderness, diagnostic considerations should include tear of medial head of gastrocnemius or plantaris muscles, cellulitis, fasciitis, compartment syndrome, venous insufficiency, and ruptured popliteal cyst.

One of the most common and clinically important diagnosis is DVT. Since it leads to pulmonary embolism it prompt anticoagulation is indicated. On the other hand, anticoagulation after ruptured Baker's cyst can cause bleeding and posterior compartment syndrome.

Q2: What physical examination sign was the clue for the final diagnosis?
Ruptured Baker's cyst simulates DVT of the calf veins, both causing painful, swollen calf with overlying skin erythema. Homan's sign, an accepted but non-sensitive sign, may also appear in both. Ecchymosis of the foot, especially in a malleolar region, termed the haemorrhagic crescent sign, has been reported after this rupture. Although this is a relatively rare finding, it is not found in DVT and its presence should lead the clinician to the diagnosis of ruptured Baker's cyst.

Q3: What are the diagnostic modalities you would use to confirm the diagnosis?
Ultrasound (duplex scan) is the method of choice for evaluation of posterior calf disease, although it may be difficult to diagnose Baker's cyst after its rupture. Recently, with improvement of ultrasonic images, this method can simultaneously confirm ruptured Baker's cyst and exclude DVT. Alternative imaging modalities are magnetic resonance imaging and computed tomography. Previously, the gold standard diagnostic method for ruptured Baker's cyst was an arthrogram. In this procedure the knee joint is injected with contrast material, and after mobilisation of the extremity, the radiographic dye can be demonstrated in the cyst and extravasating to the surrounding calf structures.

Q4: What treatment is indicated?
Ruptured Baker's cyst is treated by elevating the affected extremity, local heating, and intra-articular corticosteroid injection after aspiration of the synovial fluid from the knee.

Discussion
In our patient diagnosis of ruptured Baker's cyst was confirmed by ultrasonic examination obtained during his visit to the rheumatology clinic. A fluid collection along the intermuscular fascia layers of the calf (fig 1 below) was demonstrated. The patient was treated as described above, with disappearance of the symptoms within two weeks.

Popliteal cyst is usually caused by tear of the knee joint capsule, allowing a communication between the joint space and gastrocnemius or semimembranous bursa. It was named after the British surgeon William Morton Baker, who described an association of this cyst with knee synovitis. Popliteal cysts are secondary to degenerative (osteoarthritis) or inflammatory arthritis with increased synovial fluid production. The most common complication of Baker's cyst is a rupture. This is usually caused by a rise in intra-articular pressure during powerful knee extension.

Extravasation of inflammatory and proteolytic content of synovial fluid leads to inflammation of surrounding calf structures. The patient with a ruptured cyst will therefore present with a hot, red, tender and swollen posterior calf, while the cyst is no longer palpable, and the knee is less swollen. Homan's sign may be positive in both DVT of the calf and ruptured Baker's cyst. Thus the two entities are almost indistinguishable by physical examination.

The only clinical sign differentiating ruptured Baker's cyst from DVT is bruising below the malleolus. This results from drainage of inflamed synovial fluid dissecting the calf structures to the foot. Discoloration of the malleolus area should therefore alert the physician to the diagnosis of ruptured Baker's cyst.

Finally, DVT and Baker's cyst are not mutually exclusive diagnoses as the cyst pressure on a calf vein may cause stasis and thrombosis.

Final diagnosis
Ruptured Baker's cyst.

References

Figure 1: Ultrasound of the posterior medial site of the left calf, showing an anechoic lesion 3.4 cm x 7.6 cm with some echogenic debris on the dependent side.

Young male with pancytopenia: an unusual cause

Q1: What is the finding on slide of bone marrow examination (fig 1; see p 300)?
The bone marrow aspiration smear shows microfilariae of Wuchereria bancrofti. The microfilariae (embryos) are usually found in the blood of infected patients. They have sheathed, transparent bodies 290 μm in length and 6–7 μm in width. They have a hyaline sheath and central column of nuclei, which do not extend up to the tail.

