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AB0676

BODY COMPOSITION IN MYOSITIS PATIENTS AND THE ASSOCIATION WITH DISEASE SPECIFIC FEATURES, PHYSICAL ACTIVITY AND PLASMA LEVELS OF INFLAMMATORY CYTOKINES

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Background: Skeletal muscle, pulmonary, and articular involvement in idiopathic inflammatory myopathies (IIM) limit the mobility/self-sufficiency of patients, and can have a negative impact on body composition.

Objectives: To assess body composition and physical activity of IIM patients and healthy controls (HC) and the association with selected inflammatory cytokines in IIM.

Methods: 54 patients with IIM (45 females; mean age 57.7; disease duration 5.8 years; PM, (22)/DM, (25)/IMNM, (7)) and 54 age-/sex-matched HC (45 females, mean age 57.7) without rheumatic/tumor diseases were included. PM/DM patients fulfilled Bohan/Peter criteria for PM/DM. We assessed body composition (densitometry: iDXA Lunar, bioelectric impedance: BIA2000-M), physical activity (Human Activity Profile, HAP questionnaire), disease activity (MITAX and MYOACT activity score), muscle involvement (manual muscle test, MMT-8 and functional index, FI2) and plasma levels of 27 cytokines (commercial multiplex ELISA kit, Bio-Rad Laboratories). Data are presented as mean±SD.

Results: Compared to HC, patients with IIM had a trend towards significantly increased body fat% (BF%) as assessed by iDXA (39.9±7.1 vs. 42.4±7.1 %, p=0.077), but significantly decreased lean body mass (LBM) as assessed both by iDXA (45.6±8.1 vs. 40.6±7.2 kg, p=0.001) and BIA (52.6±8.8 vs. 48.7±9.0 kg, p=0.023), and increased extracellular mass/body cell mass (ECM/BCM) ratio (1.06±0.15 vs. 1.44±0.42, p<0.001), reflecting deteriorated nutritional status and worse muscle predispositions for physical activity. Compared to HC, IIM patients had significantly lower bone mineral density (BMD: 1.2±0.1 vs. 1.1±0.1 g/cm², p<0.001). Disease duration negatively correlated with BMD (r=-0.392, p=0.004) and LBM-BIA (r=-0.272, p=0.047). Disease activity assessed by both MITAX and MYOACT positively correlated with LBM-BIA (MITAX: r=0.294, p=0.031; MYOACT: r=0.335, p=0.013) and LBM-DXA (MITAX: r=0.341, p=0.012; MYOACT: r=0.368, p=0.007), similarly as with basal metabolic rate (BMR; MITAX: r=0.336, p=0.014; MYOACT: r=0.351, p=0.010), and fat free mass (FFM; MITAX: r=0.338, p=0.014; MYOACT: r=0.356, p=0.009). CRP was positively associated with BF% assessed both by DEXA (r=0.276, p=0.035) and BIA (r=0.306, p=0.025). Higher BF%-DEXA was associated with worse physical endurance (FI2: r=-0.311, p=0.026) and worse ability to perform physical activity (HAP: r=-0.292, p=0.032). MMT-8 score negatively correlated with ECM/BCM ratio (r=-0.385, p=0.006). Plasma levels of IL-1ra positively correlated with body mass index (BMI, r=0.359, p=0.009), BF% (r=0.363, p=0.009), and visceral fat (VF, r=0.409, p=0.003). Plasma levels of MCP-1 positively correlated with ECM/BCM ratio (r=0.387, p=0.005). Increased plasma levels of IL-10 were associated with decreased LBM (r=-0.473, p=0.014).

Conclusion: Compared to healthy age-/sex-matched individuals we found significant negative changes in body composition of our IIM patients, which are associated with their disease activity and duration, inflammatory status, skeletal muscle involvement, and physical activity. Plasma levels of several inflammatory cytokines were associated with alterations of body composition in IIM patients and highlight the potential role of inflammatory status on impaired body composition during the course of IIM.

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AB0677

TREATMENT OF IDIOPATHIC INFLAMMATORY MYOPATHIES – A SINGLE CENTRE EXPERIENCE

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Background: There is no consensus or recommendations for the treatment of idiopathic inflammatory myopathies (IIM).

Objectives: We explored how we treated incipient IIM patients in our secondary/tertiary rheumatology centre.

Methods: We retrospectively included cases with IIM diagnosed between January 2010 and June 2018, who were followed at least 6 months. The remission inducing treatment and the treatment at the last follow-up were recorded.

Results: During the 102-month period we identified 102 IIM cases (71.6% female, median (IQR) age 62.7 (52.2–72.1) years): 27.5% dermatomyositis, 22.5% anti-synthetase syndrome, 14.7% myositis in an overlap syndrome, 13.7% immune mediated necrotizing myopathy, 11.5% polymyositis, 6.9% cancer associated myositis, 2.9% inclusion body myositis (IBM), 1% unspecified myositis. 94 (92.2%) patients received glucocorticoids (GC), 18/94 (17.6%) as monotherapy. The remaining 81 (79.4%) were treated with additional immunomodulatory agents (Table 1), most commonly methotrexate. In addition to therapy presented in Table 1, 13 (12.7%) also received human immunoglobulins (IVIG), one (1%) received plasma exchange therapy.

Of the 8 (7.8%) cases who were not treated with GC 3 received methotrexate, 1 rituximab, and 4 no additional treatment (1 paraneoplastic polymyositis received chemotherapy, 2 IBM, 1 mild polymyositis in elderly, multimorbid patient).

In the first 6 months following diagnosis, 11 patients died (3 due to infection, 2 as a consequence of IIM, 1 due to haemorrhagic shock, 5 of unknown causes), and 5 were lost to follow-up. The remaining 86 patients were followed for a median (IQR) 37.7 (20.2–62.6) months. At last follow-up 49 (57%) patients were still receiving GC, 8 (9.3%) in monotherapy. 9 (10.5%) cases were off GC and other maintenance treatment. Maintenance treatment at last follow-up is presented in Table 2. Treatment was changed in 13 (15.1%) patients (due to progression of pulmonary involvement in 3, active myositis in 4, pulmonary involvement progression and active myositis in 1 and due to intolerance to medication in 6 cases). In addition to therapy presented in Table 2, 2 (2.3%) patients received IVIG (1 concurrently with rituximab and mycophenolic acid due to recalcitrant anti-synthetase syndrome and 1 due to IBM complicated with dysphagia).

Table 1. Induction treatment of idiopathic inflammatory myopathy

Type of treatment	N (%)
Cyclophosphamide; median dose (range)	23 (22.5); 6 (2–12) g
Methotrexate	43 (42.2)
monotherapy	30 (69.8)
+ cyclosporine A	8 (18.6)
+ cyclosporine A and rituximab	4 (9.3)
+ chloroquine	1 (2.3)
Rituximab monotherapy	4 (3.9)
Azathioprine	3 (2.9)
Mycophenolic acid	3 (2.9)
Cyclosporine A monotherapy	1 (1.0)