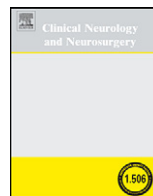




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Letter to the Editor

Intracerebral hemorrhage following epinephrine application for anaphylactic reaction

Keywords:

Intracerebral hemorrhage
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Dear Sir,

Primary, non traumatic intracerebral hemorrhage results from the rupture of small vessels mostly damaged by chronic hypertension or amyloid angiopathy [1]. Commonly, it occurs in the basal ganglia, thalamus, brain stem, cerebellum and the cerebral lobes. Secondary intracerebral hemorrhage is induced by vascular abnormalities or impaired coagulation. Chronic hypertension is the most important risk factor for spontaneous intracerebral hemorrhage [2]. Recent studies speculate that acute hypertension may contribute to intracerebral hemorrhage [3,4]. A few patients were reported to have suffered hemorrhagic stroke in association with the use of over-the-counter sympathomimetics like phenylpropanolamine, pseudoephedrine and epinephrine [5,6]. Rarely, intracerebral hemorrhage occurred after intravenous administration of epinephrine [7] or therapeutic upper-GI endoscopy with application of epinephrine [8,9]. An 8-year-old boy developed intracerebral hemorrhage after local infiltration of epinephrine during adenotonsillectomy [10].

We report a 41 year old woman who suffered intracerebral hemorrhage after epinephrine application due to anaphylactic reaction. The patient was unaware of any medical co-morbidity except for an allergy without known triggering agent. Acute urticaria and paroxysmal dyspnea had prompted the call of an emergency physician, who saw the woman in reduced general condition and found systolic blood pressure of 60 mmHg. At this time the patient showed no neurological symptoms. Under the suspected diagnosis of an anaphylactic reaction, he administered 40 mg dexamethasone and 5 mg epinephrine in 500 ml Ringer solution and admitted her to our emergency department. During the epinephrine application, the patient developed tachycardia, headache and a left sided hemiparesis. Blood pressure was not documented during transport. On clinical examination in hospital, the patient was awake and orientated to all qualities. Speech was fluid without deficit. Cranial nerve status showed a left sided hypesthesia and hypalgesia. The rest of cranial nerve status was normal. Motor function test revealed a left sided hemiparesis (arm MRC 3 (medical research council) and leg MRC 4). Sign of Babinski was positive on the left side. Further examination showed a hypesthesia and hypalgesia of the left side. Coordinative functions revealed a left sided paretic ataxia. CT scan of the brain on admission showed an intracerebral hemorrhage in the right basal ganglia area with a

size of 2 cm × 3 cm (Fig. 1). MRI (day 3) showed the intracerebral hemorrhage without any signs for intracerebral tumor or small vessel disease (Fig. 2a–d). MR angiography was normal, without hints for vascular malformations. No abnormalities of cardiac or pulmonary function were found. ECG and chest X-ray were unremarkable. Extracranial ultrasound and ultrasound of both renal arteries were normal. Transthoracic echocardiography was unremarkable, without signs of chronic hypertensive changes and with a normal ejection fraction. Continually taken blood pressure values were normotensive throughout hospital stay and remained normal without antihypertensive therapy during the following weeks. Laboratory tests showed slightly elevated troponin at admission with 0.06 µg/l (normal <0.03), that renormalized the following day. In addition, there was a leucocytosis with 11.5/nl (normal 4.5–11.0) with normalization after 1 day. The rest of laboratory tests including serum creatinine, urea, electrolytes, CK, CK-MB, hemoglobin, erythrocytes, hematocrit, thrombocytes, liver enzymes, TSH and coagulation parameters (PTT and INR) were normal. Urine analysis was unremarkable.

In our patient, cerebral imaging showed a typically located hypertensive intracerebral hemorrhage in the basal ganglia. History and additional diagnostic work-up revealed no hints of chronic hypertension. In CT scan and MRI of the brain, including MR angiography, no evidence was seen for vascular abnormalities or intracerebral tumor. Although it cannot be entirely excluded that another pathogenetic mechanism causes the intracerebral hemor-

