CASE 9: WHEN THE T WAVES ARE BIZARRE AND INVERTED

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A 39-YEAR-OLD woman, with no history of heart disease and taking no medications, was brought to the emergency room with one-hour history of altered state of consciousness, preceded by sudden headache and vomiting. Her temperature was 37.7 °C, pulse was 70 beats/min and irregular, respiration was 14/min, and blood pressure was 110/70 mm Hg. She was confused and irritable with moderate neck stiffness and had no localized neurological deficit. Examination of the heart was negative apart from the irregular heart beats. The serum electrolytes were normal and the ECG is shown below.

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Questions

(1) What is the ECG diagnosis?

(2) What is the underlying disorder?

(3) What is the therapeutic approach?

ECG Analysis

The ECG shows an irregularly irregular rhythm with no clear P waves consistent with atrial fibrillation. Course fibrillation/flutter waves can be seen in V1. The axis and the QRS morphology are normal. The T waves are large, wide, and deeply inverted in leads V2 to V6. T waves are also inverted in leads II, III, and aVF with a prominent U wave seen mainly in lead II. The QT interval is prolonged with a QTc = 0.61 s.

Although the T-wave changes suggest myocardial ischemia, the patient's age, history, examination, and the diffuse nature of the T-wave changes can rule out this diagnosis. The history is compatible with the diagnosis of cerebrovascular accident. The ECG finding of atrial fibrillation/flutter is suggestive of a cardiac origin of an embolic cerebrovascular accident. The otherwise normal cardiac examination and the absence of localizing signs on the neurological examination in a patient who had no history of heart disease make this suggestion less likely.

The history of sudden onset of headache and vomiting in an otherwise healthy young female, the nonspecific and non-localized neurological abnormalities, and the ECG findings are typical of subarachnoid hemorrhage (SAH). CT scan demonstrated the presence of subarachnoid blood, and cerebral angiography confirmed the diagnosis of SAH from the middle cerebral artery aneurysm for which surgical clipping was successfully performed.

Discussion

Acute cerebrovascular disorder may induce central nervous system-mediated electrocardiographic abnormalities which occur in the absence of coexisting cardiac disease. The association between these electrocardiographic abnormalities and cerebrovascular events has long been noted.1-5 This is particularly true of subarachnoid and intracranial hemorrhage. In one report, ECG changes were seen in 71.5% of patients with SAH and in 57.1% with cerebral hemorrhage.6 The pattern of the ECG abnormalities is such that if the association is not recognized, an erroneous diagnosis of ischemic heart disease, electrolyte imbalance, or drug effect is often made.

The most characteristic ECG findings in patients with SAH are large, wide, upright, or deeply inverted T waves; prolongation of QTc interval; and prominent U waves.7-9 These changes may persist for as long as 11 days.5 Less typical T-wave changes consisting of lowering of the amplitude or notching may be seen.10 Diffuse ST-segment depression as well as elevation, which simulate the injury pattern of ischemic heart disease, may occur.11 In some cases, transient abnormal Q waves develop7-8 and the erroneous diagnosis of myocardial infarction may be made.11 Peaked, broad, or bifid P waves may be seen.7-12 Rhythm disturbances that may occur in patients with SAH include sinus bradycardia, sinus tachycardia, wandering pacemaker, supraventricular tachycardia, atrial fibrillation (as in this case), atrioventricular junctional rhythm, premature ventricular beats, and ventricular tachycardia.7,12-17

Many reports support the idea that the pathogenesis of the characteristic ECG changes is related to the altered autonomic tone in patients with SAH. Autonomic neural stimulation from the hypothalamus can cause arrhythmias and a variety of ECG changes which mimic acute myocardial infarction.15,19 In animal studies, these ECG changes could be prevented by spinal cord transection, vagal interruption, or by satellite ganglion blockade, indicating that the hypothalamus appears to exert these cardiac effects by direct nerve-to-end organ innervation.20 Other methods of autonomic blockade including vagolytics21 and adrenergic blockers given intravenously22 had similar blocking effects. Direct intracoronary injections of epinephrine15,23 and acetylcholine24 have been shown to induce ECG changes that are characteristic of myocardial ischemia without evidence of myocardial damage on autopsy. Vasopressor responses, but not ECG responses caused by hypothalamic stimulation, can be blunted or inhibited by bilateral adrenalectomy.25

From the above reports, it is uncertain whether the autonomic nervous system-mediated ECG changes are caused by direct nerve innervation or
intravascular release of autonomic mediators or both. Both of these mechanisms are capable of causing ECG changes and, most likely, a combination of factors is operative in causing observed effects. 16

Tachyarrhythmias occurring in SAH may cause major hemodynamic compromise in patients who are already unstable due to acute neurologic problem. Beta-blocking agents have proven useful in controlling both SAH-related supraventricular and ventricular arrhythmias, including torsades de pointes.26,27 Very little has been reported about the treatment of arrhythmias in SAH, therefore, firm recommendation cannot be made. Nonetheless, beta blockers appear to be the most useful agents because most of the arrhythmias are presumably secondary to or mediated by elevated plasma catecholamine levels. 16

Although many patients with SAH have ECG changes, very few have demonstrable myocardial damage, in general, or ischemic damage, in particular. 16 Catecholamines can cause SAH-induced myocardial damage which might be due to their coronary vasoconstrictor effects.28,29 Since the two main mechanisms that might mediate the ECG changes in patients with SAH include autonomic stimulation of the hypothalamus-elevated catecholamine levels, it is possible that the first might be the cause of ECG changes without association which in turn may cause myocardial damage, whereas the second may result in myocardial damage.

References
21. Smith M, Ray CT. Cardiac arrhythmias increase intra-cranial pressure and the autonomic nervous system.
22. Hockman CH, Mauck HP, Hoff EC. ECG changes resulting from cerebral stimulation II. A spectrum of ventricular.