Traumatic subdural haematoma with electrocardiographic abnormalities simulating myocardial infarction

A.J.H.H.M. van Oostrom, H.W. Mauser, E.F.D. Wever

A 58-year-old man with a history of alcohol abuse and smoking presented with a subdural haematoma due to head trauma after alcohol intoxication. He was disorientated; general and specific cardiological and neurological examinations were unremarkable. Cardiac troponin T (0.053 µg/l), N-terminal pro-B type natriuretic peptide (768 pg/ml) and serum ethanol (3316 mg/l) were elevated. Creatinine kinase (21 U/l) and lactate dehydrogenase (214 U/l) were normal. Other liver function tests and mean erythrocyte cellular volume suggested chronic alcohol abuse. CT scan of the head demonstrated a right-sided subdural haematoma and contusional lesions in the right frontal lobe. At day 2, the haematoma and contusional lesions had slightly increased, without further deterioration at follow-up. His ECG showed a sinus tachycardia with a left axis, left anterior fascicular block and Q waves in the right precordial leads. Follow-up showed reversible diffuse repolarisation abnormalities, suggesting evolving myocardial infarction. Echocardiography at day 16 showed normal dimensions without regional wall motion disturbances or significant valve abnormalities. Myocardial perfusion imaging after three months showed normal left ventricular (LV) function without ischaemic changes. Therefore, the described Q waves were most likely secondary to the axis deviation and the repolarisation changes were ascribed to the traumatic intracranial lesions and not to coronary artery disease or alcoholic cardiomyopathy. Neurogenic cardiac dysfunction due to subdural haematoma has not been extensively investigated, but it is likely to be similar to

Figure 1. Serial ECGs (left panel) after a traumatic subdural hematoma (CT scan of the brain is shown in the right panel).

that seen in subarachnoid haemorrhage (SAH). In the latter, reversible left ventricular dysfunction, troponin release and ECG abnormalities have been observed. In 8 to 15% of these patients a diminished LV ejection fraction (LVEF <50%) was shown; however, cardiogenic shock rarely occurred.^{1,2} Wall motion disorders usually occurred in the first days after SAH, did not match coronary artery distribution and generally showed spontaneous improvement.¹ Troponin T release has been demonstrated in 20 to 40% of SAH patients and was positively correlated with the severity of neurological symptoms.^{2,3} ECG abnormalities have been shown in 50 to 100% of SAH patients and mostly consisted of ST-T changes, QT prolongation and U wave abnormalities, whereas rhythm and conductance

A.J.H.H.M. van Oostrom

E.F.D. Wever

Department of Cardiology, St Antonius Hospital, Nieuwegein, the Netherlands

H.W. Mauser

Department of Neurology, St Antonius Hospital, Nieuwegein, the Netherlands

Correspondence to: A.J.H.H.M. van Oostrom
Department of Cardiology, St Antonius Hospital,
PO Box 2500, 3430 EM, Nieuwegein, the Netherlands
E-mail: a.van.oostrom@antonius.net

disturbances were less frequent.⁴⁻⁶ It is thought that improvement in LVEF parallels normalisation of the ECG. Raised intracranial pressure with stimulation of the hypothalamus resulting in sympathetic activation with catecholamine release is the proposed mechanism of neurogenic cardiac dysfunction.^{6,7} Histological examination of the myocardium showed contraction band necrosis and myocytolysis.^{7,8} After brain injury, cardiac involvement may affect outcome, but the major causes of death are neurological and infectious disease.⁶ Specific cardiac treatment should be supportive in case of LV dysfunction and arrhythmia. Furthermore, differentiating myocardial infarction from reversible neurogenic cardiac dysfunction is critical for early neurosurgical intervention and a potential heart donor procedure. ■

References

- 1 Banki N, Kopelnik A, Tung P, Lawton MT, Gress D, Drew B, et al. Prospective analysis of prevalence, distribution, and rate of recovery of left ventricular systolic dysfunction in patients with subarachnoid hemorrhage. *J Neurosurg.* 2006;105:15-20.
- 2 Bulsara KR, McGirt MJ, Liao L, Villavicencio AT, Borel C, Alexander MJ, et al. Use of the peak troponin value to differentiate myocardial infarction from reversible neurogenic left ventricular dysfunction associated with aneurysmal subarachnoid hemorrhage. *J Neurosurg.* 2003;98:524-8.
- 3 Naidech AM, Kreiter KT, Janjua N, Ostapkovich ND, Parra A, Commichau C, et al. Cardiac troponin elevation, cardiovascular morbidity, and outcome after subarachnoid hemorrhage. *Circulation.* 2005;112:2851-6.
- 4 Brouwers PJ, Wijdicks EF, Hasan D, Vermeulen M, Wever EF, Frericks H, et al. Serial electrocardiographic recording in aneurysmal subarachnoid hemorrhage. *Stroke.* 1989;20:1162-7.
- 5 Lanzino G, Kongable GL, Kassell NF. Electrocardiographic abnormalities after nontraumatic subarachnoid hemorrhage. *J Neurosurg Anesthesiol.* 1994;6:156-62.
- 6 Sakr YL, Ghosn I, Vincent JL. Cardiac manifestations after subarachnoid hemorrhage: a systematic review of the literature. *Prog Cardiovasc Dis.* 2002;45:67-80.
- 7 White M, Wiechmann RJ, Roden RL, Hagan MB, Wollmering MM, Port JD, et al. Cardiac beta-adrenergic neuroeffector systems in acute myocardial dysfunction related to brain injury. Evidence for catecholamine-mediated myocardial damage. *Circulation.* 1995;92:2183-9.
- 8 Kolin A, Norris JW. Myocardial damage from acute cerebral lesions. *Stroke.* 1984;15:990-3.

In this section a remarkable 'image' is presented and a short comment is given.

We invite you to send in images (in triplicate) with a short comment (one page at the most) to Bohn Stafleu van Loghum, PO Box 246, 3990 GA Houten, e-mail: l.meester@bsl.nl.

'Moving images' are also welcomed and (after acceptance) will be published as a Web Site Feature and shown on our website: www.cardiologie.nl

This section is edited by M.J.M. Cramer and J.J. Bax.