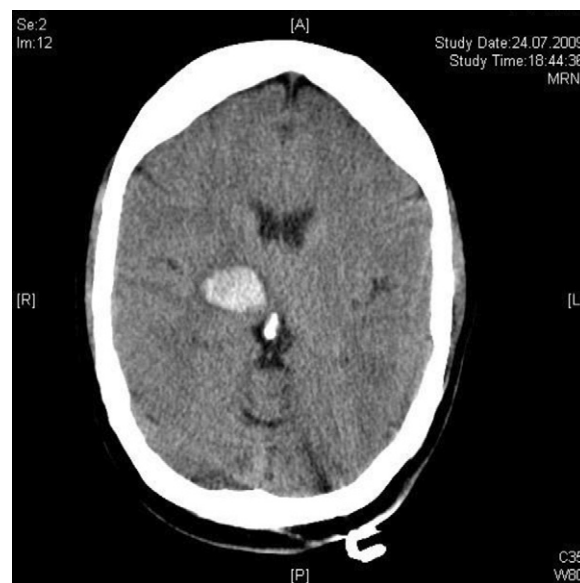


Fig. 1. CT scan of the brain shows the intracerebral hemorrhage in the right basal ganglia area.

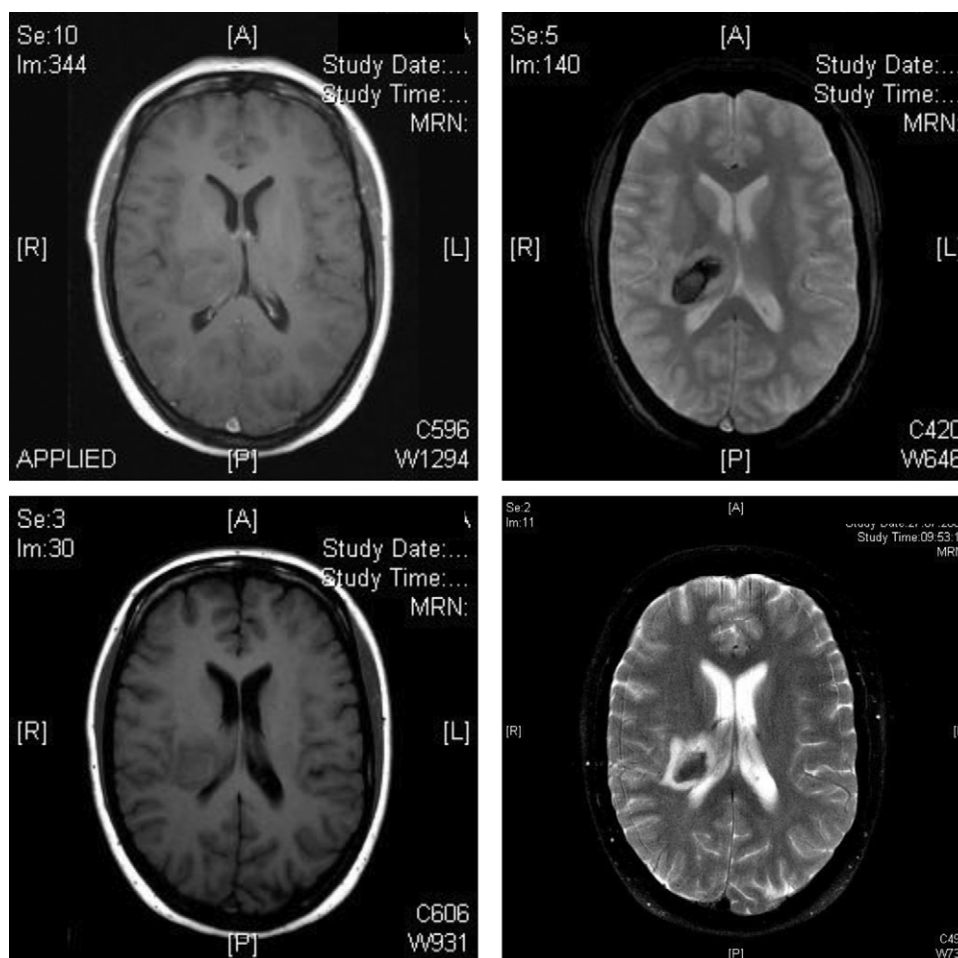


Fig. 2. (a–d) MRI shows the intracerebral hemorrhage without any signs for intracerebral tumor or vascular leukoencephalopathy.

rhage, the close temporal association with epinephrine application suggested a hypertensive etiology. This hypothesis was supported by the initial elevated troponin, which can be interpreted as an acute hypertensive cardiomyopathy. Mostly, primary intracerebral hemorrhage results from chronic hypertension. This report suggests that intracerebral hemorrhage can also result from acute hypertension, without a chronic damage of small vessels of the brain.

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