another case, a 69-year-old woman presented with right-sided abdominal pain and hepatomegaly—a laparotomy was performed before the diagnosis of GCA was made [9]. There are few reports of liver biopsy in GCA. These are mostly normal, or show non-specific changes including cholestasis, steatosis, perisinusoidal lipocyte hyperplasia and hepatocellular necrosis [10].

Our case demonstrates that GCA can present in unusual ways. Presentation with gastrointestinal symptoms can lead to diagnostic delay, unnecessary investigations and occasionally surgery. Timely treatment is the most important factor in preventing serious complications, including blindness, aortic aneurysm and dissection, myocardial infarction and cerebral vascular accident. Furthermore, since GCA is a condition of the elderly, its prevalence in an ageing population is expected to increase. We therefore recommend that GCA should be considered in a setting of fever, gastrointestinal symptoms and raised ESR in those >50 years.

**Rheumatology key message**

- GCA can present with gastrointestinal symptoms in the absence of other localizing symptoms.

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**References**


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**A rare case of chondrocalcinosis in the left sterno-clavicular joint**

Sir, We report about the case of a 68-year-old woman admitted to our department for the first time in January 2006. Since April 2005 she complained of a severely painful, cold swelling in the whole left foot. The patient also reported a history of hepatitis C-related sicca syndrome, regularly followed-up by hepatologists and without indications to anti-viral treatments. When asked about causative traumas, the patient reported a previous attack of arthritis in the left knee, coincidental with the swelling of the left foot and unrelated with any trauma, which spontaneously resolved within 10 days.

Among the investigations carried out at our centre, the complete blood count and inflammatory markers were normal, as well as blood levels of uric acid, alkaline phosphatase, PTH, calcium, phosphate, 25-OH vitamin D. The electrophoresis of serum proteins confirmed a polyclonal hypergammaglobulinaemia (19 g/l) previously noticed at the time of the first investigations carried out because of the hepatitis C infection. The 24-h urinalysis did not yield altered levels of electrolytes (particularly, calcium and phosphate) or pyridinium cross-links.

The lumbar bone densitometry revealed osteoporosis (BMD 0.885 c/cm2; T-score −2.63; measure site from L2 to L4 vertebrae). Conventional X-ray imaging showed patchy osteoporosis of the left foot and chondrocalcinosis in the knees bilaterally. The anterior radionuclide bone scan revealed hot spots in the left tarsus and calcaneum. We diagnosed a reflex sympathetic dystrophy of the left foot associated with idiopathic chondrocalcinosis, post-menopausal osteoporosis and hepatitis C infection. The patient therefore received a course of i.v. clodronic acid followed by standard doses of oral
amino-bisphosphonate plus vitamin D and calcium supplementation as maintenance therapy for osteoporosis. Within a month the painful swelling of the left foot disappeared completely.

In April 2010 the patient suddenly complained of tender swelling at the anterior left side of the base of the neck, without fever. On examination, we found tenderness and swelling of the left sterno-clavicular joint associated with normal body temperature. The anterior radionuclide bone scan showed a hot spot on the same anatomical site, consistent with arthritis. Compared with the values found in 2006, the bone densitometry (lumbar along with femoral sites) revealed increased levels of BMD.

The patient received a single IA injection of triamcinolone acetonide, 40mg. Pain and joint swelling markedly improved, with almost complete recovery of the articular function. Two months later, symptoms relapsed. Physical examination, once again, confirmed a painful swelling over the left upper angle of the sternum, suggestive of joint effusion in the sterno-clavicular joint. The CT scan (Fig. 1) revealed chondrocalcinosis of sterno-clavicular joints bilaterally.

Due to its uncommon occurrence, the real nature of sterno-clavicular arthritis is often hard to establish. In daily rheumatological practice, frequent causes of sterno-clavicular inflammation are sero-negative SpAs. Among them, SAPHO syndrome shows relevant predilection for those peculiar joints. Altered inflammatory markers along with fever are indicative of septic arthritis, whereas elderly onset should suggest degenerative or metabolic diseases such as chondrocalcinosis. In summary, this patient displayed pseudogout attacks in the left sterno-clavicular joint due to an uncommon localization of calcium pyrophosphate dihydrate disease [1].

**Rheumatology key message**
- Advanced imaging techniques like CT scan may be the key for diagnosis of a pseudogout attack.

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**Fig. 1** CT scan of cranial sternum. On the left side, joint-space narrowing, sub-chondral bone sclerosis, slight IA calcifications (▶) and erosions were present. The right joint showed intra-cartilaginous calcifications (→).

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**Calcinosis cutis associated with primary Sjögren’s syndrome: strong expression of osteonectin and matrix Gla protein**

SIR, Calcinosis cutis is characterized by the deposition of calcium and phosphate salts in the skin. Calcinosis cutis is classified into four main groups: dystrophic, metastatic, idiopathic and iatrogenic. Although dystrophic calcification is often associated with autoimmune diseases, such as scleroderma [1] and JDM [2], its occurrence in patients with primary SS is quite rare [3]. Although the pathogenesis underlying dystrophic calcification in those diseases remains unclear, several lines of evidence have indicated that the disordered expression of promoter and inhibitor of calcification in the lesional skin and muscle may contribute to the calcification in SSc and JDM [1, 2]. Osteonectin, a calcium-binding glycoprotein, is an important promoter of calcification [4], while matrix Gla protein (MGP) is a major vitamin K-dependent inhibitor of calcification in the soft connective tissue [5]. Here, we present an intriguing case of calcinosis cutis associated with primary SS, with an immunohistochemical study on the expression of osteonectin and MGP in the lesional skin.