Methods Each patient completed a multidimensional health assessment questionnaire (MDHAQ) at each visit to assess functional disability, pain and drugs taken. Drugs taken, changes in scores for functional disability pain, and possible corticosteroid toxicities were analysed using descriptive statistics.

Results Among the 102 consecutive patients, 96 (94%) were taking low dose prednisone over a median of 6.1 consecutive years. All but 6 patients were also taking disease-modifying antirheumatic drugs, including 86% taking methotrexate. The mean starting dose of prednisone in 74 patients whose prednisone was begun at this rheumatology clinic was 4.3 mg (median dose 5 mg/day), compared to 13.1 mg/day (median dose 10 mg/day) in 22 patients whose prednisone was begun at other clinical sites. At the most recent visit, 53 patients (52% of all patients and 55% of corticosteroid users) were taking < 5 mg/day; 48 of these patients had their prednisone begun at this clinic. By contrast, 4 patients were taking >10 mg/day, all of whom had prednisone begun at another site. Median functional disability scores were 0.5 (range 0–3), and 3.4 for pain (range 0–10); scores for functional disability and pain tended to be higher in patients who took higher doses of prednisone, and the highest scores were seen in the 4 patients who took >10 mg/day. Compared to baseline 0.1–18 years earlier, 40% of patients had MDHAQ functional disability scores indicating improvement by >0.25 units, 39% were unchanged by <0.25 units, and 21% had progression of functional disability by 0.25 units, results considerably different from reports published in the 1980s. In analyses of possible corticosteroid toxicities, 32% of patients had hypertension, 4% had diabetes mellitus, and 20% had cataracts, percentages that are similar to those found in published reports of patients with RA.

Conclusion Long-term low-dose prednisone with a starting and maintenance dose of <5 mg/day appears to be a safe therapy, in contrast to high-dose prednisone, with long-term efficacy and low toxicities. Further studies are needed lead to more accurate characterise of the risks and benefits of long-term low dose corticosteroid therapy in RA.

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THU0209 FOLLOW UP OBSERVATIONS OF THE PATIENTS WITH RHEUMATOID ARTHRITIS (RA) AND REACTIVE AMYLOIDOSIS

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Background Secondary amyloidosis is a serious complication of rheumatoid arthritis and may result in significant morbidity and early death. The knowledge on standard and effective management of patients with secondary amyloidosis is limited.

Objectives The objective of the study is follow up observation of RA patients with established reactive amyloidosis concerning the clinical aspects of the disease, acute phase reactants and the response to cytostatic treatment.

Methods 43 patients with rheumatoid arthritis and histologically confirmed secondary amyloidosis were followed for 3 to 6

(mean 4,6) years. The patients were treated with cyclophosphamide or methotrexate combined with small doses of prednisone. Clinical symptoms, laboratory tests and general outcome were analysed after 3 to 6 years of development of amyloidosis.

Results The time from the diagnosis of RA to the development of amyloidosis was 4 to 16 years, mean 8,7 years. In all patients advanced radiological changes were observed (radiological Steinbrocker stage III or IV).

The most common symptoms of amyloidosis were: proteinuria-27(62,8%), nephrotic syndrom -12(27,9%), diarrhoea-4 (9,3%). In most of the patients the course of RA was very severe since the onset and high activity parameters were observed despite treatment.

Amyloidosis was histologically confirmed by subcutaneous fat tissue biopsy in 38(88,4%) cases. In 8 patients amyloid deposits were found in other organs (kidney, stomach, salivary gland).

After the treatment with cyclophosphamide or methotrexate we observed decrease of some activity parameters (ESR, CRP) and decrease of proteinuria level. The treatment was well tolerated and we didn't observe serious side effects. In the observed group 7(16,3%) patients died after 3 to 6 years of the diagnosis of amyloidosis. The most common cause of death were uremia (4/7) and infections (2/7).

