sradial approach, and may be underestimated due to its uneventful clinical course.[3,4] Various risk factors including larger sheath size, lower arterial / sheath diameter, repeated cannulation, inadequate anticoagulation, longer duration of compression and high compression pressure have been implicated in radial artery occlusion.[2,5-7]

The exact pathophysiological mechanism of radial artery occlusion is unclear. Dual blood supply of the hand makes it difficult to diagnose silent episodes of radial artery occlusion after earlier catheterization. Also, protective approaches such as the modified Allen's test do not guarantee the prevention of radial artery occlusion. As mentioned in the study by Aykan et al.,[1] patients involved in active outdoor work have larger radial artery diameters, which might be due to adaptive responses. While larger radial artery diameter seems advantageous for patients with an outdoor occupation in preventing various procedural complications, it was not clear for those patients when undesired complications like radial artery occlusion occurred in the active hand. All those patients will need their active hand after transradial cardiac catheterization, both for continuing active occupations and repeated catheterization or graft harvesting in the future. Therefore, some of the advantageous factors should be interpreted and evaluated carefully, especially for patients undergoing transradial catheterization and working at outdoor occupations. In cases of silent and/or symptomatic radial artery occlusion, patients with actively working at outdoor may be disabled. Thus, before transradial catheterization, patient occupation should be evaluated carefully, not only for prediction of radial artery diameter, but also for prevention of postprocedural disabling.

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Left ventricular hypertrabeculation/ noncompaction in hyperoxaluria

To the Editor,

We read with interest the article by Arat et al. about a 19-year-old Caucasian male with primary hyperoxaluria resulting in renal failure nephrectomy, hemodialysis, and kidney and liver transplantation, who also presented with left ventricular hypertrabeculation/ noncompaction (LVHT).[1] We have the following comments and concerns.

LVHT is frequently associated with neuromuscular disorders (NMD)[2] and oxaluria may accompany myopathy.[3] Patients with oxaluria may also develop neuropathy.[3] Was this patient ever seen by a neurologist? Did he ever undergo nerve conduction studies or needle electromyography? Did he ever develop symptoms such as muscle weakness, wasting, muscle cramps, fasciculations, exercise intolerance, muscle aching, or sensory disturbances? Why are the authors so sure that fatigue was of cardiac origin and not attributable to muscle or nerve disease? Systolic function was almost fully preserved.[1]

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.