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LETTER TO THE EDITORS

Case report: panic attacks as symptom of aortic regurgitation and aneurysm

Christian Spies[1] · Ulrich Laufs[1] · Andreas Hagendorff[1] · Michael Metze[1]

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Sirs:

We report a previously healthy, 31-year-old man, who had presented himself already three times in a psychiatric outpatient clinic. On 26/02/2018, he was hospitalized for depression by his general practitioner. He reported an inner restlessness and sleep disturbances due to nocturnal attacks with shortness of breath after going to bed for about 6 months. The primary psychiatric diagnosis was adjustment disorder with depressive symptoms. During the 5-day inpatient stay, the patient felt very tense and complained of palpitations with shortness of breath several times a day. Medical treatment with mirtazapine at night and lorazepam for the treatment of panic attacks was started. The patient was sent for consultation to the Department of Cardiology 5 days later.

The clinical exam showed a tall man (203 cm, 89 kg, body surface area 2.24 m^2, body mass index 22.6, blood pressure 141/69 mmHg, heart rate 100 bpm, body temperature 36.0 °C) with tachypnea, diastolic heart murmur and bilateral pulmonary rales and peripheral edema.

The patient did not reveal signs of connective tissue diseases. The ECG revealed a sinus tachycardia with right axis deviation, preterminal negative T waves in II, III, aVF, an R loss V1–3 with a delayed R-progression (Fig. 1). Laboratory tests showed an increased gamma-GT of 1.33 µkat/l (normal range 0.17–1.19 µkat/l) as well as a hyperbilirubinemia of 50.3 µmol/l (normal < 17, 1 µmol/l) with an approximately equal proportion of direct and indirect bilirubin. Testing during the subsequent cardiopulmonary resuscitation showed a pro-BNP of 41.521 pg/ml (normal < 125 pg/ml), a troponin T of 125 pg/ml (normal < 14) and slightly increased creatinine: 113 µmol/l (normal 59–104 µmol/l).

Transthoracic echocardiography displayed an eccentrically hypertrophied left ventricle (relative wall thickness = 0.25, threshold 0.42, end-diastolic volume of approximately 600 ml) and an ejection fraction of approximately 25% (Simpson). The aortic ring was dilated to 95 mm and the aortic valve was incompetent: grade IV regurgitation, regurgitation area of 2.2 cm^2, regurgitation volume of 125 ml and fraction about 80% (Fig. 2).

For the preoperative assessment of the aneurysm and the possible involvement of the supraaortic vessels, a computed tomography scan (CT) was performed. Before entering the CT, the patient left the bed to go to the toilet, fainted and went into pulseless electrical activity. Upon arrival of the resuscitation team, he was intubated and resuscitated using a mechanical resuscitation aid (LUCAS™ 2, Company Physio-Control®) for about 30 min until a spontaneous circulation could be restored. The CT showed an aneurysm of the ascending aorta with a maximum diameter of 11.4 cm without signs of rupture or dissection (Fig. 3). In addition, the abdominal aorta was dilated at the thoracoabdominal junction (3.5 cm) and infrarenal region (3 cm).

During the transport and after the arrival at the intensive care unit, the patient went repeatedly into low-output failure. To stabilize the patient, a veno-arterial extracorporal membrane oxygenation (ECMO) was introduced despite the aortic regurgitation (right femoral vein: 23F 50 cm cannula, left femoral artery 19F 18 cm cannula, Cardiohelp® Maquet®, ECMO flow approx. 5 l/min) (Fig. 3). Subsequently, the patient was transferred immediately to cardiac surgery for successful aortic root replacement with a bioprosthetic valved conduit.

After discharge the patient underwent cardiac rehabilitation and was discharged with no further evidence of his "psychiatric" symptoms.

