
Point of view

Celiac disease in idiopathic dilated cardiomyopathy

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A significantly increased prevalence of celiac disease (5.7 vs 0.18% of the general population)¹ has recently been reported² in patients with idiopathic dilated cardiomyopathy (IDCM) by testing serum antiendomysium antibodies. Positive antiendomysium antibody patients received an intestinal endoscopy and biopsy that confirmed a celiac disease to be present.

The identification of a celiac disease in patients with IDCM raises critical questions on the relationship between the two entities. In fact it is known that a reciprocal negative interaction between the heart and small intestine can occur whenever each organ is severely compromised. In particular intestinal absorption can be impaired in patients with IDCM and chronic heart failure, while a celiac disease can limit the absorption of essential nutrients (electrolytes, proteins, amino acids, etc.) leading to structural and functional abnormalities of cardiomyocytes that may determine or exacerbate an IDCM.

In their article Curione et al.² suggest an autoimmune link between celiac disease and IDCM, in other words the existence of an autoimmune process towards antigenic components of both the myocardium and small bowel. However they do not provide any supportive evidence to their claims.

In fact an assessment of cardiac autoantibodies has not been performed. Moreover the bioptical approach to their patients has been limited to the right ventricle, the histologic results are not reported in detail and finally no immunohistochemical or polymerase chain reaction study on the possible presence of viral genomes has been undertaken. Indeed it is known that right ventricular biopsy provides a low sensitivity for

the histologic assessment of myocarditis^{3,4} while left ventricular and mostly biventricular endomyocardial biopsy furnish more reliable results when a comparison with *post-mortem* studies is carried out⁵. It is likely, then, that at least some of the patients who received a diagnosis of IDCM because of the presence of non-specific changes in the hypertrophy-dilated heart might have had a myocarditis.

On the true setting of IDCM, viral infection seems to have an increasing role as in up to 50% of patients with IDCM a viral genome has been identified in frozen endomyocardial tissue by polymerase chain reaction⁶⁻⁸. The assessment of the pathogenetic mechanism (i.e. immuno-mediated or viral-mediated) in IDCM has not got, from the recent past, only a speculative value. Indeed immunosuppressive therapy may provide beneficial results in active lymphocytic myocarditis associated with detection in the serum of cardiac autoantibodies⁹ and giant cell myocarditis¹⁰ in which an immune mechanism is detectable. On the other hand treatment with large doses of gammaglobulins was shown to be effective in patients with myocardial infection by Cytomegalovirus¹¹, Herpes Simplex virus and Parvovirus^{12,13}, while interferon-beta has recently been used with promising results for inflammatory dilated cardiomyopathy caused by adenovirus and enterovirus^{14,15}.

The association of celiac disease with IDCM may have relevant practical implications. In particular an undiagnosed celiac disease can prevent the restoration of intestinal absorption of essential nutrients and cardiovascular drugs. Moreover celiac disease is invariably associated with an in-

creased intestinal permeability¹⁶ that can in turn allow the transluminal passage of potential antigens such as ingested food proteins, endotoxins, bacterial breakdown products and active enzymes, thus favoring myocardial and coronary vascular disorder.

References

1. Catassi C, Ratsch IM, Fabiani E, et al. Coeliac disease in the year 2000: exploring the iceberg. *Lancet* 1994; 343: 200-3.
2. Curione M, Barbato M, De Biase L, Viola F, Lo Russo L, Cardi E. Prevalence of coeliac disease in idiopathic dilated cardiomyopathy. *Lancet* 1999; 354: 222-3.
3. Frustaci A, Bellocchi F, Olsen EG. Results of biventricular endomyocardial biopsy in survivors of cardiac arrest with apparently normal hearts. *Am J Cardiol* 1994; 74: 890-5.
4. Chimenti C, Calabrese F, Thiene G, Pieroni M, Maseri A, Frustaci A. Inflammatory left ventricular microaneurysms as a cause of apparently idiopathic ventricular tachyarrhythmias. *Circulation* 2001; 104: 168-73.
5. Chomette G, Auriol M, Delcourt A, Gamallo-Amat C, Cabrol A, Cabrol C. Endomyocardial biopsy and post-mortem studies in human cardiac transplantation. *Arch Anat Cytol Pathol* 1985; 33: 233-9.
6. Fujioka S, Kitaura Y, Ukimura A, et al. Evaluation of viral infection in the myocardium of patients with idiopathic dilated cardiomyopathy. *J Am Coll Cardiol* 2000; 36: 1920-6.
7. Pankuweit S, Portig I, Eckhardt H, Crombach M, Hufnagel G, Maisch B. Prevalence of viral genome in endomyocardial biopsies from patients with inflammatory heart muscle disease. *Herz* 2000; 25: 221-6.
8. Pauschinger M, Paproth F, Doerner A, et al. Entero- and adenoviral persistence is associated with progression of left ventricular dysfunction in dilated cardiomyopathy. (abstr) *Circulation* 2000; 102 (Suppl II): 976A.
9. Frustaci A, Chimenti C, Maseri A. Global biventricular dysfunction in patients with symptomatic coronary artery disease may be caused by myocarditis. *Circulation* 1999; 99: 1295-9.
10. Frustaci A, Chimenti C, Pieroni M, Gentiloni N. Giant cell myocarditis responding to immunosuppressive therapy. *Chest* 2000; 117: 905-7.
11. Maisch B, Hufnagel G, Pankuweit S. Cytomegalovirus hyperimmunoglobulin treatment in cytomegalovirus myocarditis and its influence on serum titers of cardiotropic viruses. (abstr) *J Am Coll Cardiol* 2001; 37 (Suppl A): 152A.
12. Hufnagel G, Pankuweit S, Richter A, Schonian U, Maisch B. The European Study of Epidemiology and Treatment of Cardiac Inflammatory Diseases (ESETCID). First epidemiological results. *Herz* 2000; 25: 279-85.
13. Kishimoto C, Takamatsu N, Kawamata H, Shinohara H, Ochiai H. Immunoglobulin treatment ameliorates murine myocarditis associated with reduction of neurohumoral activity and improvement of extracellular matrix change. *J Am Coll Cardiol* 2000; 36: 1979-84.
14. Kuehl U, Pauschinger M, Schwimmbeck PL, Schultheiss HP. Interferon- β therapy in patients with idiopathic left ventricular dysfunction and enteroviral persistence. (abstr) *Circulation* 2000; 102 (Suppl II): 2197A.
15. Kuehl U, Pauschinger M, Schwimmbeck PL, Seeberg B, Schultheiss HP. Interferon- β treatment of patients with enterovirus caused left ventricular dysfunction. (abstr) *Circulation* 2000; 102 (Suppl II): 3806A.
16. van Elburg RM, Uil JJ, Mulder CJ, Heymans HS. Intestinal permeability in patients with coeliac disease and relatives of patients with coeliac disease. *Gut* 1993; 34: 354-7.