Case Report

Clinical Cure of Fulminant Myocarditis Associated to *Chlamydophila Pneumoniae* and Parvovirus B19 Superinfection

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ABSTRACT

Fulminant myocarditis is superacute myocarditis, rapidly lethal or curable. The authors report the first case of FM with concomitant *Chlamydophila pneumoniae* and Parvovirus B19 superinfection, proved by positive serology tests. The patient was rapidly asymptomatic.

INTRODUCTION

Fulminant Myocarditis (FM) is a superacute myocarditis related to the Lieberman [1] clinicopathologic description as « Patients with FM become acutely ill after a distinct viral prodrome, have severe cardiovascular compromise, multiple foci of active myocarditis by histologic study and ventricular dysfunction that either resolves spontaneously or results in death ». The authors report the first case of FM with concomitant *Chlamydophila pneumoniae* (CP) and Parvovirus B19 (PVB19) superinfection.

OBSERVATION

A 24 years old black African man is hospitalized on the 19 december 2004 in *Cardiology Department* with a diffuse chest pain, curtly set up and permanent since 18 hours (NYHA IV). No antecedent is found (no cardiovascular risk factors) except a brief febrile period before the chest pain emergence. At clinical examination : temperature at 37,7° C, arterial pressure = 100/70 mm Hg, heart rate = 94/mn, breath rate = 20/mn, attenuated heart sounds. The body mass index is 24. The chest X-ray is normal with cardio-thoracic ratio = 0.47. At echocardiographical examination, the heart is hypokinetic with a low Left Ventricular Ejection Fraction (LVEF) = 50%. The plasma troponin I is very high at day 0 (406 ng/l) and only 8 ng/l at day 2. The treatment during hospitalization is : sodium enoxaparin (Lovenox®), 0.6 ml subcutaneously each 12 hours, Atenolol (Tenormine®) 50 mg/24 hours *per os*, lysine acetylsalicylate (Aspegic®), 250 mg/24 hours, *per os*, buprenorphine (Temgesic®), sublingual way in case of pain. The clinical evolution is rapidly asymptomatic : at day 15 NYHA is I. But echocardiographical data and Sokolow-Lyon index return to normal more slowly (Table). At day 180 the patient is apparently in good health with LVEF at 68%. The following tests are realized at day-0 and day-15 (Table) : antistreptolysins and antistreptodornases, Human Immunodeficiency Virus (HIV) with 2 Elisa methods (Abbott® and Biotest®), Epstein-Barr virus, (Diasorin®), Parvovirus B19, (PVB19, Elisa specific IgG and IgM antibodies, Biotrin®), Cytomegalovirus (CMV, Bio-Merieux®), *Chlamydophila Trachomatis* (CT) and

Chlamydophila pneumoniae (CP, Elisa specific IgG and IgA antibodies, Ani- Labsystems®), *Mycoplasma pneumoniae* (MP, Ani-Labsystems®), Adenovirus (ADV) and a specific Elisa IgA antibody test for enterovirus (Genzyme Virotech GmgH®). The cut-off values for evaluation of positive tests are those indicated by the firm documentation. The plasma PVB19 genome, searched in plasma sample (LightCycler Parvovirus B19®), was negative. The only significant positive results concern PVB19 and *Chlamydophila pneumoniae* (Table). PVB19 : at day-0 positive IgG and IgM antibodies, at day-15 only positive IgG (PVB19 primary infection). *Chlamydophila pneumoniae* : at day-0 and 15, high positive IgG and IgA specific antibodies (evolutive infection).

Table: Clinical and serological data.

	Day 0	Day 2	Day 7 (Exit)	Day 15	Day 90	Day 180
	Diffuse chest					
Clinical symptoms	pain	No	No	No	No	No
NYHA	IV	II	II	I	I	I
Arterial pressure (mmHg)	100/60	100/60			120/70	120/80
Troponine I (ng/l)	406	8				
ECG	sinus rhythm, 94/mn P-R depression	No Q wave				Normal
Sokolow-Lyon index (mm)	32	32	38		39	20
Echocardiography LVEF %	50, hypokinetic		60		65	68
Parvovirus B19 (Elisa)	**IgG = 2,8 IgM = 1,5**			**IgG = 2,3 IgM negative**		
Chlamydia pneumoniae (Elisa)	**IgG = 3,9 IgA = 2,4**			**IgG = 3,8 IgA = 2,1**		

LVEF: Left ventricular ejection fraction; NYHA: New york heart association

DISCUSSION

The diagnosis of FM, despite the absence of endomyocardial biopsy, is convincing. The transient, precocious and large elevation of troponin I, without Q wave in the next hours, is a strong evidence of superacute myocardial suffering without myocardial infarction (no ECG Q wave). Thus, in the literature a few cases of FM are described mimicking acute myocardial infarction [2,3]. Either the precocious death or the rapid and regressive evolution, with a clinical and echocardiographical cure, are specific traits of FM [1]. Among numerous serologic investigations made to identify an infectious agent, 2 Elisa tests were positive in 2 plasma samples, 2 weeks interval : *Chlamydophila pneumoniae* and parvovirus B19. The serologic data for CP are high positive IgG and IgA specific antibody tests. The presence of plasma IgA 2 running weeks is a reliable sign of a CP evolutive infection. Only two cases of FM in relation to CP is reported. One by Hoefer *et al.*, [4], one other by Poelzl *et al.*, in Austria [5]. FM in relation with PVB19 infection is a possible condition, reported in 2001 [6] for the first time and since this date only a few cases were described in literature [2,3]. Thus, PVB19 is a new cardiac infectious agent to consider possibly in the diagnosis of heart diseases, especially myocarditis. The demonstration of 2 synchronous and concomitant infections in FM is exceptionally reported : only one case is pointed out by Rohayem *et al.*, in 2001 [6] : the fatal FM was due to an acute co-infection with PVB19 and human herpes virus 6 (HHV-6). The authors suggest that the HHV-6 induced immunosuppression and enhanced dissemination of PVB19, which led to fatal myocarditis. More recently Poelzl *et al.*, [5] described one case of FM in a 24 years old female patient. Reverse transcription-polymerase chain reaction, realized on endomyocardial biopsy material, revealed co-infection with CP and PVB19 but blood cultures and antibody status were negative. Our observation is the first case of FM in relation to superinfection : PVB19 primary infection (positive plasma specific IgM antibody) in a patient with CP evolutive infection. PVB19 or CP was separately implicated in FM but the

evidence of co-infection (Poelzl *et al.*,) or superinfection (our case) is a new way to explain the clinical aspect of hyperacute myocardial infection. The troponin elevation in our patient is doubtless the consequence of multiple small necrosis foci as Bultmann *et al.*, [2] suggest : PVB19 infection of endothelial cells is sufficient to induce impaired coronary microcirculation with secondary cardiac myocyte necrosis. The presence of an other infection with CP, evolutive, which also strikes endothelial cells, is an interesting way to elucidate the hyperacute evolution of FM, lethal or cure. At last the presence of CP evolutive infection as a possibly cause of lethal disease induces an urgent therapeutic attitude with antibiotics (macrolides).

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