Methods: Eleven public rheumatology centers from all of the 5 regions of Brazil enrolled ~100 consecutive RA patients each (1987 ARA or 2010 ACR-EULAR). This cohort is being followed prospectively for 1 year, with systematic data collection at time 0, 6±1 months and 12±1 months, and registration of all other visits during this 1-year period. Data collection began in 08–2015, using a single online electronic medical record, and included demographic, socioeconomic, clinical, lab, radiographic and therapeutic characteristics, along with functional status, quality of life and adherence to treatment information.
Results: 1125 patients were enrolled (Table), ~90% were female, with a mean age of 56 years and median disease duration of 13 years. Median BMI was 27 kg/m^2, with 64% of the patients classified as overweight or obese. The interval between symptoms and diagnosis varied from 1 to 457 months (median 12 months). Almost half of the patients were on glucocorticoids, 96% on DMARDs, with 36% on biologics. Only 7% were seronegative for both rheumatoid factor and ACPA. Median HAQ-DI was 0.875 and median DAS28-ESR was 3.5, with 58.6% of patients presenting moderate or high disease activity.

Table 1. Patients baseline characteristics

	N	
Age, years*	1125	56.7 (22.1–88.8)
Female	1125	89.5%
Current or former smoker	1125	39.6%
BMI, kg/m^2*	1055	26.6 (15.8–56.2)
Disease duration, years*	1124	12.7 (0.7–56.7)
Rheumatoid factor+	1105	78.7%
ACPA+	479	76.8%
Erosive disease	1105	55.2%
Extra-articular manifestation ≥1	1118	23.3%
Drugs in use:		%
– Glucocorticoids	1125	47.0
– NSAIDs	1125	9.0
– Synthetic DMARDs	1125	90.8
– Methotrexate	1125	66.5
– Biologic DMARDs	1125	36.1
Erythrocyte sedimentation rate (ESR), mm/1st hour*	933	21 (1–140)
C-reactive protein, mg/dL*	954	0.7 (0–76.1)
DAS28-ESR*	932	3.5 (0.3–8.2)
HAQ-DI*	1121	0.875 (0–3)

*Results in median (range).

Conclusions: The delay in diagnosis may explain the high percentage of patients with moderate or high disease activity and erosive disease. The low level of physical dysfunction observed in this established, predominantly seropositive RA population may be explained by the large proportion of patients on glucocorticoid and biologic therapy. Our findings suggest that, despite current treatment concepts being well known and accepted by Brazilian rheumatologists, there is still a gap in early diagnosis and management of RA.
Disclosure of Interest: None declared
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AB0321 RELATIONSHIP OF CAROTID FEMORAL PULSE WAVE VELOCITY WITH AGE AND TIME OF EVOLUTION IN PATIENTS WITH RHEUMATOID ARTHRITIS

C. Ramos[1], G. Alanis[1], D. Cardona[2], S. Totsuka[1], E. Cardona[1], F. Pérez[1], A. Coldivar[1], J. Gómez[1], M. Vázquez del Mercado[1]. *[1]Physiology, University of Guadalajara; [2]Physiology, University of Gudalajara, Guadalajara, Mexico*

Background: Early vascular aging occurring in Rheumatoid Arthritis (RA) may be a consequence of chronic inflammation. The measurement of Carotid-femoral pulse wave velocity (cfPWV) is the gold standard to evaluate arterial stiffness. Vascular aging is a result of a change in the biomechanical properties of the vascular wall. This process can be accelerated by the accumulated damage of high mechanical stress (high blood pressure), chronic inflammation and comorbidities such as smoking, Diabetes Mellitus and dyslipidemia[1]
Objectives: The aim of this study was to evaluate variations in the Carotid-Femoral Pulse Wave Velocity (cfPWV) and it's association to age and time of disease evolution in patients with rheumatoid arthritis.
Methods: RA patients were matched for age and sex with healthy controls. Subjects with a history of smoking, cardiovascular disease, hypertension, diabetes mellitus, cancer, liver disease, thyroid disease and kidney disease were excluded. The cfPWV was calculated using the Pulse Pen ® (Diatechne, Italy) device.
Results: We included 76 women with RA and 28 healthy women, mean age (44.3±10.92 vs. 43.0±16.26, P=0.654). cfPWV demonstrated good correlation with age (r=0.459, P<0.01), disease evolution time (r=0.311, P=0.008), triglycerides (r=0.289, P=0.03), total cholesterol (r=0.421, P<0.01) and atherogenic index (r=0.320, P=0.02). No association with disease activity was found. cfPWV was higher in those patients with RA >10 years evolution compared to patients with <10 years of disease evolution and to controls (P<0.05).
Conclusions: A significant association between cfPWV was seen in patients with RA, and was also correlated to age and to a disease evolution >10 years long without finding a significant association with increased disease activity.
References:
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AB0322 THE ROLE OF HOMA-IR AND HOMA-ISLET INDICES IN DIFFERENT CARBOHYDRATE METABOLISM DISORDERS DURING GLUCOCORTICOID THERAPY

