

Dynamic ECG changes in a patient with subarachnoid haemorrhage

Dynamiczne zmiany w EKG u chorego z krwotokiem podpajęczynówkowym

Piotr Kukla¹, Marek Jastrzębski², Wojciech Kurdzielewicz¹, Leszek Bryniarski², Wiktor Zajchowski⁴, Adrian Baranchuk³

¹Department of Internal Disease and Cardiology, Specialistic Hospital, Gorlice, Poland

²^{1st} Department of Cardiology, Interventional Electrophysiology and Hypertension, University Hospital, Krakow, Poland

³Division of Cardiology, Kingston General Hospital, Queen's University, Kingston, Canada

⁴Polpharma Commercial Office Department, Warsaw, Poland

Acute cerebro-vascular disorders (ACVD) such as subarachnoid haemorrhage (SAH) increase sympathetic activity and α -adrenergic stimulation. Pathologic α -adrenergic stimulation can provoke several electrocardiogram (ECG) changes including ST-segment depression, wide, broad T-waves, U-waves merging into the T-waves, and QTc prolongation. Previous reports have shown that J-waves can appear in patients with ACVD and a brain injury [1–3]. J-wave is a deflection occurring at the J-point described by Osborn and called 'the injury current' in experimental models of hypothermia in dogs [4]. In addition to hypothermic patients, the J-wave can

be observed in hypercalcaemia and arrhythmogenic disorders such as in patients with idiopathic ventricular fibrillation (VF) who usually depict the so-called 'Haissaguerre pattern' [5, 6]. We previously reported the case of patient with SAH and a prominent J-wave associated with VF [7].

The aim of this presentation is to report an unusual ECG presentation in a patient with SAH.

We present the case of a 36-year-old man with recent SAH. He was admitted to the ER because of persistent and 'in-crescendo' headache. ECG on admission showed sinus rhythm at 60 bpm, PQ interval of 180 ms, QTc of 480 ms,

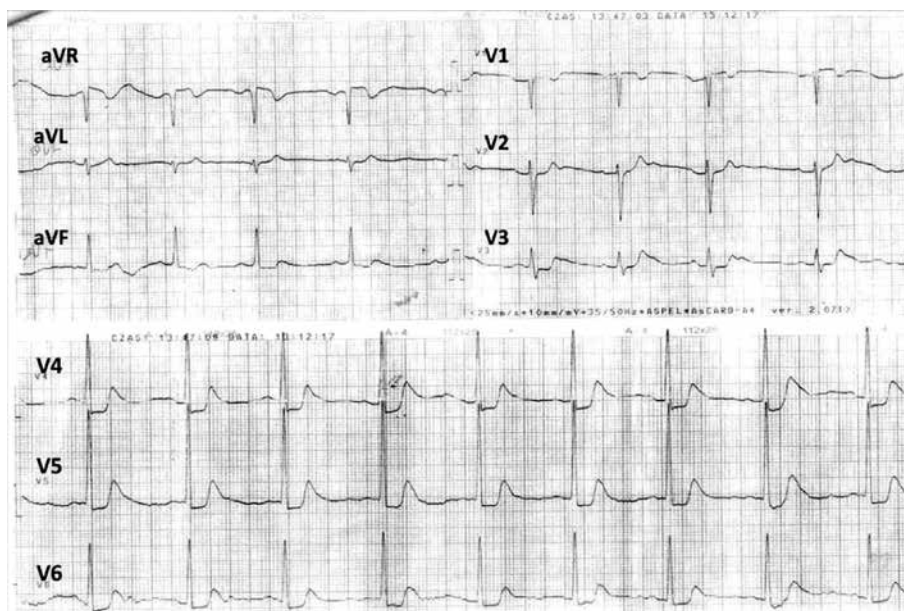


Figure 1. Electrocardiogram on admission (see details in the text). Diffuse ST-segment depression with prolonged $T_{peak}-T_{end}$ interval

Address for correspondence:

Piotr Kukla, MD, PhD, Department of Internal Disease and Cardiology, Specialistic Hospital, ul. Węgierska 21, 38–300 Gorlice, Poland, tel: +48 18 35 53 415, e-mail: kukla_piotr@poczta.onet.pl

