CT. We favor the early administration of urokinase following percutaneous catheter drainage to reduce the length of hospitalization and prevent progressive fibrosis of the pleural surfaces.

Local urokinase therapy has not been reported to produce any significant complications. Lahorra et al measured hematocrit level, prothrombin time, partial thromboplastin time, platelet count, and serum fibrinogen and fibrin degradation products in 26 patients and found no significant change without urokinase therapy. We believe that urokinase therapy can be considered for hemorrhagic effusions in the immediate postoperative period or in the setting of trauma, but serial hematocrit values should be obtained despite the evidence suggesting that local infusion does not produce systemic fibrinolysis. We would also postpone this therapy in a patient with known active bleeding.

The transient fever in case 2 may have represented a systemic response to absorbed urokinase, although similar doses given subsequently produced no febrile reaction. Nonhemorrhagic systemic reactions to urokinase have been reported. These include rigor, hypotension, and anaphylactoid reaction.11,12 Such complications have been noted following intravascular administration of urokinase but have not been reported in conjunction with localized therapy where systemic absorption is much less.

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Torsades de Pointes Arrhythmia in a Patient With Left Ventricular Myxoma*

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A 38-year-old woman suffered from syncope due to torsades de pointes arrhythmia. Echocardiography showed a left ventricular mass that was resected. By immunohistochemical investigations, an organized thrombus was excluded and the diagnosis of a rare ventricular myxoma was confirmed. Postoperatively the arrhythmias resolved without recurrence. We conclude that left ventricular myxoma may cause life-threatening arrhythmias, possibly by irritation of cardiac mechanoreceptors. To our knowledge, this is the first case of a left ventricular myxoma associated with a torsades de pointes arrhythmia.

(LQTS=long-QT syndrome; QT syndrome; QT=QT interval corrected for heart rate; VT=ventricular tachycardia)

Myxomas represent 50 percent of all cardiac neoplasms; 75 percent of these tumors are localized in the left atrium.1 Isolated myxoma of the left ventricle is an exceedingly rare condition, accounting for only 2.5 percent of all reported cardiac myxomas.2 Symptoms of this localization are related to progressive obstruction of the aortic valve or the left ventricular outflow tract, resulting in intermittent or paroxysmal congestive heart failure, chest pain, dyspnea, syncope, or even sudden death.1,3 Systemic embolization occurs in about two thirds of patients.4 Cerebral emboli are most common and often cause permanent neurologic deficits.

Constitutional symptoms (i.e., loss of weight, arthritis, vasculitis, dysproteinemia),1,4 which are commonly seen with atrial myxomas, have not been observed in myxomas of ventricular localization.2 On rare occasions, ventricular myxomas remain clinically silent and are discovered by chance. Because of the rarity and variety of clinical manifestations, diagnosis is difficult and often not made until autopsy. Since diagnostic procedures and particularly echocardiography have improved, myxomas represent a potentially curable disease if diagnosis is made early.

CASE REPORT

A 38-year-old woman (height, 176 cm; weight, 82 kg) was admitted to a community hospital after a syncope. Initial examinations revealed no pathologic findings except a lowered serum potassium level of 3.2 mmol/L. Until the sudden onset of the syncope, the patient had been healthy. Medication history was negative except for a diuretic (zipamide, 10 mg/d) for treatment of edema following anticonceptive medication. All drug treatment had been discontinued several months previously. Hypokalemia was corrected and no other drugs were given. On the second day after hospital admission, a 20-s lasting and self-limiting episode of ventricular...
lar tachycardia (VT) occurred, initiated by a premature ventricular complex (Fig 1). The VT recurred twice during the following 48 h and was refractory to lidocaine. During each VT, the patient fell unconscious. Serum potassium level was normal at that time.

After transfer of the patient to the cardiology department, results of clinical and laboratory investigations, including serologic assays for cardiotropic viruses, were normal. A chest radiograph and the clinical examination yielded no pathologic findings, particularly no deafness.