Conclusion Aggressive treatment with cytostatics influences the survival of patients with reactive amyloidosis in RA.

The time of survival in 83,7% of the observed patients was longer than 4,6 years.

The outcome of patients with RA and reactive amyloidosis is better when the diagnosis of amyloidosis is confirmed in the early stage of the disease.

THU0210 CLINICAL LONGTERM UPDATEABLE EVALUATION (CLUE) FORMS FOR CLINICAL FEATURES AND MEDICATIONS USED IN THE MANAGEMENT OF PATIENTS WITH RHEUMATOID ARTHRITIS

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Background Rheumatologists collect similar data in the care of patients with rheumatoid arthritis (RA) concerning clinical features, such as extra-articular disease, comorbidities, and classification criteria, as well as a history of therapies used in patient management. However, these data generally are collected in non-standard formats. Therefore, much clinical data are not easily accessible to the clinician in routine long-term care of individual patients, or to workers in clinical research concerning cohorts of patients at one centre or multiple centres. Advances in laboratory and clinical science are enhanced when data are collected in a standard format.

Objectives To develop two clinical long term updateable evaluation (CLUE) standard forms for use in the management of patients with RA, one concerning clinical features, and the second concerning medication history, which can be maintained indefinitely at the front of the medical record.

Methods Two one page CLUE forms were developed for use in usual clinical care. The RA clinical features CLUE form includes extra-articular disease, comorbidities, classification criteria for RA, possible family history of RA, records of joint surgeries/dates and radiographs. The medication review CLUE form includes all DMARDs and most current NSAIDs. Each form is designed to

be updated easily at each visit, without a need to collect the data again.

Results The CLUE forms have been used to characterise 102 consecutive patients with RA in routine clinical care. Medications in current use included prednisone (median dose 4 mg per day) by 94% of patients, methotrexate (median dose 15 mg per week) by 86%, hydroxychloroquine by 28%, leflunomide by 10%, cyclosporine by 8%, etanercept by 8%, infliximab by 8%, auranofin by 3%, gold injections by 3%, azathioprine by 2%, penicillamine by 1%, and sulfasalazine by 1% of patients. Extra-articular disease included malaise reported by 72% of patients, dry mouth by 46%, carpal tunnel syndrome 37% dry eyes by 35%, and scleritis by 9% of patients at any time in the course of RA. Comorbidities included chronic back pain in 36% of patients, hypertension in 31%, peptic ulcer in 27%, esophageal symptoms in 25%, cataract in 20%, thyroid disease in 11%, cancer in 10%, diabetes mellitus in 4%, renal disease in 3% of patients. Sixty-three percent of patients had smoked cigarettes and 28% were current smokers.

Conclusion Two simple clinical longterm updateable evaluation (CLUE) forms are easily completed in usual clinical care to characterise patients with RA. These forms are complemented by a patient questionnaire and joint count form, to provide simple, standardised measures of RA for improved clinical care, clinical research, and clinical outcomes.

THU0211 ADULT ONSET STILL'S DISEASE: A RARE ASSOCIATION WITH AUTOIMMUNE THYROIDITIS

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Background Adult onset Still's disease (AOSD) was first described by Bywaters in 1971. Subsequently more than 300 cases have been reported. AOSD is a systemic inflammatory disease with high spiking fever, a typical rash and arthritis or arthralgia as major clinical features. The disease is accompanied by a neutrophilic leukocytosis. Other common features are sore throat, intensive myalgias, lymphadenopathy, hepatosplenomegaly, pericarditis and pleuritis.

