VIA MEDICA

CASE REPORT

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Cruel surprise — stroke in the shadow of chloroquine and prolonged QT interval: a case report

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ABSTRACT

Stroke is one of the leading causes of death and disability in adults. It can present itself in many different ways beginning with focal neurological symptoms and ending with sudden cardiac arrest. Stroke predisposes to ventricular arrhythmias due to prolongation of the QT interval. This problem mainly affects patients with additional risk factors that affect QT interval prolongation. The following case report presents the story of a 68-year-old patient with diabetes mellitus, hypertension, heart failure and rheumatoid arthritis taking chronic chloroquine diagnosed in the ED and subsequently admitted to the Cardiac Intensive Care Clinic for out-of-hospital ventricular fibrillation cardiac arrest in the course of recurrent ventricular tachyarrhythmias, who was diagnosed with ischaemic stroke. Coordinated management is presented to achieve a stable outcome.

Keywords: defibrillators, implantable; ischaemic stroke; out-of-hospital cardiac arrest; tachycardia, ventricular

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Introduction

CNS (central nervous system) stroke is defined as cell death in the brain, spinal cord or retina attributable to ischaemia, based on neuropathological, neuroimaging and/or clinical evidence of permanent damage. The main cause of ischaemic stroke is the presence of atherosclerotic plaques in the cerebral or carotid arteries or their rupture [1]. On electrocardiography in stroke, the most commonly observed ECG (electrocardiogram) changes are prolonged QTc interval (36%), ST-segment depression (24.5%), atrial fibrillation (19.9%), and T-wave inversion (17.8%) [2]. The QT interval on the ECG represents the time of ventricular depolarisation and repolarisation, and prolongation of the heart rate-corrected QT interval is associated with functional re-entry loop, torsade de pointes and sudden cardiac death [3]. According to current guidelines, prolonged QT syndrome is diagnosed at values > 480 ms or > 460 ms if accompanied by syncope. QT prolongation can be congenital or acquired. Causes of acquired QT prolongation include metabolic disorders, bradycardia including grade II-III atrioventricular blocks, acute coronary syndromes, stroke, and the use of certain drugs [4]. A full list of drugs that prolong the QT interval can be found at www.qtdrugs.com [5].

Case report

A 68-year-old man was admitted to the ED (emergency department) after OHCA (out-of-hospital cardiac arrest) due to ventricular fibrillation. On admission, the patient was conscious, cardiovascularly and respiratorily efficient and reported no significant symptoms. According to witnesses, the patient had a seizure

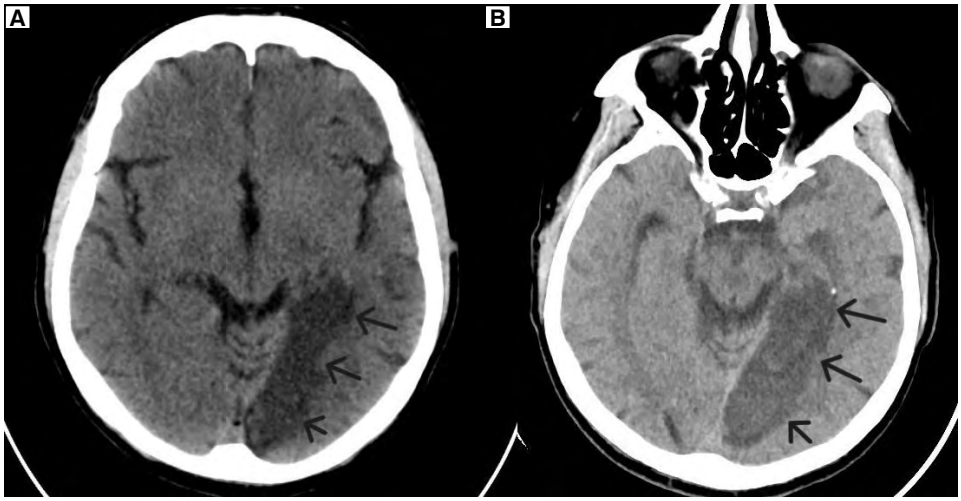

Figure 1. CT scan of patient's head A) on admission to ED B) after 6 days. The arrows indicate the foci of the stroke

and fell from a ladder. The day before, he reported mood deterioration and memory impairment. The patient was previously treated for hypertension, type 2 diabetes mellitus, hypercholesterolaemia, gout and rheumatoid arthritis with perindopril, acetylsalicylic acid, atorvastatin, metformin, allopurinol, methotrexate, chloroquine. A head CT (computed tomography) performed in the ED showed an area of hypodensity of approximately 25 × 75 mm in the left temporal-occipital lobe, the lesion was interpreted as ischaemic probably acute. Compression of the temporal horn and triangle of the left lateral ventricle (Fig.1).

The patient was disqualified from reperfusion therapy due to exceeding the time window for thrombolysis (4.5 h) and thrombectomy (6h) [6, 7]. Subsequently, he was admitted to the Cardiac Intensive Care Clinic due to attacks of ventricular tachycardia. Through the patient's documentation, it was established that 3 weeks before admission, the patient had a coronary artery angio-CT scan which showed no significant stenosis, a muscular bridge over the anterior descending branch, left ventricular ejection fraction of 47%, Calcium score 7. The ECG showed prolongation of the QTc interval to 570ms, right bundle branch block, left anterior bundle branch block, and deep negative T-waves in the pre-cardiac leads (Fig. 2).