G. Nurullina. *Kazan Medical State University, KAZAN, Russian Federation*

Background: Diabetogenic effect limits the use of glucocorticoids (GC), especially in patients with diabetes risk factors. At present HOMA index is widely used for the evaluation of insulin resistance (IR) and β-cell function. HOMA-IR index increase is an indirect measure of IR progression and cardiovascular risk elevation.
Objectives: To evaluate the role of modified HOMA-IR and HOMA-islet indices in different carbohydrate metabolism disorders (CMD) during oral (OGCT) and PULSE (GCPT) glucocorticoid therapy (GCT).
Methods: A prospective study including 118 patients with systemic lupus erythematosus (SLE) (n=63) and systemic vasculitis (SV) (n=55) was performed. Seventy one patients received GCPT (i.v. infusion of 10–15 mg/kg of prednisolone with 250 ml of normal saline per day, for 3 consecutive days (1 course of 3 sessions); course dose was 1800–3000 mg; 47 patients received oral GC 15–30 mg/day. HOMA-IR index and β-cell function (HOMA-islet index) were calculated as follows: Homa-IR =1.5 + fasting blood glucose (mmol/l) x fasting C-peptide level (pmol/l) /2800. HOMA-islet =0.27 x fasting C-peptide level (pmol/l)/(fasting blood glucose (mmol/l) – 3,5).
Results: GCPT was associated with less CMD compared to OGCT. Impaired fasting glucose (IFG) was observed in 7 (9.9%) and 9 (19.1%), impaired glucose tolerance (IGT) – in 9 (12.7%) and 14 (29.8%) and diabetes mellitus (DM) – in 9 (12.7%) and 13 (27.7%) patients in GCPT and OGCT groups, respectively. There was a significant decrease of HOMA-islet during glycemic peak in DM patients from 13.96 to 6.17 after GCPT (p<0.05), compared to insignificant changes in other groups. Decrease of HOMA-islet index reflects a disturbance of overall functional activity of β-cells in DM patients. After a course of GCPT HOMA-islet index was significantly lower in DM patients compared to patients with no CMD (11.8 vs. 15.4), as GCPT is associated with a decrease of β-cell function which does not return to baseline as a consequence of β-cell reserve depletion. No rapid decrease of β–cell function was observed in OGCT group, instead there was a compensatory increase, which was insufficient to maintain normoglycia because of high insulin resistance. C-peptide, HOMA-IR and HOMA-islet levels in OGCT patients demonstrated the same trend as in GCPT patients. Significant differences were observed in patients with IGT and DM before and after oral glucose tolerance test (OGTT) on C-peptide (1042 pmol/l vs. 1978 pmol/l in IGT; 1306 pmol/l vs. 2286 pmol/l in DM) and HOMA-IR (4.53 vs. 9.81 in IGT; 5.6 vs. 11.27 in DM patients), whereas in patients without CMD and in patients with IFG, C-peptide before OGTT was 489 pmol/l vs. 743 pmol/l, after - 1295 pmol/l vs. 1488 pmol/l, HOMA-IR – 2.59 vs. 2.88 before OGTT and 2.88 vs. 5.85 after the test in the absence of CMD and in IFG patients, respectively. A significant decrease of β–cell function was observed in DM patients, reflected by a decrease of HOMA-islet index after OGTT compared to baseline (147 vs. 78.4).
Conclusions: Evaluation of blood glucose level, which was normal in all included patients, and isolated C-peptide evaluation are insufficient for the evaluation of carbohydrate metabolism before GCPT and long-term OGCT. The evaluation of modified HOMAA-IR and HOMA-islet indices before the start of intensive GC treatment and during OGCT may improve early detection of risk groups for serious CMDs – IGT and DM.
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AB0323 CARBOHYDRATE METABOLISM AND GLUCOCORTICOID THERAPY IN PATIENTS WITH SYSTEMIC INFLAMMATORY DISEASES

G. Nurullina. *Kazan Medical State University, Kazan, Russian Federation*

Background: Glucocorticoid therapy (GCT) is one of the risk factors of carbohydrate metabolism disorders (CMD) in patents with systemic inflammatory diseases. CMD development is a concern not only with long-term therapy, but also during intensive short-term GC administration, which can lead to different CMDs, including impaired glucose tolerance (IGT) and diabetes mellitus (DM).
Objectives: to evaluate the prevalence of CMD after long-term and intensive GC treatment (GCT) in patients with systemic lupus erythematosus (SLE), systemic vasculitis (SV) and chronic glomerulonephritis (CGN).
Methods: Ninety eight patients with systemic inflammatory diseases were included (SLE - 53, SV – 35), among them 68 received GC pulse-therapy (GCPT) (1 series of 3 sessions), and 30 – oral GCT (OGCT). All patients underwent standard clinical and laboratory evaluation, oral glucose tolerance test (OGTT), evaluation of C-peptide, HOMA-IR and HOMA-islet indices.
Results: CMDs developed less often in patients receiving GCPT compared to long-term OGCT (p=0.035). In patients receiving OGCT the most prevalent CMDs were IGT and DM – in 9 (30.0%) and 8 (26.7%) patients respectively, which