bital myositis. *Curr Opin Rheumatol* 1997; 9: 504-12.
7. SHAMBAL S, LINDNER A,ZIERZ S: Successful treatment of orbital myositis with intravenous immunoglobulins. *Muscle Nerve* 1998; 21: 1359-60.
8. SANCHEZ-ROMAN I, VARELA-AGUILAR JM, BRAVO-FERRER J: Idiopathic orbital myositis: treatment with cyclosporin. *Ann Rheum Dis* 1993; 52: 84-5.
9. SHAH SS, LOWDER CY, SCHMITT MA,WILKE WS, KOSMORSKI GS, MEISLER DM: Low-dose methotrexate therapy for ocular inflammatory disease. *Ophthalmology* 1992; 99: 1419-23.
10. PANFILIO CB, HERNANDEZ-COSSIO O, HERNANDEZ-FUSTES OJ: Orbital myositis and rheumatoid arthritis: Case report. *Arq Neuropsiquiatr* 2000; 58: 174-7.
11. AJITSARIA R, DALE R,FERGUSON V, MAYOU S, CAVANAGH N: Psoriasis, psoriatic arthropathy and relapsing orbital myositis. *Clin Exp Dermatol* 2001; 26: 274-5.
12. SEIDENBERG KB, LEIB ML: Orbital myositis with Lyme disease. *Am J Ophthalmol* 1990; 109: 13-6.

High prevalence of thyroid autoantibodies in newly diagnosed rheumatoid arthritis patients

Sirs,

Organ specific autoimmune diseases may be linked to non-organ specific autoimmune conditions. In particular the present study had the aim to determine the prevalence of antithyroid antibodies and their correlation to RA clinical and serological data in a group of newly diagnosed patients with RA from the province of Naples,Southern Italy, an area with frequent cases of iodine deficiency (1).

During a six-month period we recruited consecutively 28 outpatients with a first diagnosis of RA according to ACR revised criteria. All patients were women, with a mean age of 45.3 ± 14.5 years. Twenty-seven patients were seropositive for the rheumatoid factor (RF). A family history of thyroid diseases (TD) was recorded in 5 patients. Our data were compared with the data from 30 sera obtained from normal age-matched females usual blood donors in the hospital, all coming from the same geographic area, without evidence of past or present TD. For all patients informed written consent was obtained. Thyroid hormones, antiperoxidase (anti-TPO) and antithyroglobulin (anti-Tg) antibodies were analyzed. The number of affected joints, CRP, RF, ANA, ENA C3 and C4 complement fractions were determined.

We calculated the prevalence of antithyroid antibodies among cases and controls. Rate ratios (RR) with 95% confidence intervals (95% CI) were used when comparing rates.

In a second step we evaluated the correlation of antithyroid antibodies with the patient's serological and clinical variables. Regression coefficients were calculated by multiple logistic regression analysis using anti-tg positivity (0= no; 1= yes), anti-TPO positivity (0= no; 1= yes) and positivity of any of the two antibodies (0= no; 1= yes) as dependent variables in three groups of different models, respectively. Age was used as covariate in the models. Each biochemical and clinical variable was then used as independent variable in a separate model to calculate regression coefficients. Positivity for ANA, ENA, RF was found respectively in 1 (3.5%), 3 (10.7%), and 27 (96.4%) patients. FT3 and FT4 values were in the normal range and were not significantly different from the controls (FT3: 4.2 ± 1.6 vs 3.9 ± 1.1pg/ml; FT4:12.4 ± 2.9 vs 11.9± 2.7 pg/ml). Six patients with RA presented subclinical hypothyroidism (21%) with increased TSH values (4.8 ± 0.7 mIU/ml), which candidates them to l-thyroxine treatment. Normal TSH levels were detected in the remaining patients (mean:1.7 ± 0.9 mIU/ml) and in controls (mean: 1.5 ± 0.7 mIU/ml) .

