

Clinical case

## Isolated aortic valvular and pulmonary involvement during essential eosinophilia

*Atteinte valvulaire aortique isolée avec lésions pulmonaires au cours d'une hyperéosinophilie essentielle*

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### Abstract

Endomyocardial fibrosis is the most classic and the most known complication of prolonged hypereosinophilic syndrome, whatever the cause. In Burkina Faso, this complication is most frequently encountered in idiopathic form of the syndrome. It commonly involves the apex of the ventricles with possible involvement of atrioventricular valves. The clinical picture is that of restrictive cardiomyopathy with poor prognosis. We report the case of a 22-year-old man with atypical cardiac involvement during idiopathic hypereosinophilic syndrome. Echocardiographic examination showed isolated aortic valve involvement. Left and right ventricular function was preserved. The apex of ventricles was free of lesion. Pulmonary CT-scan showed massive bilateral lung involvement. Treatment consisted of strict control of the eosinophilic process and pulmonary management. The patient suddenly died sudden pulmonary distress one month after first being seen.

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**Keywords:** Hypereosinophilic syndrome; Aortic valve; Prognosis

### Résumé

La fibrose endomyocardique est la complication la plus classique et la plus connue du syndrome d'hyperéosinophilie prolongée, quelle que soit la cause. Au Burkina Faso, cette complication est plus fréquemment rencontrée au cours de la forme idiopathique du syndrome. Il s'agit généralement d'une atteinte de l'apex des ventricules avec la participation éventuelle des valves auriculo-ventriculaires. Le tableau clinique est celui d'une cardiomyopathie restrictive de mauvais pronostic. Nous rapportons le cas d'un homme de 22 ans, avec une atteinte cardiaque atypique au cours d'un syndrome d'hyperéosinophilie idiopathique. L'échocardiographie a montré une atteinte isolée de la valve aortique. La fonction systolique des deux ventricules était conservée. Le sommet de ventricules était libre de lésion. La TDM pulmonaire a montré une atteinte pulmonaire bilatérale massive. Le traitement consistait en un contrôle strict du processus éosinophilique et des symptômes pulmonaires à l'aide d'hydroxyurée, de corticoïdes et d'oxygène. Mis en exeat après amélioration clinique et biologique le patient décède un mois plus tard d'une détresse respiratoire après interruption volontaire de son traitement.

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**Mots clés :** Syndrome hyperéosinophilique ; Valves aortiques ; Pronostic

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## 1. Introduction

Hypereosinophilic syndrome (HES) is a rare multisystemic disease with 40% cardiac involvement and poor prognosis [1]. Idiopathic HES has been defined by Chusid et al. [2] as an unexplained increase of eosinophils greater than 1500 cells/mL, persisting longer than six (06) months. This hypereosinophilia can lead to organ system dysfunction due to cytotoxic injury by eosinophils. Cardiac involvement is the most disastrous complication of this clinical situation [3]. The overall mortality rate is approximately 75% in untreated patients three years after diagnosis [4]. We present the case of a young Burkinabe in whom severe cardiopulmonary lesions had rapidly conducted to death.

## 2. Observation

A 22-year-old man was admitted in August, 2012 in Yalgado Ouedraogo Teaching hospital emergency unit for severe exacerbation of dyspnea and lower limb edema. The history noted that the patient had chest discomfort for at least six months without medical care. There was no history of systemic disease, underlying lungs or bronchial diseases, pulmonary tuberculosis, asthma or parasitosis.

At medical examination, respiratory rate was elevated (26 bpm) even as heart rate (126 bpm), a moderate fever ( $T = 37.9^{\circ}\text{C}$ ) and blood pressure was normal (120/60 mmHg). Moderate lower limb edema was present as hepatomegaly, jugular vein and hepatic jugular reflux.

Cardiopulmonary examination found tachycardia even as significant pulmonary bilateral crackles.

