Kardiologia Polska 2013; 71, 5: 545; DOI: 10.5603/KP.2013.0111 ISSN 0022–9032

STUDIUM PRZYPADKU / *CLINICAL VIGNETTE*

Characteristic electrocardiographic repolarisation changes in a 9 year-old boy with hypertensive encephalopathy

Charakterystyczne zmiany repolaryzacji w EKG u 9-letniego chłopca z encefalopatią nadciśnieniową

Ana Veličković[1], Sergej Prijić[2], Bojko Bjelaković[3]

[1]Health Department, Department of Paediatrics, Leskovac, Serbia
[2]Mother and Child Health Institute, Belgrade, Serbia
[3]Clinic of Paediatrics, Clinical Centre, Medical Faculty, University of Niš, Serbia

Electrocardiographic (ECG) abnormalities, as well as rhythm changes associated with the central nervous system (CNS) pathology, have been known for decades. They have been reported commonly in patients with subarachnoid and intracerebral haemorrhages, but also in a variety of other CNS disorders such as CNS infections, tumours, traumas and neurosurgical procedures. An elevated intracranial pressure has also been suggested as a potential cause of neurogenic ECG abnormalities. However, it is interesting that there is still no published data describing ECG changes in patients with hypertensive encephalopathy. We report the case of a 9 year-old boy presenting with loss of consciousness, systemic arterial hypertension, and generalised seizures. A 12-lead ECG obtained at admission revealed sinus bradycardia and non-specific ECG repolarisation changes, together with markedly prolonged QTc interval (QTc of 0.60 s) (Fig. 1). Since the cerebrogenic ECG changes due to hypertensive encephalopathy had not previously been described, a reasonable doubt that a child could have a primary myocardial electric (a likely aetiology or comorbid condition for this patient's presentation) made us unable to make a correct initial diagnosis. After the definitive diagnosis of hypertensive encephalopathy had been established in the next few hours (Fig. 2), the actual aetiology of such ECG changes was revealed to us. Serial ECG tracings over the next few days, eventually becoming normal on the 5th day of hospitalisation (Fig. 3), finally ruled out primary long QT syndrome as a possible pathologic condition in our patient, and pointed to a 'cerebrogenic' origin of the noted electrophysiological changes. It is possible that these could have been responsible (prolongation of QT interval and possible cerebrogenic cardiac arrhythmias) for the loss of consciousness and convulsions in our patient. Although increased intracranial pressure might potentially explain sinus bradycardia, systemic hypertension as well as cardiac repolarisation changes in our patient, computed tomography scan of a head showing ischaemic brain damage in left occipitally cortico-subcortical region, strongly supported a different etiopathogenesis for such ECG alterations. This is coincident with the results of a few studies showing that the incidence of ECG abnormalities in patients with subarachnoid haemorrhage or brain tumours does not increase noticeably in a group of patients with elevated intracranial pressure, compared to those patients without elevated intracranial pressure. Since a primary or secondary prolonged QT interval is frequently considered as a major risk determinant for cardiac arrhythmias and mortality, it is of great importance to clarify the real aetiology of similar ECG changes as a potential differential diagnostic clue and clinical hallmarks in all children suspected of having CNS disorder. Our case highlights the importance of interpreting changes in the ECG as a potential differential diagnostic clinical tool in all children suspected of having CNS disorder. To the best of our knowledge, a similar clinical presentation has not been described before in the literature.

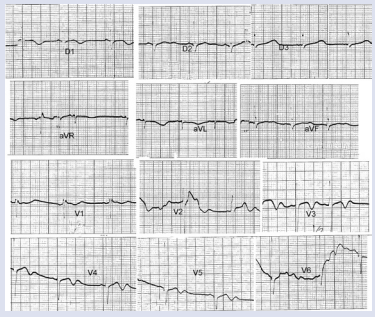

Figure 1. The standard 12-lead ECG demonstrating negative T wave in leads D1 and aVL, notched T wave in leads V_4–V_6, as well as markedly prolonged QTc interval, QTc = 0.60 s in all leads

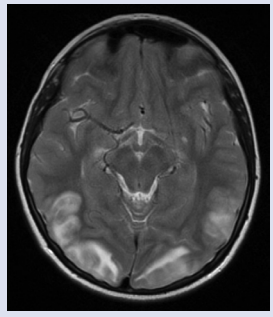

Figure 2. Multislice computed tomography scan of the head showing the hypodense area in the left occipitally cortico-subcortical region

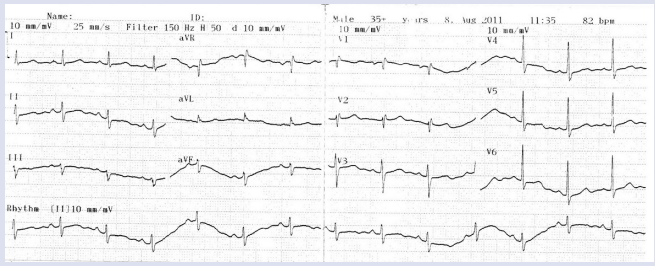

Figure 3. The normal appearance of standard 12-lead ECG on the hospital discharge day

Address for correspondence:
Ana S. Veličković, MD, Health Department, Department of Paediatrics, Leskovac, 116 Svetozara Markovića St., 16000 Leskovac, Serbia, tel: +38116251244, fax: +38116251244, e-mail: avelickovic7@ptt.rs

Conflict of interest: *none declared*

This work was supported by a grant No 175092 from the Ministry of Science and Technology of the Republic of Serbia.