Q2: What are the causes of pancytopenia?
Pancytopenia is the simultaneous presence of anaemia, leucopenia, and thrombocytopenia. Pancytopenia therefore exists in adults when haemoglobin concentration is less than 135 g/l in males or 115 g/l in females, the leucocyte count is less then 4 x 10³/μl, and platelet count is less than 150 x 10³/μl. Presenting symptoms are usually due to anaemia or thrombocytopenia. Leucopenia is an uncommon cause of the initial presentation in the patients.

Causes of pancytopenia

(A) Pancytopenia with hypopcellular bone marrow
• Acquired aplastic anaemia.
• Inherited aplastic anaemia (Fanconi's anaemia).

(B) Pancytopenia with cellular bone marrow
1: Primary bone marrow diseases
• Myelodysplastic syndromes.
• Paroxysmal nocturnal haemoglobinuria.
• Myelophthisis.
• Bone marrow lymphoma.
• Hairy cell leukaemia.

2: Secondary to systemic diseases
• Systemic lupus erythmatosus.
• Hypersplenism.
• B12, folate deficiency.
• Overwhelming infection.
• Alcohol.
• Brucellosis.
• Sarcoidosis.
• Tuberculosis.
• Leishmaniasis.

Q3: How will you treat this patient?
The drug of choice for treatment of filariasis is diethylcarbamazine. It is effective in killing microfilariae. The effect of the drug on adult worms is uncertain. The dose of diethylcarbamazine used for treatment of bancroftian filariasis is 6 mg/kg body weight per day orally for 12 days given preferably in divided doses after meals. Diethylcarbamazine may produce severe side effects: (A) those caused by the drug itself—for example, headache, nausea, vomiting, and dizziness. These are observed a few hours after the first dose of diethylcarbamazine and generally do not last for more than three days. (B) Allergic reactions due to destruction of the microfilariae and adult worms—for example, fever, orchitis, lymphadenitis, transient lymph oedema, and
hydrocele. The local reactions tend to occur later in the course of treatment and last longer. If the drug is given in divided doses, the systemic reaction is much less severe and less frequent after the second dose and rare at subsequent doses. These reactions disappear spontaneously and rarely require interruption of treatment.

Other drugs

Ivermectin—It is a semisynthetic macrocide antibiotic with activity against various nematodes and ectoparasites. It causes a gradual decrease in microfilariae level over 2–4 weeks to less than 10% of pretreatment values. However, these low levels are sustained for at least six months after treatment. Maximum effectiveness is seen at the higher dosage of 200 µg/kg body weight.

Albendazole—400 mg twice for 21 days also has macrofilaricidal efficacy.

Discussion

Two types of filarial infection—that is, W bancrofti and Brugia malayi, occur in India. Out of these two, bancroftian filariasis is responsible for 98% of infection. Man is the definitive host and the intermediate host is bancroftian and burgian filariai. The adult worm is found in the lymphatic system, skin, and serous membranes of man. The males are about 40 mm long and females 50–100 mm long. Females give birth to as many as 50 000 microfilariae per day, which find their way into blood circulation via the lymphatic system. The lifespan of microfilariae is not known exactly, but is probably up to a year or more. The adult worms live in the afferent lymphatic or sinususes of lymph nodes for years and causes lymphatic dilatation and thickening of the vessel walls. A lymphatic vessel remains patent as long as the worm remains viable and death of the worm leads to enhanced granulomatous reaction and fibrosis resulting in lymphatic obstruction. The manifestations of filariasis are due to both the direct effects of worms and the immune response of the host to the parasite. Classical disease manifestations can be divided into two distinct clinical types: (A) lymphatic filariasis caused by parasites in the lymphatic system and (B) occult filariasis caused by immune hyper-responsiveness of the human host (for example, tropical pulmonary eosinophilia) which most commonly affects persons of Asian origin. The lymphatic filariasis are asymptomatic microfilaraemia, acute adenolymphangitis, and chronic lymphatic disease. The chronic obstructive lesion may present as hydrocele, elephantiasis, and chyluria. Rarely chylous arthritis, filarial pleural effusion, glomerulonephritis, and breast lump have been described. Our patient manifested the disease as pancycopenia due to the involvement of bone marrow. Until now bone marrow filariasis has not been described. It is possible that liberation of some toxic metabolites by growing larvae—that is, microfilariae, might have caused toxic suppression of the bone marrow. The patient was given diethylcarbamazine and two whole blood transfusions. The patient started improving after a week as pancycopenia due to the involvement of bone marrow. The haemoglobin value rose to 70 g/l. The peripheral blood smear examination showed considerable improvement in total leucocyte and platelet counts. Bone marrow examination repeated after two weeks showed responsive bone marrow and degenerated microfilariae. However, since this is the first case report of bone marrow filariasis, the exact mechanism of bone marrow invasion and suppression needs further elucidation.