A resting electrocardiogram showed sinus rhythm of 63/min with recurrent episodes of supraventricular extrasystoles. ST segments were lowered to a maximum of 0.2 mV in leads 2, 3, aVF. The T wave was markedly flattened in all leads and retarded R-progression in V1 to V3 was noted. A prolonged QT interval corrected for heart rate (QTc) was measured (QTc = 0.56 s, >140 percent). Echocardiography revealed an intracavitary mass attached to the left lateral ventricular wall near the apex and partially ejected through the aortic valve in systole. Doppler echocardiography showed no significant gradient and no aortic stenosis or obstruction of the outflow tract was seen. Contractility and chamber sizes were estimated within normal ranges. Coronary angiography was performed to rule out tumors coronary embolism or ischemic heart disease as a probable cause of recurrent VT. Coronary arteries were normal and the left ventriculogram confirmed a highly mobile left ventricular filling defect, which was located in the apex during diastole but moved toward the aortic valve in systole. Hemodynamics were within normal limits.

The patient was referred to our hospital for cardiac surgery. Physical examination yielded normal findings: particularly, no murmurs were heard. There were no specific signs of systemic embolism or neurologic deficit present. The ECG showed the signs of ischemia described above. All laboratory examinations were normal. Magnetic resonance imaging scan confirmed the echocardiographic findings (Fig 2). Since gadolinium-GDPA uptake was poor, an intracavitary thrombus was suggested.

A polypoid and gelatinous tumor was excised from the posterior-lateral wall with a small fibrotic pedicle (3X3X1 cm; weight, 9.5 g). via aortotomy and a transvalvular approach during cardiopulmonary bypass and cardioplegic arrest. Microscopy and immunochemical examinations (Fig 3) confirmed the diagnosis of a pedunculated myxoma of the left ventricle, which was entirely resected. The postoperative course was uneventful and no further arrhythmias or syncope occurred. At 3-month follow-up, echocardiography revealed no local recurrence. The ECG showed sinus rhythm with normal frequencies. The ischemic signs and prolonged QT intervals seen previously had completely resolved.

**DISCUSSION**

Early symptoms due to myxomas may be nonspecific and the tumors are capable of mimicking almost every cardiovascular disorder, including valvular and coronary heart disease. The neoplastic origin of myxomas is now generally accepted and supported by the observation of chromosomal aberrations and the typical occurrence of myxomas in normal hearts in the absence of any organic or hemodynamic factors for enhanced thrombogenesis. Yet, only one case has been described presenting with life-threatening arrhythmia as the possible first manifestation of an intraventricular tumor.

Our patient had no signs of embolism, no obstructive complications, or no constitutional symptoms. The absence of these findings in patients with proved left ventricular myxoma is unusual. Since echocardiography and cardiac catheterization revealed no obstruction of the left ventric-

**FIGURE 1.** During sinus rhythm, a premature ventricular complex (VPC) initiates a rapid polymorphic ventricular tachycardia. Lead 2 shows nonsustained VT with undulations baseline characteristic for torses de pointes.

**FIGURE 2.** Gated transaxial spin-echo magnetic resonance imaging transversely through both, left (lv) and right (rv) ventricle, showing myxoma (T) residing within the left cavity attached to the lateroposterior wall; s =ventricular septum.

**FIGURE 3.** Characteristic morphology of myxoma showing a network of stellate-shaped cells with elongated nuclei. The stroma is characterized by a myxoid vacuolated texture. Vimentin reaction is intense and associated to the cytoplasm in the polygonal cells (vimentin-PAP stain, X400). Insertion of cardiac muscle cells into the myxoma was detected by positive desmin reaction at cross section line (not shown). Staining for factor VIII-related antigen was restricted to the enclosed vessels; surrounding tumor cells and interconnecting stroma were nonreactive (not shown).
ular outflow tract, arrhythmia must be assumed as a cause of recurrent syncope. It cannot be entirely excluded that the first syncope in our patient was initiated by a ventricular arrhythmia due to hypopotassemia. Changes in serum potassium levels may cause ventricular arrhythmias, including torsades de pointes as a result of early after depolarizations.\(^7\) These were clearly documented by prolonged QT intervals in the ECG of our patient.