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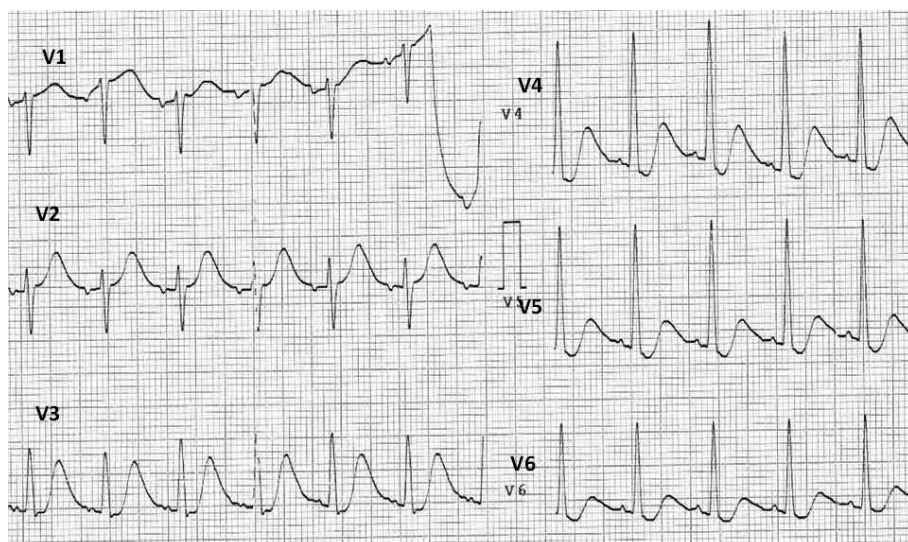


Figure 2. ECG recorded 24 h after admission (see details in the text). Broad, positive T-waves in leads V_2 – V_5 . Note, the ‘slow’ descending arm of the T-wave (prolonged $T_{\text{peak}}-T_{\text{end}}$ at 160 ms)

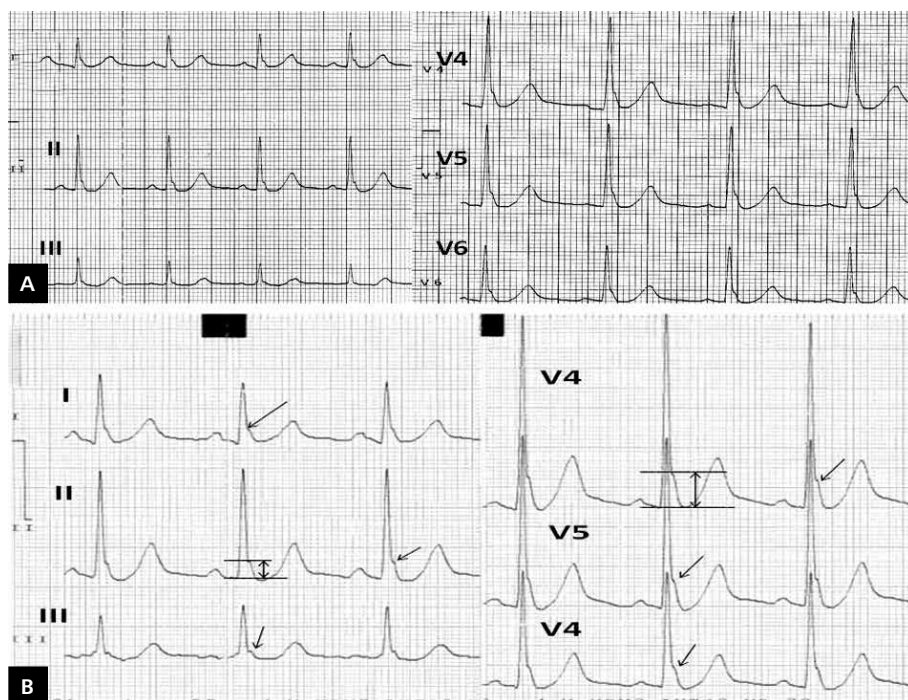


Figure 3. A. ECG recorded on the second day of admission. Leads I–III, V_4 – V_6 . The J-wave can be seen (see details in the text); **B.** The same ECG as in Figure 3A but recorded with double amplitude. Note the J-waves (arrows)

diffuse ST-segment depression in leads I, II, III, aVF and V_2 – V_6 (maximum depression: -2.5 mm in lead V_5), with ST-segment elevation in lead aVR and prolonged $T_{\text{peak}}-T_{\text{end}}$ interval (Fig. 1). Echocardiogram performed on admission revealed no wall motion abnormalities. Due to persistent headache, elevated blood pressure, and normal echocardiography in spite of diffuse ST-segment depression changes, a head

computed tomography (CT) scan was performed. This scan showed acute SAH. ECG recorded 24 h after admission presented broad, positive T-waves in leads V_2 – V_5 and prolonged $T_{\text{peak}}-T_{\text{end}}$ interval to 160 ms (Fig. 2). A later ECG recorded on the second day showed sinus rhythm with ST-segment normalisation. Additionally, new J-waves were observed in all leads, being negative in leads aVR and V_1 , and positive in the

rest of the leads (Figs. 3A, B). The maximum amplitude of the J-wave was 2 mm in the limb leads (lead II) and 4 mm in the precordial leads (lead V₄) (Fig. 3B). No dangerous ventricular arrhythmias were observed. Diffuse ST-segment depression similar to the one observed in acute coronary syndromes can occur in patients with ACVD [8]. ECG changes can be dynamic involving also T-wave inversion and QT prolongation.

The above mentioned ECG changes, in a patient with acute and persistent headache, should raise a concern about possible ACVD.

Conflict of interest: none declared

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