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The Assessment of Autoimmune Disease-Related Fatigue Syndrome in a Dysmetabolic Obesity Elderly Patient with Dermatomyosistis

Magro VM[1]*, Coppola C[2], Boccalone E[1], Caturano M[3] and Verrusio W[4]

[1]*Department of Internal Medicine and Geriatry, University of Campania "Luigi Vanvitelli", Naples, Italy*

[2]*Department of Intensive Rehabilitation, Casa di Cura Alma Mater S.p.A – Camaldoli Hospital, Naples, Italy*

[3]*Department of Internal Medicine, AORN San Giovanni di Dio e Ruggi d'Aragona, Salerno, Italy*

[4]*Department of Cardiovascular, Respiratory, Nephrological, Anesthesiological and Geriatric Sciences, Sapienza University of Rome, Rome, Italy*

Abstract

Fatigue is a term used to describe an overall feeling of tiredness or lack of energy. It is a common symptom of many medical conditions that range in severity from mild to serious. Many medical conditions can cause fatigue. Examples include anemia, arthritis, cancer, metabolic disorders and/or autoimmune disorders all conditions that can be found alone but also associated in the elderly subject. We describe the clinical case of a patient in whom this condition we discovered was associated with the presence of autoimmune disorder. We tried to be learned from this clinical case report the implications, as well as clinics, but also of behavior and suspicion, in an elderly patient.

Keywords: Fatigue; Autoimmune disorders; Elderly patient; Alterations of carbohydrate metabolism; Polymyositis/dermatomyositis

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Correspondence:
Valerio Massimo Magro, Department of Internal Medicine and Geriatry, University of Campania "Luigi Vanvitelli", Piazza L. Miraglia 2, Naples, Italy.
Tel: *(+39) 3492224922*
E-mail: *valerio_magro@hotmail.com*

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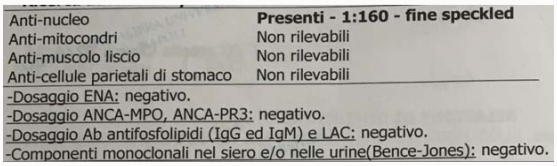
Introduction

Fatigue is one of the most common problems that we can found in a patient with a diagnosis of autoimmune disease, single or multiple [1-4]. Since this symptom is strongly non-specific and it can often complain even in elderly subjects and/or with dysmetabolism [5-8], there is always a great deal of attention to these subjects in order to avoid the possible under diagnosis of some times unknowable and potentially serious or secondary pathologies to other serious prognostic frameworks [9,10]. We exemplify a case we have taken over and treated as outpatients setting, without any need for hospitalization, and subsequently routed in the diagnostic-therapeutic care pathway (PDTA).

Case Presentation

A 67-year-old patient, already suffering from obesity (Body Mass Index BMI 31 kg/m^2) and metabolic syndrome, came to our observation of generalized asthenia in the four limbs associated with the presence of a muscular dystonia. The patient was in therapy with monacolin k and valsartan/hydrochlorothiazide 320/25 mg. Vital signs in the limits except pain (number rating scale NRS 8/10). The multidimensional evaluation element is normal expept a decrease of the humoral tone (Geriatric Depression Scale GDS 5/15). Hematochemical analysis showed total protein 5.5 g/dl (normal values 6.6-8.7), C-HDL 29 mg/dl (>35), AST-GOT 155 U/l (5-40), ALT-GPT (60-190), alkaline phosphatase 33 U/l (40-129), LDH 800 U/l (240-480), ferritin 1380 ng/ml (30-400) transferrin 150 mg/dl (191-337), OH vitamin D 11.8 ng/ml (>30). Normal hemocromocytometric examination and renal function. Presence of a reduced carbohydrate tolerance. For the suspect of an acute inflammatory myopathy, we suspended K monacoline and short-term clinical and laboratory follow-up was performed without the regression of the symptomatology (Visual Analogic Scale VAS 6-7/10, despite antalgic therapy), CPK values ranging from 1200 and 4100 U/l in three consecutive drawings, with the appearance of a diffuse erythema predominantly localized to the four limbs (greater in the upper limbs than in the lower limbs) with smaller but present extension to the back and throne, associated with intense itching. Further laboratory investigations were performed (anti-TPO 920 UI/ml, vn<16; positive ANAs, 1:160 title, fine speackled pattern; negative hepatic markers) (Figure 1) and a EKG (presence of a sinus rhythm with diffuse repolarization



Anti-nucleo	**Presenti - 1:160 - fine speckled**
Anti-mitocondri	Non rilevabili
Anti-muscolo liscio	Non rilevabili
Anti-cellule parietali di stomaco	Non rilevabili

-Dosaggio ENA: negativo.
-Dosaggio ANCA-MPO, ANCA-PR3: negativo.
-Dosaggio Ab antifosfolipidi (IgG ed IgM) e LAC: negativo.
-Componenti monoclonali nel siero e/o nelle urine(Bence-Jones): negativo.