However, it is remarkable that multiple episodes of torsades de pointes and VT occurred with normal serum potassium levels and several days after initial hypopotassemia had been corrected. Therefore, it seems unlikely that hypopotassemia was the cause of QT prolongation and all subsequent torsades de pointes arrhythmias in our patient.

The finding of a prolonged QT interval and QTc in our patient in the absence of electrolyte disturbances suggest that an underlying long-QT syndrome (LQTS) acted as a trigger for the VT. The LQTS is characterized by recurrent syncopes and sudden death from ventricular arrhythmias, whereas the diagnosis is based on a prolonged QTc interval and on clear family history in case of inherited Jervell-Lange-Nielsen syndrome and Romano-Ward syndrome.\(^8\) These familial LQTSs could be excluded in our patient because neither deafness (characteristic for Jervell syndrome) nor familial involvement (seen with autosomal-dominant Romano syndrome) could be demonstrated. Schwartz et al\(^9\) found that episodes of alterations of the T wave due to sympathetic imbalance are characteristic of LQTS and often precede torsades de pointes.

Apart from reasons named above, abnormal activation of cardiac mechanoreceptors may induce life-threatening arrhythmias.\(^10\) Three different receptors of the heart are sensitive to pressure changes. Besides myelinated vagal afferent nerves from the venous-atrial junction there are two distinct types of mechanoreceptors with a predominant localization in the ventricular wall.\(^11\) One group of mechanoreceptors is served by unmyelinated afferent vagal nerves (C-fibers) and responds to increased volume of the heart.\(^12\) A second group of receptors is supplied by myelinated and unmyelinated fibers\(^2\) that travel to the spinal cord via sympathetic nerves.\(^13\) Both left ventricular mechanoreceptors respond to changes in preload, afterload, and cardiac contractility under normal conditions.\(^13\) Experimental and clinical investigations have shown that simultaneous stimulation of both the vagal and sympathetic afferent nerves (ie, by mechanical irritation) may result in increased vagal and sympathetic efferent activity to the heart, instead of the usual reciprocity between these two systems. Since both systems are activated, electrical inhomogeneity may occur leading to an increased susceptibility of the patients to life-threatening ventricular arrhythmias by decreasing the ventricular fibrillation threshold.\(^14\) Particularly, sudden increase (ie, by mechanical irritation) in sympathetic activity is known as a trigger for coronary vascular spasm,\(^10\) acting in a setting of cardiac electrical instability. A possible cause is seen in a "sympathetic imbalance"\(^9\) mediated by a quantitatively dominant activation of the left stellate ganglion, which leads to prolonged QT intervals (idiopathic LQTS).\(^14\)

Our patient demonstrated three of these findings during her clinical course: QT intervals were markedly prolonged, whereas case history and follow-up gave no evidence for the presence of hereditary LQTS. Underlying serum electrolyte disturbances, particularly hypopotassemia and hypomagnesemia, were also excluded. Nevertheless, recurrent syncopes due to torsades de pointes arrhythmias occurred. Simultaneously, distinct signs of myocardial ischemia were noted, although an underlying ischemic coronary heart disease was ruled out. After surgery, all pathologic findings resolved completely. No further arrhythmias were seen and the QT interval returned to normal ranges.

The absence of familiar QT-syndrome, electrolyte disturbances, and ischemic heart disease in our patient led to the suggestion that the mechanical irritation of ventricular mechanoreceptors by the myxoma is the possible cause of the QT-prolongation and torsades de pointes arrhythmia. Among the wide variety of clinical manifestations, isolated ventricular arrhythmia must be considered as a possible sign of cardiac myxoma. This case should alert physicians to the fact that a syncopal episode or arrhythmia with sudden onset, especially in young persons, may be a sign of a cardiac tumor, even if the chest radiograph and ECG are normal.

### References