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Cardiac abnormality in a prechemotherapy assessment for leukaemia in a 14-year-old girl

CLINICAL INTRODUCTION

A 14-year-old girl is referred for echocardiography prior to induction chemotherapy for acute T-lymphoblastic leukaemia with over 95% blasts and hyperleucocytosis >100 000 G/L.

Before induction chemotherapy, including anthracycline, a cardiology consultation with echocardiography was performed (figure 1). The global systolic LV function was normal with a LV ejection fraction at 65% using Simpson biplane method. The mitral flow showed diastolic dysfunction with high filling pressures ($E/E' = 2.3$). The ECG showed deep T-waves inversion in the anterior leads. Our patient had no personal or family history suggesting any inherited cardiac disorder.

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ANSWER: D

The assessment revealed a biventricular hypertrophic cardiomyopathy, a LV posterior wall was hypoechogenic and hypokinetic and the LV two-dimensional strain analysis found a segmental hypokinesia with an impaired global longitudinal value of -11.5% . The cardiovascular MRI completed the assessment; it showed a diffuse biventricular oedema presenting as an hypersignal on T2STIR sequences (figure 2A). The T1 mapping corroborated this finding with values above 1050 ms (figure 2B). Early and late gadolinium enhancement sequences showed diffuse and heterogeneous hypersignal (figure 2C) but no subepicardial location as usually observed in acute myocarditis.

In this case, our decision making process used multidisciplinary approach focusing on all possible etiologies of hypertrophic cardiomyopathy (HCM) in paediatrics.^{1,2} Based on first-line clinical and paraclinical assessments, metabolic disorders (Fabry disease), mitochondrial cardiomyopathies, neuromuscular disease, malformation syndromes, endocrine disorders and drugs or infiltrative diseases (amyloidosis) were least likely diagnoses. Therefore, we considered only two diagnoses were relevant: HCM associated with sarcomere protein gene mutations³ and leukaemic cell myocardial infiltration. As our patient had no personal or family history suggesting any inherited

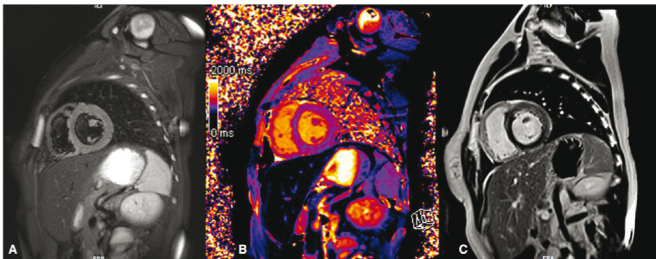


Figure 2 Cardiovascular MRI. (A) T2 weighted sequence in short axis. (B) T1-mapping sequence in short axis. (C) Late gadolinium enhancement sequence in short axis.

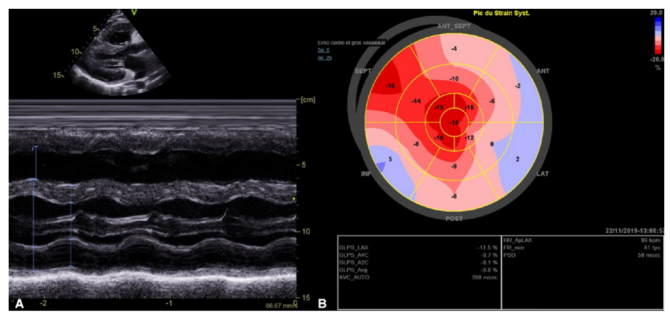


Figure 1 Echocardiography. (A) M-mode parasternal long axis. Right ventricle free wall at 12 mm, interventricular septum at 12 mm (Z score +2.1) and left ventricle posterior wall at 18 mm. (B) Left ventricular two-dimensional strain.

QUESTION

What is the most likely diagnosis?

- Sarcomeric hypertrophic cardiomyopathy.
- Infiltrative diseases like amyloidosis.
- Metabolic disorders like Fabry disease.
- Tumoural cellular infiltration.
- Viral acute myocarditis.

cardiac disorder and considering both the low prevalence of sarcomeric HCM in children, we concluded that the risk-benefit ratio was more favourable to leukaemia treatment, including anthracycline.

One week after induction therapy was initiated, echocardiography showed a nearly complete regression of the hypertrophic myocardial aspect. It suggests tumourous cellular infiltration of the myocardium as described in historical autopsy series.⁴

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Patient consent for publication Not required.

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