An aneurysm is defined as enlargement of more than 150% of the expected diameter which depends on gender,

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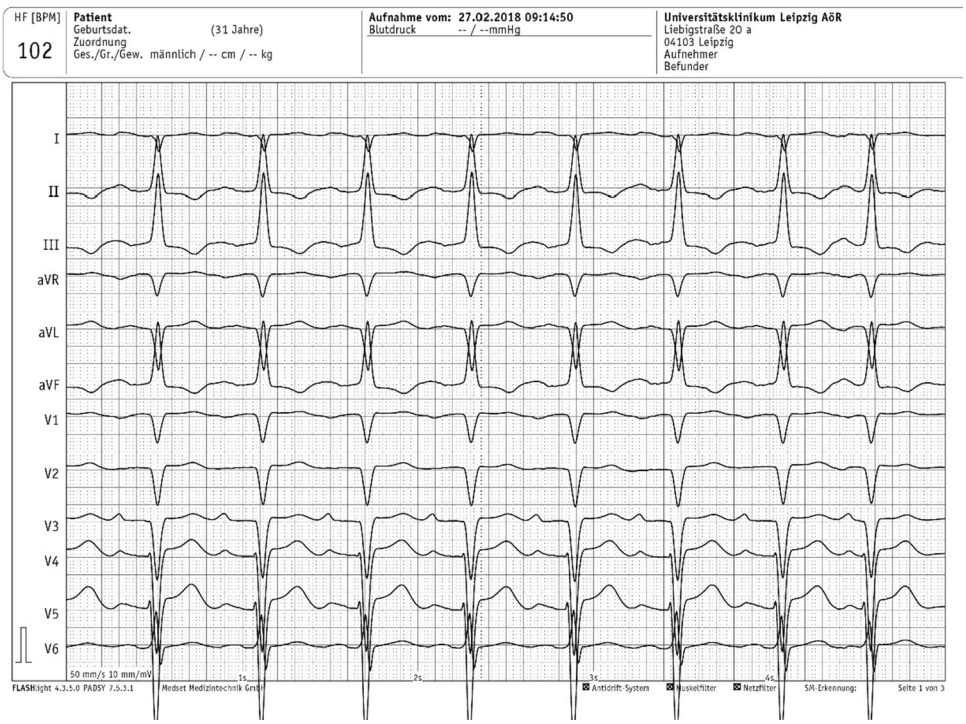
✉ Christian Spies
christian.spies@medizin.uni-leipzig.de

[1] Klinik und Poliklinik für Kardiologie, Universitätsklinikum Leipzig, Liebigstraße 20, 04103 Leipzig, Germany