Final diagnosis

Filarial infection mediated toxic depression of bone marrow.

References


Recurrent painful locking of the elbow joint

Q1: What are the features seen on the radiographs (see p 301)?

The radiographs of the left elbow show multiple radio-opaque loose bodies of variable size.

Q2: What is the likely diagnosis?

The diagnosis is synovial chondromatosis of left elbow.

Q3: What is the line of management?

If the patient is symptomatic, removal of loose bodies and synovectomy by arthroscopy or arthrotomy is advocated.

Discussion

Synovial chondromatosis is a synovial proliferative disease in which cartilaginous or osteocartilaginous metaplasia occurs within the synovial membrane. It most commonly involves knee and hip joints, but any joint, bursa, or tendon sheath may be affected.

Involvement of the elbow joint is very rare. Patients are observed in their second to seventh decades of life, and usually report a gradual onset of pain, stiffness, or an enlarging mass around the affected joint. Limitation of motion is a characteristic finding on clinical examination.

There are three phases of the disease:

1. Early with synovial chondrometaplasia but no loose bodies.
2. Transitional with active synovial disease and loose bodies.
3. Late with loose bodies but no synovial disease.

Routine roentgenograms may show multiple loose bodies, but especially in the hip, routine films are often normal. Other studies such as arthrography, magnetic resonance imaging, or arthroscopy may be needed.

At surgery multiple loose bodies, both free and attached to the synovium, are visible. Microscopically discrete nodules of disorganised cartilaginous tissue in the synovium, characterised by cellular crowding with cytologic atypia is remarkable. This disorder appears as an arthroscopic lesion in patients with a history of irritable joint (either by arthroscopy or open operation). Recurrence frequently occurs after surgery and this can progress to osteoarthritis. There are several reports of malignant transformation to chondrosarcoma.

Final diagnosis

Synovial chondromatosis of the left elbow joint.

References


A verrucous lesion of the palm

Q1: What is your diagnosis?

The diagnosis is cutaneous rhinosporidiosis. The patient gave past history of reddish polyps in the nostrils. He had a reddish friable mass in the nostrils. He had a reddish friable mass.

Q2: How can you confirm your diagnosis?

Giemsastained imprint smears taken from the surface of the lesion showing typical sporangia can easily clinch the diagnosis. Scrape cytology of the lesion demonstrates typical spherical spores of variable sizes with transparent capsules, and eosinophilic globular bodies (8–10) within the spores in haematoxylin and eosin or Papanicolaou stained smears. Fine needle aspiration cytology from the lesion could be an additional diagnostic tool.

Causes of multiple loose bodies

• Osteochondritis dissecans.
• Osteoarthritis.
• Synovial chondromatosis.
• Tuberculosis: fibrous or fibrinous loose bodies.

