RESEARCH ARTICLE

THE DIAGNOSIS OF DILATED CARDIOMYOPATHY DURING EXTENDED PSORIASIS: CASE REPORT

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Abstract

A 32-year-old patient has a bi-ventricular insufficiency that had developed over the past two months revealed by cardiac symptomatology. Echocardiography revealed bi-ventricular dilated cardiomyopathy. He had skin lesions for three years that were clinically and histologically identified as psoriasis. The association of cardiomyopathy with psoriasis is rare and intriguing and the link between these two entities on a common inflammatory background is discussed.

Introduction:

Clinical Case:
A 32-year-old patient with no cardiovascular risk factors, known to have poorly monitored skin psoriasis, without any other specific personal or family pathological history has been admitted to the Department of Cardiology and Vascular Diseases for stage III dyspnea of NYHA associated with an edematous syndrome of gradual installation in two months, the patient has reported the progression of skin lesions over the last three years by thrust and transiently responding to topical treatment, the details of treatment are in this case not known by the patient. The clinical examination objectified a normal state of consciousness, a heart rate at 85 bpm/min and respiratory at 27 cycles/min, a correct blood pressure at 125/75 mmHg and a temperature at 37.7°C, with swelling of the face and swelling of the lower limbs; the skin examination notes the presence of scaly erythematous plaques covered with silvery scales spread over the trunk, abdomen, back and scalp respecting mucous membranes (Figure 1).

Figure 1: Erythematous scaly plaques of psoriasis
The clinical examination found congestive signs of bilateral basithorac crackling rail type with a murmur of mitral insufficiency, turgescence of the jugular veins, ascites of medium abundance, hepatogalia with hepatojugular reflux, hydrocele and lower extremity edema. The patient was hospitalized with initial conditioning in the cardiac intensive care unit; oxygen therapy, half-seatedposition, one treatment was administered: Furosemide 60 mg/8h, Ramipril 2.5 mg/jr and Spironolactone 25 mg/jr. The electrocardiogram objectified a regular sinus rhythm with a heart rate of 85bpm and a left axial deviation without other notable abnormalities. Chest x-ray revealed cardiomegaly with a cardiothoracic index of 0.8. The results of transthoracicechocardiography after stabilization supported dilated cardiomyopathy with severe biventricular dysfunction; showing the biological balance objected to the blood count a hemoglobin level at 12.8g/dl, white blood cells at 9640 e/mm3, platelets at 330000/mm3. The blood urea was at 0.25 g/dl and the creatinine at 5mg/l, a level of ASAT: 18 IU/l, ALAT: 12 IU/l, PAL: 22 IU/l, GGT: 49 IU/l, a high CRP at 97.6 mg/l. Blood electrolytes were normal as well as thyroid function with TSHus dilated left ventricle without hypertrophy, site of overall hypokinesia, with severe systolic dysfunction (ejection fraction estimated at 12%), moderate mitral insufficiency, low filling pressures, presence of a pericardial effusion blade and pulmonary hypertension at 53 mmhg with a dilated inferior vena cava (Figure 2).

Figure 2: Echocardiography showing dilated cardiomyopathy with severe biventricular dysfunction

levels at 3.11 and T4L at 14.1. A skin biopsy was recommended showing a characteristic image of psoriasis made of hyperkeratosis with parakeratosis and acanthosis of the epidermis with at the dermal level the existence of an infiltrate with T-lymphocytesCD4 and large capillary development with elongated skin papillae (Figure 3).

Figure 3: Anatomopathological section of skin biopsy performed in our patient
A coronary angiography was performed without abnormalities. The evolution was marked by the regression of dyspnoea with at the same time, a therapeutic increase in the dosage of Ramipril at 5mg/d and a reduction of that of furosemide at 40mg/d orally with the introduction of a cardioselective beta-blocker treatment at low dose. Treatment with topical steroids was introduced for psoriatic lesions, so the diagnosis

Discussion:-
Psoriasis is a chronic inflammatory dermatological condition with a poorly understood etiology. There is a current genetic component involved; 50% of cases have a family history and the link to HLA Cw6, HLA DR7 has been well established.

Skin lesions are characterized by infiltration by activated T-cells. The cytokines developed by these activated T-cells in cooperation with growth factors stimulate and develop keratinocytic hyperproliferation. As a result, clinical trials are currently underway to slow or suppress the characteristic inflammation of psoriasis targeting TNF-alpha and other proinflammatory cytokines. Recently, "characteristic inflammation" in cardiomyopathy has been unravelled and a distinct entity has been described: inflammatory cardiomyopathy defined as myocarditis coupled with cardiac dysfunction. This is in addition to the five main forms of cardiomyopathy: dilated, restrictive, hypertrophic, right ventricular and non-classified cardiomyopathy[2]. A central role of autoimmunity in dilated cardiomyopathy is supported by the presence of specific autoantibodies of organs, inflammatory and pro-inflammatory infiltrates, and cytokines. Autoantibodies and patterns of abnormal cytokines were first-degree controls of asymptomatic left ventricular dilatation and the fact that family cases of dilated cardiomyopathy are observed in 20-30% of cases suggest the involvement of disturbed humoral activity and early cellular immunity in the development of the disease. Similar humoral activity and cellular immune dysregulation are observed in autoimmune diseases, these autoimmune diseases such as rheumatoid arthritis, thyroiditis, psoriasis and asthma are common and in the first degree in subjects with dilated cardiomyopathy. Thus, it is assumed that the same multiple genes in combination with environmental factors lead to many different autoimmune diseases, including dilated cardiomyopathy [2]. The genetic implication of this condition is thus important to recognize because of the number of cases described in the same family in 20 to 30% of cases. For example, screening of first-degree family members of affected patients is currently common practice and has identified the presence of cardiac autoantibodies and abnormal cytokine profiles. This suggests that genetic factors play an important role in the pathogenesis of dilated cardiomyopathy, either as a contributor to at the exit was a biventricular dilated cardiomyopathy associated with extensive psoriasis.

