LVHT is also frequently found in patients with chromosomal defects.[4] Additionally, a patent ductus arteriosus is frequently associated with chromosomal aberrations. Chromosomal defects may occur with increased frequency in patients with consanguineous parents. Did the patient or his siblings ever undergo cytogenetic investigations, including FISH analysis? Did he or his consanguineous parents present with any dysmorphic features?

Echocardiography in the presented patient showed biatrial enlargement and there was an impaired diastolic filling pattern.[1] Did the patient fulfil the diagnostic criteria for restrictive cardiomyopathy? Restrictive cardiomyopathy has been previously reported in association with oxaluria[5] and could be explained by deposition and accumulation of hydroxyl-butyrate or oxalate in the myocardium.[5]

Complications of LVHT include cardiac embolism, heart failure, ventricular arrhythmias, or sudden cardiac death. Was there any indication for arrhythmias, cardio-embolic events, or heart failure in the presented patient? Was the history positive for syncope, leg edema, stroke or embolism, or palpitations?

Insoluble oxalate may also accumulate in the brain.[1] Did the patient present with any clinical manifestations of cerebral degenerative disease, such as dementia, movement disorder, or epilepsy? Did he ever undergo cerebral imaging, in particular MRI, to exclude involvement of the brain in primary hyperoxaluria or previous ischemic stroke from LVHT? Did he ever develop epilepsy?

LVHT may be diagnosed according to various diagnostic criteria, such as Chin's, Jenni's, or Stöllberger's? Which echocardiographic diagnostic criteria did the authors apply to diagnose LVHT in the presented patient? Was LVHT also confirmed by cardiac MRI?

The patient is reported to have undergone kidney and liver transplantation and thus long-term immunosuppression.[1] Immunosuppression may cause muscle disease. Did the patient develop clinical or subclinical manifestations of secondary skeletal muscle dysfunction during follow-up attributable to any of the immunosuppressive agents applied?

To conclude, this interesting case would benefit from more widespread investigation not only of possible complications of LVHT, but also of involvement in hyperoxaluria of organs other than the heart, and monitoring of possible long-term complications of immunosuppression. For genetic counselling of the parents and their offspring, it would also be helpful to screen the patient and his siblings for chromosomal defects.

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Authors' reply

To the Editor,

The neurologist evaluated the patient and performed electroencephalography (EEG) and electromyography (EMG), with no pathologic findings related to muscle or neurologic involvement of the disease. We considered that fatigue was of cardiac origin since the patient had pulmonary hypertension and hemodialysis 3 times a week due to chronic renal failure.

There were no dysmorphic features in either the patient or his family members. Unfortunately, we were not able to study cytogenetic investigations due to technical impairments in our center's laboratory.

Upon detailed echocardiographic examination, the patient did not fulfill the criteria for restrictive cardiomyopathy. He presented with only first degree diastolic dysfunction; there were no indications for arrhythmias, cardio-embolic events, or clinical heart failure. Nor did he have positive history for syncope, leg edema, stroke, embolism, or palpitations.

There were no symptoms related to neurodegenerative disease. Brain magnetic resonance imaging (MRI) without contrast, computed tomography (CT) imaging, and EEG study were assessed within normal findings.

Diagnosis of non compaction cardiomyopathy was made by echocardiography. The ratio of noncompacted/compacted myocardium (NC/C ratio) was >2:1 at end-diastole, meeting 1 of the Jenni criteria. The patient had NC/C ratio <0.5 at end-systole, meeting 1 of the criteria published by Chin, and with at least 3 apical trabeculations perfusing the intertrabecular spaces, also meeting 1 of the Stöllberger criteria.

Immunosuppressive treatment consisted of tacrolimus, mycophenolate mofetil (MMF), and methylprednisolone. Methylprednisolone was tapered within 3 months after liver transplantation. In the present case, the patient had no muscle disease after transplantation.

We agree that genetic counseling of the parents and their offspring would be helpful in order to screen the patient and his siblings for chromosomal defects. We will consider genetic screening of the patient and his parents for chromosomal defects in another center.

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