system. The aim of the present study was to assess the frequency of causes of minor discrepancy between bone densities of hip and spine.

Methods In this study, from all patients referred to bone marrow densitometry centre of Loghman from September 2008 to May 2009 in order to BMD assessment due to several causes and underwent spine and hip BMD. 171 cases with minor discrepancies were enrolled and causes of discrepancies were determined.

Results The mean age of patients was 92.69 ± 13.13 years. 87.78% of patients were female. The causes of minor discrepancy were respectively Osteoarthritis (45.03%), Osteoporosis (28.65%), Aortic Calcification, Osteoarthritis Deformans, Syndesmophytes and DISH each with 2.92%. These causes in female gender were Osteoarthritis (48.67%), Osteoporosis (27.33%), Aortic Calcification and Syndesmophytes with 3.33%; and in male they were Osteoarthritis (38.1%), Osteoporosis and DISH each with 19.05%.

Conclusions The minor discrepancy in T-score is a common finding which can result diagnostic problems in densitometry analysis. The results of the present study show that the most common causes of minor discrepancy were Osteoarthritis, Osteoporosis, Aortic Calcification, Osteoarthritis Deformans, Syndesmophytes and DISH.

A6.14 mTOR DIRECTED MESENCHYMAL TISSUE RESPONSE TO INFLAMMATION IN ARTHRITIS

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Background Accumulating evidence supports the concept that fibroblast-like synoviocytes (FLS) actively participate in the destructive, inflammatory process of rheumatoid synovitis. Thus, FLS frame a synovial microenvironment that augments and perpetuates synovial inflammation. Moreover, FLS, together with macrophages form an aggressive mass of cells ("pannus"), which invades and destroys the articular cartilage. The mechanistic target of rapamycin (mTOR) is best known for coupling energy and nutrient abundance to the execution of essential cellular processes, including cell growth and cell survival. More recent data indicate that mTOR directs the cellular response to inflammatory stimuli in cells of the immune system. It remains elusive, however, whether or not this also applies to mesenchymal cells, such as FLS in the context of rheumatoid synovitis.

Materials and Methods In order to assess mTOR activity by immunohistochemistry (IHC) as well as western blotting (WB), phosphospecific antibodies against mTOR (IHC) and mTOR substrates, including 4E-BP (IHC), AKT (WB), S6K1 (WB), and S6 (IHC)

were used. To determine the functional significance of mTOR activity in FLS, Torin-1, a well defined, specific inhibitor of mTOR, was used. To establish a role for mTOR in the mesenchymal, inflammatory tissue response, we used a previously described simplified 3-D model of the synovial tissue. IL-6 and IL-8 levels in the supernatants of 3-D cultures were measured by ELISA.

Results mTOR, 4E-BP and S6 were found to be phosphorylated in RA synovial tissues. These activated phospho-proteins were preferentially expressed in FLS, most prominently in the hyperplastic synovial lining layer. In-vitro, TNF stimulation of FLS resulted in the phosphorylation of AKT and S6K1, indicating that TNF activates the mTOR pathway in FLS. Stimulation of the 3D cultures with TNF resulted in hyperplasia of the lining layer at the surface of the spheres. Strikingly, treatment with Torin-1, prevented TNF induced lining layer hyperplasia. Unexpectedly, the combined treatment of 3-D cultures with TNF and Torin-1 resulted in increased production of IL-6 as well as IL-8 when compared to cultures that were solely exposed to TNF.

Conclusions These studies provide insight into the regulatory circuits that determine the synovial mesenchymal tissue response to inflammation and suggest a multifaceted role for mTOR in arthritis.

A6.15 REFRACTORY CHRONIC ERYTHEMA NODOSUM AND TREATMENT WITH ANTI TNF

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Introduction Erythema nodosum septal panniculitis without vasculitis, characterised by acute episodes of inflammatory and painful subcutaneous nodules affecting, in most cases, the lower extremities. Remission of lesions occurs within 1–6 weeks without scarring or residual atrophy. There are, however, some cases which become chronic or reoccur. In 50% of cases we find an underlying cause. Treatment of acute outbreak involves rest and NSAIDs. The chronic or recurrent cases are treated with oral potassium iodide, corticosteroids, colchicine, hydroxychloroquine or immunosuppressive agents.

Objectives Description of the cases of refractory chronic erythema nodosum and review of the literature.

Methods Selection of patients with refractory chronic erythema nodosum undergoing treatment with anti TNF in the Rheumatology unit from 2000 to 2010. Literature search using PubMed with keywords erythema nodosum and Adalimumab, Etanercept, Infliximab.

Results See table 1.

Conclusions In our sample, all cases have responded favourably to treatment with anti TNF. No adverse events were observed, except the occurrence of cutaneous psoriasis in one patients after

Abstract A6.15 Table 1

Patient	1	2	3	4
Sex	Female	Female	Male	Female
Age	38	32	40	43
Diagnosis	Behçet's disease	Behçet's disease	Sarcoidosis	Psoriatic arthritis
Pre-treatment	Prednisone 30 mg/24 h Azathioprine 100 mg/24 h NAID maximum dosage	Prednisone 30 mg/24 h Azathioprine 100 mg/24 h	Prednisone 30 mg/24 h Azathioprine 100 mg/24 h NAID maximum dosage	Prednisone 30 mg/24 h Azathioprine 100 mg/24 h NAID maximum dosage
Anti TNF	Infliximab 300 mg every 8 weeks Etanercept 50 mg weekly	Infliximab 300 mg every 8 weeks	Infliximab 375 mg every 8 weeks	Infliximab 300 mg every 8 weeks
Evolution	Psoriasis Outbreak with infliximab. Improvement of erythema nodosum with both anti TNF treatments a month after start of treatment	Principio del formulario Improvement a month after start of treatment with resolution in the second dose	Asymptomatic after the first infusion	Resolution of lesions after the 4th infusion of infliximab