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Slow ventricular tachycardia presenting with acute liver failure

Andreas Wannhoff[1], Christian Nusshag[2], Wolfgang Stremmel[1] and Uta Merle[1]

Abstract

Objectives: Cardiac hepatopathy is an important differential diagnosis of acute liver failure. Slow ventricular tachycardia (slow VT) is a ventricular tachycardia (VT), in which heart rate is below the typical frequency of VT. We here report a case of acute liver failure in a patient with slow VT.

Methods: The 64-year old male patient with history of cardiac pacemaker implantation for complete atrioventricular block was referred to our intensive care unit because of acute liver failure.

Results: Workup identified cardiac failure as cause of hepatopathy; however, reason for cardiac failure remained unknown even after left heart catheterization with coronary angiography. Finally, the analysis of cardiac pacemaker recordings led to the diagnosis of slow VT. This could not be terminated with either electric cardioversion or pharmacological treatment, and the patient died of cardiac failure.

Conclusion: Diagnosis of VT can be challenging if occurring at unexpected slow heart rates. Analysis of pacemaker recordings could help to make the diagnosis of slow VT.

Keywords

Slow ventricular tachycardia, acute liver failure, cardiac hepatopathy

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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% cases[2] 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 noradrenalin (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

[1]Department of Internal Medicine IV, University Hospital Heidelberg, Heidelberg, Germany
[2]Department of Nephrology, University Hospital Heidelberg, Heidelberg, Germany

Corresponding Author:
Uta Merle, Department of Internal Medicine IV, University Hospital Heidelberg, Im Neuenheimer Feld 410, 69120 Heidelberg, Germany.
Email: uta.merle@med.uni-heidelberg.de

Table 1. Summary of the patient's laboratory values, which clearly show worsening of liver function over the last days.

	At admission to our hospital	Before 1 day	Before 2 days
Hb (g/dL)	17.3		
Leukocyte count (/nL)	12.01		6.09
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.

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 predescribed 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 of stenosis was performed during coronary angiography due to the low degree of stenosis. In the meantime, the patient's family reported that the patient complains of progressive exertional dyspnea for 1 week, supporting the working diagnosis of cardiac failure. For further evaluation of the yet unclear cardiac failure, the cardiac pacemaker was checked. This revealed a normal pacemaker function, but rhythm recordings showed dissociation of the atrial and ventricular rhythm with a markedly higher ventricular rate compared to atrial frequency. This, together with the fact that there was no stimulation by the pacemaker, led to the conclusion that the current rhythm was of ventricular origin (Figure 1(b)). Based on this, the diagnosis of a slow VT was made, which, according to the pacemaker recordings, already persisted since 6 days. Due to the hemodynamic relevance, electric cardioversion was attempted a total of 8 times (up to 200 J, biphasic) but remained unsuccessful. Amiodarone 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.[1] 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.[4]

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.[5,6] 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

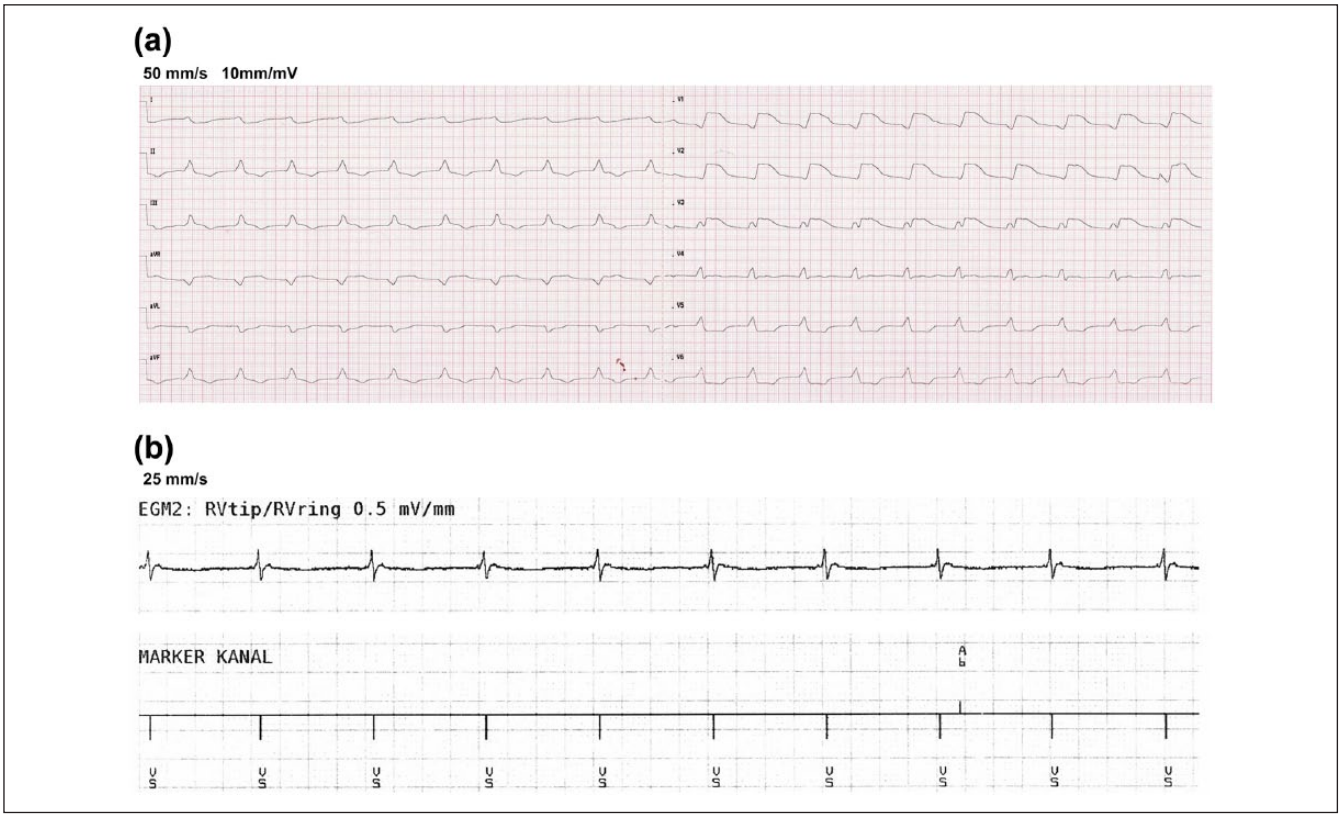

Figure 1. (a) 12-lead ECG recorded upon admission to our hospital and (b) electrogram (EGM) recorded by the pacemaker in the right ventricle, showing a frequency of 75 bpm. As seen on the marker channel, no pacing occurred, and all QRS were sensed by the ventricular lead (VS: ventricular sense). Only one atrial sense is shown on this part of the marker channel (Ab: Atrial sense in postventricular atrial blanking period). It has to be noted that this printout was made after amiodarone and lidocaine therapy were already started, which explains the low atrial frequency (17 bpm) detected in the atrial-derived EGM (not shown) at that time.

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.

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Informed consent

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