Introduction

Acute liver failure (ALF) is defined as the rapid development of acute liver injury accompanied by severe impairment of the synthetic function and hepatic encephalopathy in a patient without previous liver disease. It can result from a wide variety of causes, including cardiac failure, and requires urgent evaluation of the underlying cause.1

Ventricular tachycardia (VT) can be difficult to diagnose and might be misdiagnosed as supraventricular tachycardia (e.g. with aberrant conduction) in up to 30% cases2 and is probably even more difficult to detect if heart rate is below the typical frequency of VT (slow VT).3

Case

A 64-year-old male patient was referred to our tertiary care hospital and liver transplant center for further evaluation of ALF. Upon admission to our intensive care unit (ICU), the patient was already intubated and mechanically ventilated (biphasic positive airway pressure mode, fraction of inspired oxygen: 50%, inspiratory pressure: 18 mbar, positive end-expiratory pressure: 8 mbar, respiratory rate: 12/min) and required high doses of noradrenaline (5 mg/h) to maintain a blood pressure of 120/65 mmHg.

The patient was initially admitted to the referring hospital the day before because of increased liver values found during a visit at his general practitioner (Table 1). Due to further worsening of liver values, the patient was transferred to referring hospital’s ICU and then prepared for transfer to us. The patient had to be intubated before transfer and was already in need of catecholamine therapy. The differential diagnosis of sepsis had led to initiation of a broad-spectrum antibiotic therapy with meropenem and vancomycin.

There was no sign of preexisting liver disease on either imaging or in medical history and no indication of increased or chronic alcohol consumption either. The medical history of the patient further showed an acute coronary syndrome and a complete atrioventricular block requiring implantation...
of a cardiac pacemaker about 6 months prior to the current admission. Due to an infected pacemaker pouch, a revision was performed 4 months ago.

After admission to our ICU, the initial workup consisted of an extensive laboratory analysis, an electrocardiogram (ECG) and an abdominal ultrasonography examination. Antibiotic therapy was continued unchanged. Liver ultrasonography showed normal liver morphology without signs of steatosis or cirrhosis. Biliary obstruction could be excluded and liver vessels (portal vein, hepatic artery, liver veins) were open and showed sufficient blood flow on duplex sonography. However, hepatic veins appeared dilated, and inferior vena cava was congested. In the context of a predefined impaired cardiac function, an echocardiography was performed. The latter indicated highly impaired biventricular function with diffuse hypokinesia (left ventricular parameters: ejection fraction: 27%, end-diastolic volume: 117 mL, end-systolic volume: 86 mL, inner diameter diastolic 50 mm, inner diameter systolic: 44 mm) and thereby confirmed ALF secondary due to cardiac failure as primary working diagnosis. A dobutamine therapy was started (10 mg/h) immediately. The ECG (Figure 1(a)) was initially interpreted as atrial fibrillation (122 bpm) with aspects of a right bundle branch block, yet it already appeared atypical to the treating physicians at that time. Laboratory analysis revealed a further increase in liver values (Table 1). In addition, a markedly elevated troponin T was found (1887 pg/mL, upper limit of normal: 50 pg/mL). Thus, an urgent coronary angiography was performed. It revealed dilated left ventricle as well as coronary artery disease with 25% stenosis in all three major coronary vessels. No treatment was initiated (300 mg bolus, then 900 mg/24 h) but did not terminate slow VT either, which next lead to treatment with lidocaine 2% (25 mL bolus, then 4 mL/h). Overall, it was not possible to terminate persisting slow VT and, despite administration of increasing doses of catecholamines, the patient showed progressive hemodynamic failure, which finally led to the patient’s death the day after admission to our ICU.

**Discussion and conclusion**

ALF can result from a wide variety of causes, including cardiac failure. In general, two different mechanisms might contribute to cardiac hepatopathy: first, forward heart failure might lead to reduced arterial perfusion of the liver, which is highly sensitive to a reduced blood flow as the liver receives up to 25% of cardiac output. Second, backward heart failure might cause passive congestion of the liver. Even though arterial hypoperfusion is more common in the setting of acute cardiac failure and passive congestion is more common in the case of chronic heart failure, in most cases, both forward and backward failures coexist and potentiate each other.

Information on the incidence of slow VT (defined as heart rates of 130–186 and 101–148 bpm) was described to be between 6% and 30% in two studies on patients with implantable cardioverter defibrillator (ICD) and was only of minor clinical relevance in these patients. As noted before, diagnosis of slow VT can be difficult and in our patients was only made after analysis of the pacemaker recordings. Nevertheless, the following two points should have raised suspicion of a VT instead of atrial fibrillation on initial ECG: first, the patient had a history of complete atrioventricular block with implantation of a cardiac pacemaker. Second, the ECG showed a regular rate. Slow VT in our patient already persisted for several days and could not be terminated despite medical treatment with amiodarone and lidocaine and multiple attempts of electric

|                     | At admission to our hospital | Before 1 day | Before 2 days |
|---------------------|------------------------------|--------------|--------------|
| Hb (g/dL)           | 17.3                         | 6.09         |              |
| Leukocyte count (/nL)| 12.01                        |              |              |
| GOT (U/L)           | 4898                         | 458          | 297          |
| GPT (U/L)           | 3313                         | 607          | 411          |
| AP (U/L)            | 137                          | 218          |              |
| GGT (U/L)           | 360                          | 569          |              |
| Total bilirubin (mg/dL) | 3.10                       | 3.28         | 2.3          |
| INR                 | 4.08                         | 1.20         |              |
| Creatinine (mg/dL)  | 2.10                         | 1.9          | 1.51         |

Hb: hemoglobin; GOT: glutamic oxaloacetic transaminase; GPT: glutamic pyruvic transaminase; AP: acid phosphatase; GGT: gamma glutamyl transpeptidase; INR: international normalized ratio.
cardioversion. It thus consequently led to cardiac failure with markedly reduced systolic function as indicated by echocardiography findings and finally caused the patient’s death.

Cardiac failure is an important differential diagnosis in the setting of ALF. Slow VT can be difficult to diagnose and if persisting might be difficult to treat and cause acute cardiac failure.

Acknowledgements
The authors thank Susanne Wangler, who was involved in the patients’ care.

Declaration of conflict of interests
The author(s) declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

Ethical approval
Our institution does not require ethical approval for reporting individual cases or case series.

Funding
The author(s) received no financial support for the research, authorship and/or publication of this article.

Informed consent
Written informed consent was obtained from a legally authorized representative(s) for anonymized patient information to be published in this article.

References
1. Gotthardt D, Riediger C, Weiss KH, et al. Fulminant hepatic failure: etiology and indications for liver transplantation. Nephrol Dial Transplant 2007; 22(Suppl. 8): viii5–viii8.
2. Dancy M, Camm AJ and Ward D. Misdiagnosis of chronic recurrent ventricular tachycardia. Lancet 1985; 2: 320–323.
3. Leitz N, Khawaja Z and Been M. Slow ventricular tachycardia. BMJ 2008; 337: a424.
4. Moller S and Bernardi M. Interactions of the heart and the liver. Eur Heart J 2013; 34: 2804–2811.
5. Lusebrink U, Duncker D, Hess M, et al. Clinical relevance of slow ventricular tachycardia in heart failure patients with primary prophylactic implantable cardioverter defibrillator indication. Europace 2013; 15: 820–826.
6. Sadoul N, Metzko R, Anselme F, et al. Incidence and clinical relevance of slow ventricular tachycardia in implantable cardioverter-defibrillator recipients: an international multicenter prospective study. Circulation 2005; 112: 946–953.