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Box 1: Differential diagnosis

• Warts.
• Verrucous tuberculosis.
• Granuloma pyogenicum.
• Donovanosis (genital lesions).
• Condyloma acuminata (genital lesions).
Histopathology reveals enormous numbers of mycotic elements in the subepithelial connective tissue. These consist of sharply defined globular thick walled cysts (sporangia) up to 0.5 mm in diameter, which contain numerous rounded endospores, 6–7 μm in diameter. Immature and collapsed sporangia are also present. Occasionally, microabscesses may be formed.1,4

Q3: What is the treatment of this disease?
Surgical excision of the lesion is the preferred treatment.4 Dapsone and electodesiccation of the polyp are useful.1

Discussion
Rhinosporidiosis is a chronic granulomatous mycosis caused by Rhinosporidium seeberi. The organism has never been isolated in vitro and its taxonomic position is not clear.5 Careful inspection of the surface of the warty lesions and the presence of typical nasopharyngeal lesions helps in suspecting cutaneous rhinosporidiosis.6 The diagnosis can be confirmed by demonstrating typical sporangia and spores in histopathology and imprint smears.7,8 Surgical removal and electodesiccation are the treatments of choice.8,9 Dapsone may arrest the maturation of sporangia and accelerate degenerative changes in them. The effete organisms are then removed by an accelerated granulomatous response. For the same reasons, dapsone has been used to reduce postoperative recurrences as well.10

Final diagnosis
Cutaneous rhinosporidiosis.

References

An unusual case of clinico-radiological dissociation

Q1: What are the findings on the chest radiograph and computed tomogram of the chest (see p 302)?

The chest radiograph shows diffuse bilateral symmetrical minute nodular shadows involving almost the entire mid and lower zones with sparing of the apices. The heart and mediastinal shadows are totally obscured by these nodules, which are quite dense. The mediastinal window of the computed tomodogram shows bilateral diffuse micronodular alveolar calcification.

Q2: What is the likely diagnosis?
The diagnosis is pulmonary alveolar microlithiasis. The chest radiographic picture of “sandstorm” appearance is characteristic and diagnostic of this condition.1

Q3: What additional investigations should be performed to confirm the diagnosis?
The diagnosis of pulmonary alveolar microlithiasis can most often be made with confidence from the chest radiographic pattern and the striking radiological-clinical dissociation. Microliths (round or oval, irregular shaped with concentric laminated appearance) can be identified in sputum, bronchoalveolar lavage fluid, and transbronchial lung biopsy specimens (fig 1). Open lung biopsy is seldom, if ever, indicated.1 The microliths also bind technetium-99m, which can be a diagnostic adjunct.2

Q4: What is the treatment and prognosis of the condition?
There is no definitive treatment for pulmonary alveolar microlithiasis. Treatment is largely supportive. Bronchopulmonary lavage and corticosteroids have no effect.3 Although usually asymptomatic at presentation, pulmonary alveolar microlithiasis, on rare occasions, can produce functional abnormalities such as progressive breathlessness, pulmonary hypertension, and cor pulmonale. The prognosis is variable. Many patients remain asymptomatic with stable chest radiographs for several years. When disease does progress, it may do so very slowly. Cases have been reported in which respiratory failure and death ensued after a period as long as 40 years.

Discussion
Pulmonary alveolar microlithiasis is a rare disease of unknown aetiology and is one of the few conditions in which gross radiographic changes are present in the face of minor clinical symptoms.4 The disease is characterised by the deposition of calcium and phosphorus microliths (calcispherites) within the alveolar space.5 Most patients are asymptomatic at presentation and incidentally detected to have an abnormal chest radiograph. About 20–40% of patients may present with respiratory symptoms such as exertional dyspnoea, dry cough, chest pain, or rarely haemoptysis.6 The physical examination is usually unrevealing at presentation. As the disease progresses, respiratory insufficiency may develop and be associated with cyanosis, clubbing, and evidence of pulmonary hypertension and cor pulmonale.7 However, pulmonary function remains normal or slightly impaired for a prolonged period after the diagnosis.

