Case report

Torsades de pointes due to ischemic stroke

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Original submission: 24 December 2012; Revised submission: 11 January 2013; Accepted: 16 January 2013.

ABSTRACT

Cardiac and cerebrovascular illnesses are among the leading causes of mortality and morbidity today. Thromboembolic cases, which are the result of cardiac arrhythmia, are one of the important causes of cerebral stroke. However, various abnormalities, especially ST-T wave changes on electrocardiography can be seen in patients who do not have any heart disease history but had ischemic cerebral stroke. In this study, we have presented an interesting case of Torsades de Pointes due to ischemic cerebral stroke.

Key words: torsades de pointes, cerebral stroke, brain ischemia

INTRODUCTION

Repolarization abnormalities or electrocardiographic changes alike ischemia can be watched in the early periods of ischemic stroke (1). ST-T segment changes, T wave negative, U wave pattern, QT distance extension or supraventricular-ventricular rhythm abnormalities can be ranked as the most frequent changes. Ventricular extrasystoles are the most common ones among ventricular arrhythmia (2).

Torsades de pointes (TdP) is a potentially life-threatening condition which is characterized by QT distance extension and polymorphic ventricular tachycardia. TdP is usually self-limited but may degenerate into ventricular fibrillation. The incidence of sudden cardiac death in this rhythm abnormality is about 32% (3). Certain drugs (e.g. antidepressants, cimetidine, clarithromycin and haloperidol) and electrolyte imbalances (e.g. hypomagnesaemia and hypokalemia) may lead to TdP (3).

In this case report, we aimed to present a patient with serious ventricular arrhythmia which is eventually diagnosed as TdP due to ischemic cerebral stroke.

CASE REPORT

A 65-year-old male patient applied to Emergency Department of Abant Izzet Baysal University Hospital with the complaints of fatigue, malaise and aphasia. There was no history of trauma, drug use or systemic disease. His finger tip blood sugar was 95 mg/dL, tension arterial was 135/75 mmHg, pulse was 82/minutes, finger tip was SpO₂: 94%, axillary fever was 36.7°C. His electrocardiography (ECG) was at normal sinus rhythm and 80/minutes speed, no pathology was encountered. In the neurologic evaluation, Glasgow coma scale (GCS) was 11 (E3, M5, V3), there was no neck stiffness, bilateral Babinski was positive. Acute pathology was not detected in tomography of the head. Blood electrolytes, blood sugar, liver and kidney function tests, troponin-I value were found as normal in laboratory observations. While the monitored follow-up of the patient was going on, spontaneous TdP developed (Figure 1). Amiodarone 150 mg and magnesium sulfate 2 gr were administered intravenously. The patient did not respond to this treatment protocol and his general condition deteriorated rapidly. Cardioversion was applied with synchronized 150 joule as there was no reply to the medical treatment, the tension arterial descended to 80/40 mmHg and it was GCS:8 (E2, M4, V2). As there was no answer to the first cardioversion, second cardioversion was applied with 200 joule and regression to sinus rhythm was observed. Amiodarone infusion 900 mg/24 hour was started as 60 mg/h (6 hours) and 30 mg/h (18 hours) to the patient who was evaluated by Cardiology Clinic. In his echocardiography that was

Figure 1. The patient’s monitor screen demonstrates torsades de pointes (Ocak T, 2012)
scanned in urgent conditions, ejection fraction was 55%, but cardiac valve and wall motion disorder were not detected. Common hypotension belonging to acute infarct, which accords to posterior cerebral artery and superior cerebellar artery irrigation area, which includes mesencephalon, pons, both thalami, cerebellar hemispheres and occipital lob, was observed in control tomography of the head that was made 12 hours later in line with the suggestion of neurology (Figure 2). The patient passed away due to multiple organ failure on the 4th day of his intensive care follow-up.

Figure 2. Mesencephalon, pons, both thalami, cerebellar hemispheres, occipital lobe, involving the posterior cerebral artery and the superior cerebellar artery suited for the common hypodense of acute infarction (arrows) (Ocak T, 2012)

Over 730,000 new stroke cases are detected every year in the USA and 27% of these cases result in death (4). Acute strokes may be classified into two general subtypes: ischemic (80-85%) and hemorrhagic (15-20%) (4). Atherothrombosis, embolism and hypoperfusion are among the most frequent reasons of ischemic strokes (5). The reason of the half of cardiovascular-based embolic strokes is atrial fibrillation (4,5). In the studies, even though there was no known heart disease story detected in a patient, it has been found that new-onset cardiac conduction disorders may develop in acute cerebral ischemic or hemorrhagic cases (6). It is thought that noncardiac ischemic ECG changes observed in patients with stroke may be the result of autonomic activity depended on increased sympathetic discharge (7). In the ischemic stroke scan of Daniele et al. composed of 352 cases, ventricular tachycardia was detected in 15 cases. However, it was stated that ventricular tachycardia did not have hemodynamic effect and was not determined (8). In our case, there was no known heart disease or rhythm disorder consistent with literature. It was thought that new TdP depends on increased sympathetic discharge (9). Generated TdP was a determined state which did not respond to the treatment and needed response with electrical cardioversion. After the potential of ECG change with the area affected by ischemia, it was reported that more ECG changes were observed at brain stem lesions in the study of Norris et al (9). In our case, there was a common ischemic lesion including brain stem and right cerebellar hemisphere. In the literature scan atrial tachycardia (10), QT anomaly (11), early repolarization findings (12), to which ischemic cerebral incident is thought to be caused, were encountered in observed cases. Di Pasquale et al detected five TdP cases among 132 subarachnoid bleeding cases in 24 hours holter monitoring and found that rhythm control was enabled with medical treatment in three of these cases and two cases were lost in 24 hours related to cerebral pathology (13). In our case, electrical cardioversion was applied because there was no response to medical treatment. In the literature there were no TdP cases caused by ischemic stroke like in our case.

As a result, the follow-up of the patients should be made by clinicians and monitoring should be done in emergency observation unit at the stage of diagnosis. In case of fatal ventricular arrhythmia, such as TdP, it might develop serious ischemic cerebral complication, as it happened in our case.

FUNDING
No specific funding was received for this study.

TRANSPARENCY DECLARATIONS
Competing interests: none to declare.

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