Figure 1: Immunological Investigations. Antibody assays: anti-smooth muscle, anti-mitochondria and anti-gastric parietal antibodies were not detectable in the patient's serum. Anti-nucleus antibodies were instead present with a 1-160 titer and spekled pattern. The dosage of the ENA antibodies, ANCA-MPO, PR3-ANCA, antiphospholipid antibodies and lupus anticoagulant was negative. There were no monoclonal components in the serum.

α-Fetoproteina	2.4 ng/mL (<10)
CA 19-9	14.0 U/mL (<37)
CA 125	6.5 U/mL (<35)
Ferritina	**780.5** U/mL (<35)
PSA	0.278 ng/mL (<5)
Proteina S100	0.24 µg/L (<0.2)
CA 50	17.6 U/mL (<19)
CA 72-4	2.3 U/mL (<19)

Figure 2: Tumor markers. All neoplastic markers (alpha fetoprotein; Carbohydrate Antigens CA 19.9, 125, 50 and 72, 4; ferritin; prostate specific antigen PSA; S100 protein) were within the normal limits. The only altered parameter remained serum ferritin.

abnormalities), at the echocardiogram presence of mild eccentric ventricular hypertrophy of the left ventricle with a conserved EF, first grade diastolic dysfunction and mild mitral-tricuspid insufficiency, PAP 35 mm Hg), spirometry (forced vital capacity FVC 81%), abdominal ultrasonography (bright liver ecostructure with increased liver size), thyroid ultrasonography (unhomogeneous ecosystems and increased thyroid size as thyroiditis) and a chest radiography (within the limits).

At this point, worsening the picture, we performed both a neurological (ultrasonography ENMG, highlighting a canalicular neuropathy of the left medial nerve of the carp) and enzymatic evaluation (aldolase 40 U/l). On this basis, we did not consider it appropriate to deepen the diagnosis of the diagnosis with a biopsy, so after having discarded a paraneoplastic etiology (total body CT and negative oncomarkers) (Figure 2) [11,12] and a polygladular autoimmunity syndrome (type 3) [13], we started a therapy with methylprednisolone 1 g, spironolactone 100 mg, metformin 500 mg, acetylsalicylic acid 100 mg, rabeprazole 20 mg, gradually associating sub-maximal physiokines therapy to 4 limbs, step formation, active and passive spinning of the spine. After two weeks, the inflammatory markers marked drop in, as well as transaminase and enzymes, decreased pain (VAS 3-4/10), good pace of the march, regression to pruriginal edema until the disappearance, without tingling to a diabetes. Then, at the end of the first diagnostic and therapeutic path, we sent the patient to the rheumatologist's care for continuation of follow-up.

Discussion

Dermatomyositis is a chronic inflammatory disease affecting various apparatus including muscle but also skin (erythematous manifestations) with an autoimmunity etiology and frequent association [14,15] with other autoimmune (myasthenia gravis, Hashimoto thyroid) and more complex diseases (3 type PGA) and a good therapeutic response to immunosuppressive drugs, as in this case [16]. Given the preference for female sex and the administration of a HMG-CoA-reductase inhibitor (monacoline), the suspected dermatomyositis was delayed with the onset of the symptoms, although the laboratory and instrumental framework later led us to a diagnosis that it satisfies the criteria of rheumatic scientific societies (EULAR, ACR, Bohan and Peter criteria) [17,18]. Then, the diagnostic route was completed in order to exclude concomitance of other treatable diseases both the co-presence of a more or less metastatic neoplasm, associated with dermatomyositis, of non-infrequent delay, when this condition appears in more advanced age [19-22].

Conclusions

A moderate level of asthenia related to autoimmune disease occurs in many patients with diagnosis of dermatomyositis and in addition elderly. The reported case seems to us to be interesting and deserving of signaling, not only because it draws attention to this disease, which is not frequent in the elderly population, but also for the particular mode of clinical presentation with sordid debut but with clear symptomatic recurrence in one patient who, by his other chronological, anthropometric and pathological features associated with it, could constitute an important and potential source of misdiagnosis.

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