No other pulmonary disease has a radiographic pattern as characteristic and diagnostic as that of pulmonary alveolar microlithiasis.8 The chest radiograph shows sharply defined nodules, predominantly basal and less than 1 mm in diameter. In gross disease, as in our case, the radiographic opacity is so great that the cardiac borders are completely obscured and the heart shadow may “vanish”. Other radiological findings include presence of bullae at the lung apices, a zone of hyperlucency between lung parenchyma and the ribs (black pleura sign), and pleural calcification.9

Figure 3  Microphotograph showing calcific material (calcispherites) within an alveolus and its wall (haematoxylin and eosin stain × 550).
Although the aetiology of this condition is not known, familial occurrence in approximately half of the cases point towards either genetic or environmental factors in the pathogenesis of this condition. A history of occupational or environmental exposure to inorganic dust is usually lacking in these cases. The microliths typically consist of calcium and phosphorus. However, these patients do not have any systemic disturbances of calcium metabolism. It is possible that the disease is a manifestation of abnormal calcium metabolism localised only or predominantly to the lungs.

**Final diagnosis**

Pulmonary alveolar microlithiasis.

**References**


**Anaemia: an unusual cause**

**Q1: Describe the features on the barium follow through (see p 303)**

The barium meal follow through reveals a 4 cm x 1 cm sausage shaped pedunculated polyp in the distal ileum, which is lying 40–50 cm proximal to the ileocaecal valve.

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

The differential diagnosis includes lipoma, inflammatory fibroid polyp, other pedunculated polyps such as those associated with Peutz-Jeghers syndrome and familial adenomatosis polyps syndrome, and an inverted Meckel’s diverticulum.

**Q3: What is the diagnosis?**

This patient underwent an exploratory laparotomy, which revealed a Meckel’s diverticulum. A segment of ileum was resected and an end-end anastomosis was performed. The specimen was cut open to reveal an inverted Meckel’s diverticulum.

The histology of the specimen revealed Meckel’s diverticulum with evidence of past ulceration with pyloric gland metaplasia and Paneth cell hyperplasia.

**Discussion**

Most sources of gastrointestinal blood loss can be diagnosed with upper or lower gastrointestinal endoscopy, but 5% of gastrointestinal bleeding episodes are occult and caused by a variety of lesions within the jejunum or ileum. We present a rare case of chronic gastrointestinal bleeding caused by an inverted Meckel’s diverticulum.

Meckel’s diverticulum is the most common congenital abnormality of the small intestine, seen in 0.3%–3% of the population at necropsy. It results from the persistence of the omphalomesenteric (vitelline) duct, which is an embryonic connection between the midgut and the umbilical cord. It arises from the antimesenteric border of the ileum, and contains all layers of the intestinal wall, has its own mesentery, and derives its blood supply from a terminal branch of the superior mesenteric artery.

Painless, often haemodynamically significant, but usually not life threatening, lower intestinal bleeding is a common presentation. Bleeding is more common in children and occurs at a mean age of 5 years. Haemorrhage typically results from ulceration within the diverticulum or adjacent intestinal mucosa as a consequence of acid secretion from ectopic gastric mucosa. There is a debate about the prevalence of Meckel’s diverticulum in males versus females, however complications occur more frequently in males.

Intestinal obstruction due to Meckel’s diverticulum is more common in older patients and can be caused by intussusception, volvulus, herniation, or entrapment of bowel through a defect in the diverticular mesentery. Symptoms mimicking acute appendicitis can occur as a result of Meckel’s diverticulitis, H pylori infection of the ectopic gastric mucosa, or a foreign body in the diverticular lumen.

On barium examination an inverted Meckel’s diverticulum is seen as a solitary elongated club shaped mass (with or without intussusception) in the distal ileum. If intussusception occurs, ultrasound demonstrates a target sign with alternating layers of different attenuation with a low attenuation centre. It is important to be aware of the radiological features of inverted Meckel’s diverticulum, as surgical removal of these lesions is warranted in most cases.

The appearances of an inverted Meckel’s diverticulum on barium examination are shown. It is important to recognise the abnormality so that resection can be performed to avoid the serious complications of intussusception and bleeding.

**Final diagnosis**

Meckel’s diverticulum.

**References**


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Postgrad Med J 2002 78: 305
doi: 10.1136/pmj.78.919.305

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