To the Editor: Although the triad of palpitations, headaches, and sweating is generally recognized as the classic presentation for pheochromocytoma, we reported the case of a patient with pheochromocytoma who first presented with ventricular tachycardia (VT). This case shows the importance of a broad differential diagnosis when the clinical presentation is unusual.

A 17-year-old woman presented with episodic palpitation and headaches during physical activities for 4 years. The symptoms resolved after 3–5 min of rest. Two days before admission, she was admitted to the emergency department with persistent palpitations and headaches, and the blood pressure (BP) was 180/90 mmHg. The admission echocardiography (ECG) revealed a sinus rate of 92 beats/min. There was no family or personal history of syncope, cardiac arrest, and sudden death. The physical examination was unremarkable. The admission and peak creatine kinase-MB was 153 U/L and 157 U/L, respectively.

During follow-up, the patient presented with recurrent palpitation and headaches when sitting or standing. During these episodes the BP was 160–200/90–100 mmHg and the pulse rate was elevated at 148 beats/min. Cardiac monitoring showed VT with a heart rate (HR) of 150 beats/min [Figure 1a]. Three episodes of VT were recorded during persistent hypertension. The symptoms were relieved after maintaining a recumbent position for 1–2 min. The 24 h urinary catecholamines and urinary vanillylmandelic acid levels were elevated. Abdominal ultrasound revealed a right adrenal mass with a diameter of 6 cm which was confirmed on abdominal computed tomography [Figure 1b]. A pheochromocytoma was diagnosed.

The patient was hemodynamically stabilized in the supine position with the administration of phentolamine (a nonspecific α-adrenergic blocker) and metoprolol (a selective β-antagonist). A right adrenalectomy was performed, yielding a 6.0 cm nodule confirmed to be a pheochromocytoma by histology. The hypertension was controlled and no arrhythmias were documented during 12 months of follow-up.

Pheochromocytoma is a catecholamine-producing tumor that originates from the adrenal medulla or extra-adrenal chromaffin cells. The main clinical manifestation of pheochromocytoma is paroxysmal or persistent hypertension, accompanied by arrhythmias, such as sinus tachycardia, paroxysmal supraventricular tachycardia, atrial fibrillation, or ventricular premature contractions. We have reported a rare case of pheochromocytoma with VT as the presenting symptom. Frequent episodes of VT are almost always preceded by vital sign fluctuation (elevated HR and BP). Paulin et al.[1] reported that the sudden release of catecholamine from the pheochromocytoma is associated with the mechanism underlying VT. Excessively elevated catecholamine levels could have reported a rare case of pheochromocytoma with VT as the presenting symptom. Frequent episodes of VT are almost always preceded by vital sign fluctuation (elevated HR and BP). Paulin et al.[1] reported that the sudden release of catecholamine from the pheochromocytoma is associated with the mechanism underlying VT. Excessively elevated catecholamine levels could...
lead to abnormal myocardial electrical activity, such as over opening of ion channels, enhanced ion exchange pump function, thus causing extremely large amounts of sodium, potassium, and calcium ion flow through the membranes, resulting in intensive myocardial autorhythmicity, conductivity, and triggered activities. Furthermore, increased catecholamine levels can reduce the cardiac ventricular fibrillation threshold, predispose to ventricular fibrillation, lead to sudden death, and contribute to abnormal repolarization, thus causing all types of rapid arrhythmia. Ceremuzynski et al. have shown that plasma catecholamine concentrations are increased in dogs during acute myocardial ischemia and a relationship between the blood catecholamine concentration and the severity of ventricular arrhythmias. Cardiac abnormalities caused by pheochromocytoma includes myocarditis, acute myocardial infarction, transient left ventricular systolic dysfunction, and cardiomyopathy. Bravo et al. reported that catecholamines cause myocardial necrosis, focal myofibrillar degeneration, and subsequent fibrous scar formation. Left ventricular systolic dysfunction may resemble stress-induced cardiomyopathy, also known as takotsubo cardiomyopathy and is reversible in patients with pheochromocytoma. Moreover, long-term high catecholamine levels can lead to myocardial hypertrophy and heart failure.

The presence of sustained VT can be treated with radio-frequency ablation but such treatment would not abolish the systemic arrhythmogenic mechanism and would not resolve the symptoms or prevented the potential onset of other tachyarrhythmias. This case illustrates the importance of a complete differential diagnosis when the clinical presentation is unusual. In a hypertensive patient with a typical presentation consisting of palpitations and headache, and an ECG demonstrates VT or other tachyarrhythmias, a pheochromocytoma should be included in the differential diagnosis. In addition, biochemical testing and imaging studies will guide the diagnosis and allow complete resolution of symptoms following resection of the pheochromocytoma. The preoperative use of phentolamine, a nonspecific α-adrenergic blocker has a theoretical pharmacologic basis. The β-blocker propranolol is used when tachycardia or catecholamine-induced arrhythmias are present and should only be started after α-blocker has been established to avoid life-threatening hypertension.

In conclusion, this case shows that VT is an unusual and initial cardiovascular presenting symptom in patients with a pheochromocytoma. Pheochromocytoma must be considered in patients with frequent VT plus sudden changes in BP. Resection of chromaffin cell tumor will completely relieve the symptoms.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

REFERENCES
1. Paulin FL, Klein GJ, Gula LJ, Skanes AC, Yee R, Krahn AD. QT prolongation and monomorphic VT caused by pheochromocytoma. J Cardiovasc Electrophysiol 2009;20:931-4. doi: 10.1111/j.1540-8167.2008.01405.
2. Wittstein IS, Thiemann DR, Lima JA, Baughman KL, Schulman SP, Gerstenblith G, et al. Neurohumoral features of myocardial stunning due to sudden emotional stress. N Engl J Med 2005;352:539-48. doi: 10.1016/j.accreview.2005.05.028.
3. Ceremuzynski L, Staszewska-Barczak J, Herbaczynska-Cedro K. Cardiac rhythm disturbances and the release of catecholamines after acute coronary occlusion in dogs. Cardiovasc Res 1969;3:190-7. doi: org/10.1093/cvr/3.2.190.
4. Bravo EL. Pheochromocytoma: New concepts and future trends. Kidney Int 1991;40:544-56. doi: org/10.1038/ki.1991.244.
5. Park JH, Kim KS, Sul JY, Shin SK, Kim JH, Lee JH, et al. Prevalence and patterns of left ventricular dysfunction in patients with pheochromocytoma. J Cardiovasc Ultrasound 2011;19:76-82. doi: 10.4250/jcu.2011.19.2.76.