Echocardiography showed: generalised contractile dysfunction with a decrease in left ventricular ejection fraction to 30%. In laboratory tests, a decrease in troponin T hs after one day of hospitalisation from 142 ng/L to 90.7 ng/L without significant changes in other laboratory tests. A chest X-ray showed an enlarged cardiac silhouette and an aorta with calcifications. During

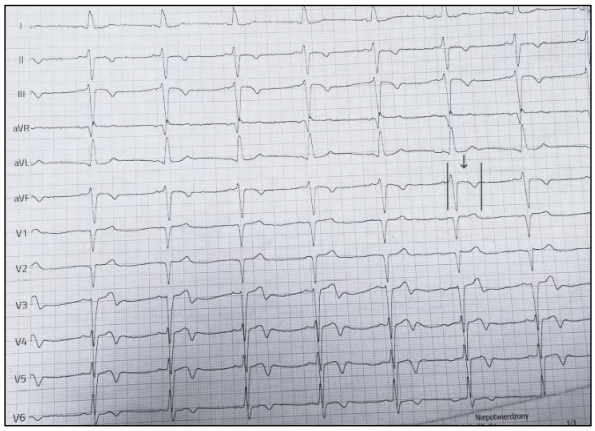

Figure 2. ECG examination of a patient showing prolonged QTc interval (570ms). The arrow indicates the QT interval

hospitalisation, a prolonged QT interval persisted and multiple episodes of non-sustained ventricular tachycardia were present (Fig. 3).

The rhythm abnormalities resolved with lignocaine infusion and recurred with dose reduction. Magnesium i.v., β-blocker i.v. and p.o. were started. QTc shortening and electrical stabilisation were achieved. Subsequently, a dual-chamber cardioverter-defibrillator was implanted for secondary prevention of sudden cardiac death. Due to the high risk of haemorrhage of the current stroke lesion and the occurrence of a recurrent stroke, as well as the available angio-CT image of the coronary arteries from 3 weeks ago, the patient was not qualified for urgent coronary angiography, but this was performed after 2 months (no significant changes in the coronary arteries were found).

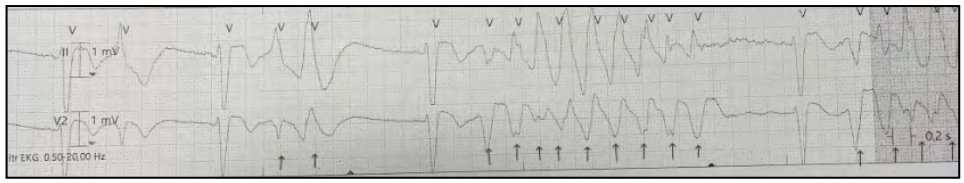

Figure 3. Ventricular tachycardia attack in a patient. The arrows indicate ventricular beats in the course of tachycardia

Discussion

In cases of OHCA, the medical history obtained from witnesses to the event is often difficult to specify. In this case, physicians, due to the tonic-clonic seizures, assumed that the primary cause of the OHCA was a neurological incident, which was unclear after specifying the history. After successful defibrillation, the patient in the ED was referred for a head CT scan to exclude post-traumatic lesions and neurological causes of OHCA. The examination showed an area of ischaemic stroke, which, independent of other factors, could have been the direct cause of cardiac arrest. CNS ischaemia is a direct cause of OHCA in 7.7%, but the majority are caused by heart disease [8]. In this case, there was an accumulation of factors causing QTc interval prolongation: reduced ejection fraction, stroke and use of chloroquine, which is one of the QT-prolonging drugs. Both acquired and congenital prolonged QT syndromes are common causes of sudden cardiac death. In prolonged QT syndromes, premature R on T ventricular beats can occur, which increases the risk of complex cardiac arrhythmias [9]. In all these conditions, attention should be paid to the possible coexistence of the above disorders. Routine head CT in patients after cardiac arrest can identify many CNS lesions, even if they are asymptomatic or difficult to detect on neurological examination. In addition, it is important to remember to perform a regular ECG at rest in patients chronically treated with QT-prolonging drugs, in order to prevent possible complications in the form of arrhythmias.

Conclusions

The above paper aims to show the possible causes of sudden cardiac death in patients and how important it is to determine the exact cause in each case. In patients with OHCA, it is important to perform both pre-hospital management (on-site CPR (CardioPulmonary Resuscitation) before the arrival of the paramedic team) and then transport the patient to a hospital intensive care unit as soon as possible to establish the potential cause (including the identification of reversible causes of cardiac arrest). The coordinated and multidisciplinary approach in the above case enabled the patient's vital signs to return and his condition to be stabilised, with the prevention of recurrent attacks of ventricular tachycardia. It is important to be aware of the possibility of QT prolongation with the use of certain drugs and congenital long QT syndromes, as missing these situations can be associated with life-threatening complications.

Article information

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