Synovial Chondromatosis Associated with Polyarteritis Nodosa

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Abstract
We report the case of a 40-year-old man with known polyarteritis nodosa who presented with bilateral hip pain, left knee pain and right ankle pain. The hip and ankle pain was caused by avascular necrosis, the knee pain by a combination of osteoarthritis and synovial chondromatosis.

We could find no previously documented link in the literature between Polyarteritis Nodosa, avascular necrosis or synovial chondromatosis. We postulate that there could be a link between the polyarteritis nodosa and the synovial chondromatosis. An autoimmune process that causes the vasculitis which underlies the effects of polyarteritis nodosa may also have caused the synovial chondromatosis.

Introduction
Polyarteritis Nodosa (PAN) is an inflammatory disease of vessels resulting in thrombosis and aneurismal dilatation. It can present with a polyarthropathy [6]. Avascular necrosis of the hips and talus has been documented as a result of steroid treatment [5] which is used in the treatment of PAN. Synovial chondromatosis is a metaplasia of synovium to cartilage [3]. There is no previous documented link between PAN, avascular necrosis or synovial
chondromatosis. We present the case of a gentleman with PAN who presents with avascular necrosis of both hips and of one talus and synovial chondromatosis of the left knee.

**Case Report**

A 40-year old man was referred to our clinic with a long-standing history of progressively worsening bilateral hip pain which had been keeping him awake at night for the preceding two months and left sided knee pain, both were severely limiting his daily activity. Physical examination demonstrated markedly decreased range of motion in both hips and the left knee. X-rays of both hips demonstrated features of advanced avascular necrosis, the left knee demonstrated findings consistent with fairly advanced osteoarthritis.

Of note in his past medical history was an eight year history of PAN, he was found to have multiple bilateral micro aneurysms of his kidneys, superior mesenteric and coeliac vasculature. This was initially treated with large dose steroids. He was then placed on cyclophosphomide for a year and then switched to methotrexate which he was taking along with low dose prednisolone (7.5mg) on presentation. The PAN was deemed to be under good control by rheumatological review just prior to presentation.

Arthroscopy of the left knee was performed a week after the initial consultation, it demonstrated tears of the lateral and medial menisci which were excised and quite severe degenerative change throughout the knee.

A bilateral total hip replacement was performed five months later, the femoral heads were sent for histological examination and were reported as showing features of osteoarthritis and of avascular necrosis. The patient recovered well from this operation.

Nine months following the initial arthroscopy of the left knee the patient returned with worsening of his symptoms and a fixed flexion deformity. A further arthroscopy was performed which was markedly different to the previous, the patient had developed synovial
chondromatosis (Fig 1) which was confirmed histologically, on top of the previous arthritis. Due to persistent pain the patient opted to undergo a left total knee replacement. Further specimens from the joint confirmed synovial chondromatosis. The patient again recovered very well following this operation.

![Figure 1: Findings at second knee arthroscopy: synovial chondromatosis.](image)

Two years following presentation the patient represented with pain and swelling of the right ankle of three months duration. He had a marked reduced range of movement in all directions. X-ray suggested a diagnosis of avascular necrosis. In April an arthroscopy was therefore performed which demonstrated synovial hypertrophy and a loose body arising from the medial side of the talar dome overlying an area of necrosis.

**Discussion**

Polyarteritis nodosa is a systemic inflammatory disease of small and medium sized arteries characterized by fibrinoid necrosis and pleomorphic cellular infiltration. This disrupts the normal architecture of the vessel wall resulting in thrombosis or aneurismal dilatation. Key
clinical features are; skin lesions, peripheral neuropathy, renal sediment abnormalities and hypertension, abdominal pain and constitutional features (fever, chills, fatigue, weight loss, and malaise). A non-deforming polyarthritis involving the larger joints of the lower extremity may occur in up to 20% of cases [6].

Little is known about the aetiology of synovial chondromatosis. It is a benign metaplasia of the synovial membrane resulting in the formation of multiple cartilaginous loose bodies within a joint [3]. This rare condition is self-limiting and non-aggressive. It usually presents in the knee but can also occur in the hip, shoulder and elbow. It presents in the 3rd to 5th decade as gradual onset of monoarticular pain and stiffness [1]. The condition can be classified as primary or secondary. Primary chondromatosis has been postulated to be caused by trauma, infection or by cartilage shed into the joint being taken up by the synovium [2]. Secondary synovial chondromatosis may present after long standing osteoarthritis.

Avascular necrosis (AVN) is caused by an alteration to the blood supply of the involved bone leading to cellular anoxia and death of bone cells [2]. Aetiologies of AVN include trauma, steroids, alcohol, pancreatitis, haemoglobinopathies, dysbarism and Gaucher’s disease [4]. It is postulated that steroids cause AVN due to vascular occlusion caused by fat emboli. Steroids can cause a fatty liver and hyperlipidaemia, conditions ripe for emboli [7].

In an extensive literature search we could find no previously documented link with PAN and synovial chondromatosis. Obviously there is a link with PAN and avascular necrosis since steroids used in the treatment of PAN are known to be a significant cause of avascular necrosis however we could find no literature on direct causation.

This case highlights the possibility of a link between PAN and synovial chondromatosis. The same autoimmune process that caused the vasculitis of PAN in this
patient may have caused the synovial chondromatosis. It is therefore quite possible that there may be an autoimmune component to synovial chondromatosis.

References