A 48 years old white woman recently presented to our observation. She had been in her usual state of good health until 7 years before, when she developed fever, arthralgias, sore throat and euthyroid non-toxic goitre. Thyroid antibodies (Thyreoglobulin, microsomal, peroxidase) were elevated. She was diagnosed to have autoimmune thyroiditis and was treated with l-thyroxine and prednisone 25 mg daily. She has done well until eight months ago when she developed fever, arthralgias, sore throat and myalgias. One month later she was admitted to her local hospital with spiking fever of 39,0°C, pleuritic chest pain, dyspnea, hepatomegaly, palpitations, fatigue and weight loss. Chest radiograph revealed bilateral pleural effusion and echocardiogram demonstrated pericardial effusion. A thoracentesis removed 30 ml of pleuric fluid, wich had the characteristic of an exudate. Ultrasound abdominal investigation revealed mild hepstomegaly. Once with the fever spike (40°C) a mildly pruritic maculopapular evanescent rash located on the extremities was observed and interpreted as drug induced. Laboratory examination revealed haemoglobin 10,2 g/dl, Erythrocyte mean corpuscular volume 71,2 fl, platelet count 628'000/mmc, White blood cell count 30'640/mmc with 96% neutrophils, ESR 76 mm/h, CRP 32,7

mg/dl, Rheumatoid Factor 121,0 UI/ml. ANA, ENA, ANCA, ACA and anti-DNA were normal. There was no serological evidence for active o recent infection and blood coltures were negative. Malignancies were excluded.

There was no response to empiric antibiotics. Therapy with NSAIDs failed. Treatment with 3,5[4,0 mg/Kg daily Cyclosporine for three months was ineffective. Prednisone 50 mg daily resulted in resolution of fever, systemic manifestations, sore throat and normalisation of her laboratory tests, including Rheumatoid Factor.

Rheumatic diseases are, more frequently than suspected, associated with autoimmune thyroiditis. To our knowledge this is the second reported case of association between AOSD and autoimmune thyroiditis. Test for Rheumatoid Factor, ANA and other autoantibodies are generally negative in AOSD. Some criteria for the diagnosis of AOSD consider a positive Rheumatoid Factor as an exclusion criteria. But it may be positive in a transient manner in up to 4 to 6% of those patients.

Objectives

Methods

Results

Conclusion

THU0212 IMBALANCE BETWEEN SYNOVIAL IL-1 ACTIVITY/IL-1RA PRODUCTION AND DISEASE COURSE IN RHEUMATOID ARTHRITIS

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Background

Objectives To evaluate in a cross-sectional study the relationship between the clinical/radiological disease outcome, the presence or absence of RA-associated HLA-DRB1* alleles and circulating levels of IL-1ra in patients with rheumatoid arthritis.

Methods 120 patients, stratified in sub-groups defined by disease duration, RF status and HLA-DRB1* typing were analysed for an association between disease course and circulating levels of IL-1ra. HLA-DRB1* allele typing was performed by standard methods, IL-1ra and other cytokines (IL-6, IL-8, sTNFR55/75) were measured using commercially available ELISA reagents. Patient's characteristics assessed included inflammatory disease activity by clinical and standard laboratory measures. Radiographic disease progression was determined by the Steinbrocker method.

Results There was inverse correlation between circulating IL-1ra levels and radiographic outcome in the patient population with a disease duration < 5 years (Pearson correlation coefficient: -0.23; p < 0.03). In most of the sub-populations analysed IL-1ra correlated strongly with the levels of IL-6, IL-8, and sTNFR 55/sTNFR75s. In Subgroups of patients defined by HLA-DRB1* typing, patients (n = 30) with clinical and radiographic evidence for a more severe disease course were positive for a double-dose of HLA-DRB1* 0404/0401 or positive for a combination of a HLA-DRB1* 040x allele with the HLA-DRB1* 0101 allele. These patients presented with significantly (p = 0.01) lower IL-1ra levels (mean: 384 pg/ml) than patients (n = 37) bearing none the RA-linked HLA-DRB1* alleles (mean: 1197 pg/ml). IL-1ra levels in healthy controls (n = 22; mean: 337 pg/ml) were comparable with IL-1ra deficient patients bearing a double gene dose. When only one of the suspected HLA-DRB1* alleles was