The prevalence of both anti-TPO and anti-Tg in patients with RA was significantly higher than in controls: anti-TPO= 32% vs 4% (RR=8.02; 95%CI=5.25-10.8); anti-Tg = 25% vs 8% (RR=3.12; 95%CI= 1.9-4.35). Even when considering antithyroid antibodies as a whole the prevalence of positive subjects was significantly higher among patients (43%), as compared to controls (10%) (RR= 4.3; 95%CI=3.01-5.58). No significant correlation was found between antithyroid antibodies positivity and biochemical and clinical variables.

Data indicate a pathogenetic link between RA and ATD and relevant data have already been reviewed regarding the thyroid hormonal pattern in RA patients (2,3). Uneven geographical distribution has been described for both RA and ATD. RA has been reported to occur more frequently among populations in northern Europe and among native Americans (4, 5). Antithyroid antibodies are serological markers for thyroditis, representing also a risk factor for clinical hypothyroidism (6). Six of the RA patients showed thyroid function test results indicative of subclinical hypothyroidism.

The prevalence of antithyroid autoantibodies in the female RA patients examined is higher than reported by others (7-10). In literature, an increased prevalence of anti-Tg and anti-TPO, varying from 3.4 to 12% and 4.4 to 33%, respectively, has been shown in RA patients when compared to a normal population. However, in the present study all patients were newly diagnosed RA, therefore never treated for the disease. This fact can explain the high prevalence rate of antithyroid antibodies, expression of the initial autoimmune disorder still unaffected by drugs. Female sex and high proportion of RF positive cases may be factors which contribute to the reported result.

In conclusion, results from the present study outline the high prevalence of ATD in newly diagnosed, unselected RA patients. Data support the usefulness of ATD screening in RA patient's management. Still unknown remain the predictive factors of this association.

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References

1. LUPOLI G, RUSSO D, FITTIPALDI MR *et al.* Evaluation of goiter endemia by ultrasound in schoolchildren in Val Sarmento (Southern Italy). *J Endocrinol Invest* 1999; 22: 503-7.
2. VALENTINO R, SAVASTANO S, TOMMASELLI AP *et al.* Hormonal pattern in women affected by rheumatoid arthritis. *J Endocrinol Invest* 1993; 16: 619-24.
3. CUTOLO M, ACCARDO S: Sex hormones, HLA and rheumatoid arthritis. *Clin Exp Rheumatol* 1991; 9: 641-4.
4. SILMAN AJ, HOCBERG MC. Rheumatoid arthritis. In: Oxford Medical Publications. *Epidemiology of the Rheumatic Diseases*. Oxford, *Oxford University Press*; 1993; 7-48.
5. DEL PUENTE A, KNOWLER WC, PETTITT DJ, BENNETT PH: High incidence and prevalence of rheumatoid arthritis in PIMA indians. *Am J Epidemiol* 1989; 129: 1170-8.
6. VANDERPUMP MJP, TUNBRIDGE WMG, FRENCH JM *et al*.: The incidence of thyroid disordes in the comunity:A twenty-year follow-up of the Whickham Survey. *Clin Endocrinol* 1995; 43: 55-68.
7. ANDONOPOULOS AP, SIAMBI V, MAKRI M, CHRISTOFIDOU M, MARKOU C, VAGENAKIS AG: Thyroid function and immune profile in rheumatoid arthritis. A controlled study. *Clinical Rheumatol* 1996; 15: 599-603.
8. MAGNUS JH, BIRKETVEDT T, HAGA HJ: A prospective evaluation of antithyroid antibody prevalence in 100 patients with rheumatoid arthritis. *Scand J Rheumatol* 1995; 24: 180-2.
9. BIANCHI G, MARCHESINI G, ZOLI M *et al*.: Thyroid involvement in chronic inflammatory rheumatological disorders. *Clin Rheumatol* 1994; 13: 479-84.
10. THOMAS DJB, YOUNG A, GORSUCH AN, BOTTAZZO GF, CUDWORTH AG: Evidence for an association between rheumatoid arthritis and autoimmune exocrine disease. *Ann Rheum Dis* 1983; 42: 297-300.