Fig. 1 Electrocardiogram



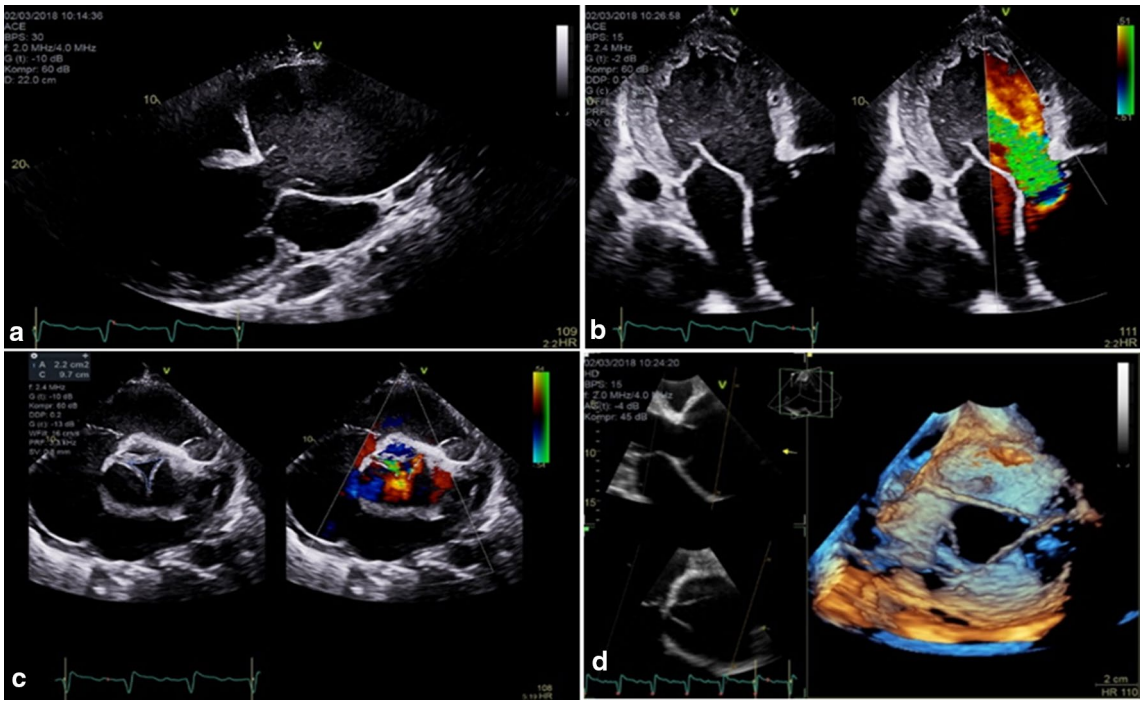


Fig. 2 Different transthoracic echocardiography views. **a** Left parasternal long-axis view, b-mode. **b** Apical long-axis view, color flow. **c** Parasternal short-axis view, colour flow. **d** Aortic valve in end-systole three-dimensional view

size and body weight. Most thoracic aortic aneurysms are caused by degenerative diseases, followed by connective tissue diseases (e.g., Marfan syndrome) [1–3]. The incidence is approximately 10.4 per 100,000. Patients are often asymptomatic and may experience compression symptoms such as dyspnea due to pulmonary compression,

Fig. 3 a: Heart, b: aneurysm, c: descending aorta, d: 23F vein cannula, e: 19F arterial cannula

dysphagia due to esophageal compression, or hoarseness due to recurrent laryngeal nerve compromise [4]. Further, the aortic root involvement may lead to aortic regurgitation and subsequent heart failure [5]. Surgical repair is indicated in aortic diameter > 55 mm, in patients with bicuspid aortic valve > 50 mm or in Marfan patients > 45 mm [5].

Our patient's main complaints were by sleep disturbances with panic attacks and dyspnea (paroxysmal nocturnal dyspnea) which were attributed to an underlying psychiatric disorder. After admission, the history of palpitations and the finding of edema and the pathologic ECG changes prompted further cardiological evaluation. Patients with heart failure often develop depressive disorders [6]. The lack of somatic complaints despite high-grade aortic valve regurgitation with significantly reduced exercise capacity, weight gain and leg edema is unusual in our case. Apparently, the symptoms were not reported or attributed differently by the patient and others.

The cause of the acute cardiac shock and subsequent arrest was most likely the severe aortic insufficiency. In severe aortic insufficiency, the coronary flow reserve is significantly reduced [7]. During the change of body from supine to an upright position, the aortic pressure may drop rapidly allowing a flow reversal in the coronary arteries, which may promote cardiac arrest [8].

The resuscitation was initially performed by manual thoracic compression and was continued by an automatic chest compression system (LUCAS™ 2) until a spontaneous circulation was restored. Here, a sternum compression at a rate of 100/min takes place with a depth of 4–5 cm with a force of about 570 N [9]. Surprisingly, the aneurysm remained intact despite the considerable extra-thoracic force. The compliance of the thorax in younger patients is higher because of higher pliability of the cartilage and bone structures. Consequently, the kinetic energy is probably absorbed to a greater extend by the thoracic wall [10].

The recurrence of cardiac arrest and the bridging of the transport to cardiac surgery made the installation of a veno-arterial ECMO necessary. The return of the blood via the common iliac artery opposing the physiological blood flow, increased in afterload. A relevant aortic valve insufficiency is considered a contraindication of veno-arterial ECMO [11–13]. There is a risk of left ventricular ballooning and pulmonary edema. Interestingly, in a recent observational study, pre-existing moderate aortic valve insufficiency increased left ventricular end-diastolic and end-systolic volumes without increasing left atrial and pulmonary arterial pressures. With an average ECMO duration of about 36 h, there were no differences in mortality, respiratory duration, intensive care stay and renewed hospitalization rate [14]. Alternatively, it would have been possible to implant a micro-axial pump (e.g., Impella® with a maximum flow 5 l/min) which, however, cannot maintain a constant forward flow in severe aortic regurgitation [15].

In conclusion, the unusual case provides three messages. Aortic aneurysm and aortic regurgitations can present with very atypical symptoms, here leading to admission to a psychiatric hospital. The giant aneurysm tolerated intensive and prolonged external trauma. And veno-arterial ECMO may be used in patients with severe aortic regurgitation as a short-term bridge-to-surgery concept.

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