Environmental susceptibility, or as a determinant of changes that contribute to the phenotypic expression of the disease [3]. To our knowledge, it is still unclear whether the presence of genetic risk factors is shared by other autoimmune diseases. A study of 52 cases reported increased prevalence of autoimmune diseases in first-degree parents of patients with dilated cardiomyopathy and a higher incidence in these same first-degree family members than in the control group [4]. Significant differences were observed for thyroid diseases, diabetes mellitus and psoriasis. The high prevalence of cardiovascular risk factors and cardiovascular disease has been demonstrated in several studies in patients with psoriasis, particularly in patients with psoriatic arthritis [5, 6]. Psoriatic arthritis is an inflammatory arthritis occurring in 10-30% of patients with psoriasis characterized by chronic inflammation with increased secretion of proinflammatory cytokines that can be considered coupled with other genetic and environmental factors, a trigger for the development of dilated cardiomyopathy [7]. A study of 370 patients with psoriatic conditions highlighted the frequency and characteristics of cardiac lesions in these patients. Myocarditis was diagnosed in 15.9% of patients, pericarditis in 18.2% of cases and valve malformations in 5.7% of cases. Aortitis was detected in 51.3% of patients with advanced disease [8]. Another study evaluating 47 patients with psoriatic arthritis found that these patients had significantly higher levels of cardiovascular risk factors such as high blood pressure, dyslipidemia and a thickened vascular wall especially in coronary arteries compared to healthy controls [9]. It will be relevant to mention a study established in a dermatology department involving 753 patients with psoriasis that 73% of these cases had comorbidity, the most common were high blood pressure, dyslipidemia, diabetes mellitus and other heart conditions [10]. In addition, microalbuminuria was reported as the only abnormality in well-documented renal tests in psoriasis vulgaris. It is also a recognized fact that this microalbuminuria is an independent risk factor for cardiovascular morbidity and mortality.
Endothelin:-

High plasma renal activity are thought to contribute to the high prevalence of high blood pressure and cardiovascular disease in psoriasis. Angiotensin II can also play a contributory role, produced by the angiotensin conversion enzyme (ACE), its action is considered pro-inflammatory endogenous. ACE is present in the uterus, placenta, vascular tissue, heart, brain, adrenal and kidney cortex, leukocytes, alveolar macrophages, peripheral monocytes, neuronal cells, epididymis cells; and can therefore play a role in atherosclerosis, heart failure, stroke, bipolar disorders, schizophrenia, dementia, Alzheimer’s disease, psoriasis, atopic and nonatopic dermatitis, eczema and several diseases as well as acute and chronic inflammatory cancers [12]. Psoriasis is a systemic inflammatory condition that confers increased cardiovascular risk, in addition to the traditional risk factors of cardiovascular disease and various comorbidities such as high blood pressure, diabetes, dyslipidemia and microalbuminuria, which have always been observed in patients with psoriasis [13]. Psoriasis has also been linked to increase the prevalence of metabolic syndrome [14]. In fact, the role of inflammation and the higher prevalence of atherosclerosis and endothelial dysfunction have been demonstrated in patients with psoriatic rheumatism [15]. In addition, the recent description of inflammatory cardiomyopathy suggests both the common underlying pathogenic association of cardiomyopathy and psoriasis in the same subject. Bibliographic research shows that the association of dilated cardiomyopathy and psoriasis in the same individual is not only interesting but also rare. A case of psoriatic arthritis associated with dilated cardiomyopathy has been reported in Japan, but cardiomyopathy has been associated with Takayasu’s disease.

Four other cases followed for psoriasis have developed dilated cardiomyopathy indicating that the link between this two entities is not a coincidence [17]. Dilated cardiomyopathy is a generally serious heart condition with several complications that may be secondary to several conditions but whose primary form is characterised by probably multifactorial autoimmune, genetic, infectious pathogenesis [4;18;19]. Primary dilated cardiomyopathy develops commonly without underlying diseases, but it can also occur after or in combination with a variety of conditions such as idiopathic myocarditis, collagen diseases, metabolic disorders, neuromuscular diseases and consumption of alcohol or narcotics [20]. This condition is one of the leading causes of severe heart failure and the most common indication of heart transplantation with a still high mortality rate [21].

Conclusion:-

Dilated cardiomyopathy in combination with psoriasis is an interesting association and the link to psoriasis may encourage the diagnosis of «inflammatory dilated cardiomyopathy». Chronic inflammation and secretion of proinflammatory cytokines with particular genetic and environmental background can be considered as a potential pathway to dilated cardiomyopathy in psoriasis’s patients. Further study of all these risk factors could lead to a better understanding of the pathogenesis of dilated cardiomyopathy and may lead to further studies that could improve the treatment of this condition not limited to the treatment of heart failure, but leading to a real saving treatment.

Bibliography:-