In biology, there was no argument for leukemia or systemic disease; and parasitologic, allergologic, and antineutrophil cytoplasm antibody test results were negative. Blood cultures were negative. ESR and CRP were normal. Aspergillosis serology was negative. Blood count found leucocytosis (WBC = 218,200 cells/mm<sup>3</sup>). The leucocyte formula showed 86% of eosinophils (187,652 cells/mm<sup>3</sup>), 9% of neutrophils (19,638 cells/mm<sup>3</sup>), 1% of basophils (2182 cells/mm<sup>3</sup>), 4% of lymphocytes (8728 cells/mm<sup>3</sup>) and 0% of monocytes. Bone marrow aspiration biopsy found eosinophils excess and signs of dysgranulopoiesis without blasts excess. No cause of this hypereosinophilic syndrome (HES) was found, and then a diagnosis of idiopathic HES was retained. The ECG showed left ventricular hypertrophy. At chest radiograph, there were opacities poorly systematized occupying the lower half of both lung fields. Echocardiography (Figs. 1–5) highlighted a thickening (17 mm) of the right anterior aortic valve with moderate aortic insufficiency but myocardial walls were free of lesion. There was a slight dilation of the cardiac cavities with preserved systolic function of the two ventricles but left ventricular diastolic dysfunction and pulmonary hypertension. The pericardium was normal. CT scan (Fig. 6) demonstrated bilateral lower half pulmonary reticular opacities with superimposed thickening of inter and intralobular interstitium. The treatment was composed of oxygen, corticosteroid, hydroxyurea, and spironolactone. One week later, symptoms disappeared. Lab examination found great improvement of biological signs. In echocardiography, aortic

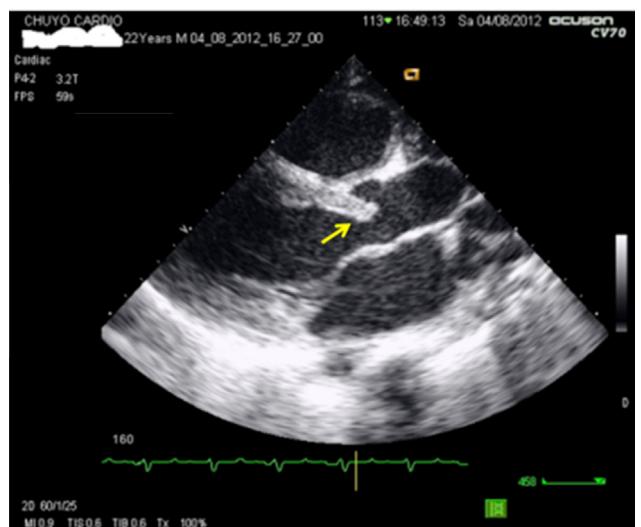


Fig. 1. Two-dimensional transthoracic echocardiography, parasternal long axis showing a hyperechogenic thickening right anterior aortic valve.

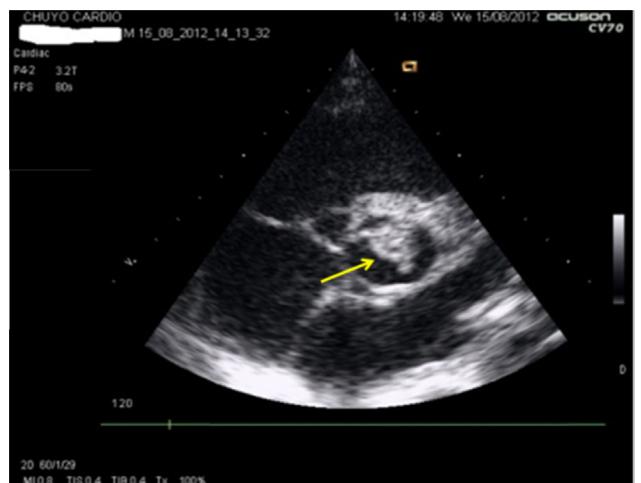


Fig. 2. Two-dimensional transthoracic echocardiography, parasternal short axis showing a hyperechogenic thickening right anterior aortic valve.

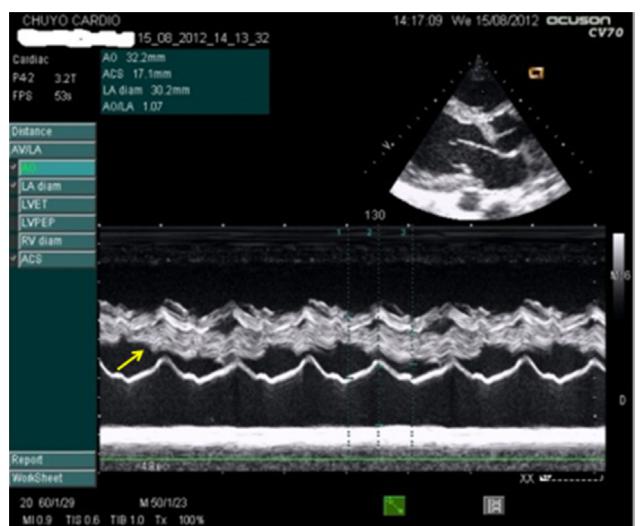


Fig. 3. Parasternal long axis TM mode transthoracic echocardiography showing a hyperechogenic thickening right anterior aortic valve (17 mm).

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