Intern Emerg Med (2017) 12:1071–1073
DOI 10.1007/s11739-017-1604-1

CE - MEDICAL ILLUSTRATION

Fluid levels in the bleeding brain: a marker for coagulopathy and hematoma expansion

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Received: 22 December 2016 / Accepted: 6 January 2017 / Published online: 13 January 2017
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Case

A 72-year-old man with baseline vascular dementia was admitted for 3 days of confusion, unintelligible speech, and incontinence. His past medical history was notable for a right middle cerebral artery territory infarction 4 years prior, coronary artery disease treated with angioplasty and stenting, hypertension, and diabetes. Upon evaluation in our emergency department, his examination was notable for expressive aphasia and confusion. He was moving all extremities spontaneously against gravity. A noncontrast head CT was performed that showed a subacute infarct in the left frontal lobe, subsequently confirmed on MRI (panel A). Since he was outside the window for thrombolysis and intervention, he was given one full-dose aspirin (325 mg tablet), and admitted to our hospital for a stroke evaluation.

During the course of his stroke work-up, he was found to have an apical left ventricular thrombus, presumably the etiology of his subacute left frontal lobe infarct. Given his increased risk of further ischemic strokes, he was started on anticoagulation 7 days after his stroke using intravenous heparin and then switched to an oral vitamin K antagonist (warfarin). While awaiting transfer to a rehabilitation facility on post-stroke day 14, he was found in a stuporous state. He was not following commands, and was only minimally arousable to noxious stimuli. An emergent head CT was performed, which showed hemorrhagic conversion of the left frontal lobe infarct with fluid levels (panel B). A radial arterial line was placed for blood pressure monitoring, and he was started on a nicardipine drip. Four units of fresh frozen plasma and intravenous vitamin K were administered enroute to the intensive care unit (ICU). While in the ICU, he was intubated and started on hypertonic saline for cerebral edema. On a repeat CT scan 6 h later, there was interval expansion of his left temporal hematoma (panel B). After a family discussion, he was made comfort measures only, and no aggressive measures were pursued (Fig. 1).

Discussion

Nontraumatic intraparenchymal hemorrhage in older patients is usually caused by hypertension, with cerebral amyloid angiopathy also a notable cause in normotensive individuals [1]. Most intracerebral hematomas that expand do so within the first 24 h, with the majority occurring in the first 6 h of the initial bleed [2]. On noncontrast head CT, fluid levels within the hematoma are associated with coagulopathy and an increased risk of expansion [3]. In the study by Pfleger et al., 22 out of 35 intracerebral hemorrhages in patients with a coagulopathy and an abnormally elevated prothrombin time or international normalized ratio (PT/INR) had blood-fluid levels, while only three blood-fluid levels were seen in 197 intracerebral hemorrhages in patients with a normal PT/INR [3]. This suggests that fluid levels in intracerebral hemorrhage are strongly associated with coagulopathy, but may rarely be seen in patients with normal coagulation parameters, probably reflecting the fact that the PT/INR does not capture all patients with coagulopathy. Higher intensity anticoagulation with increasing

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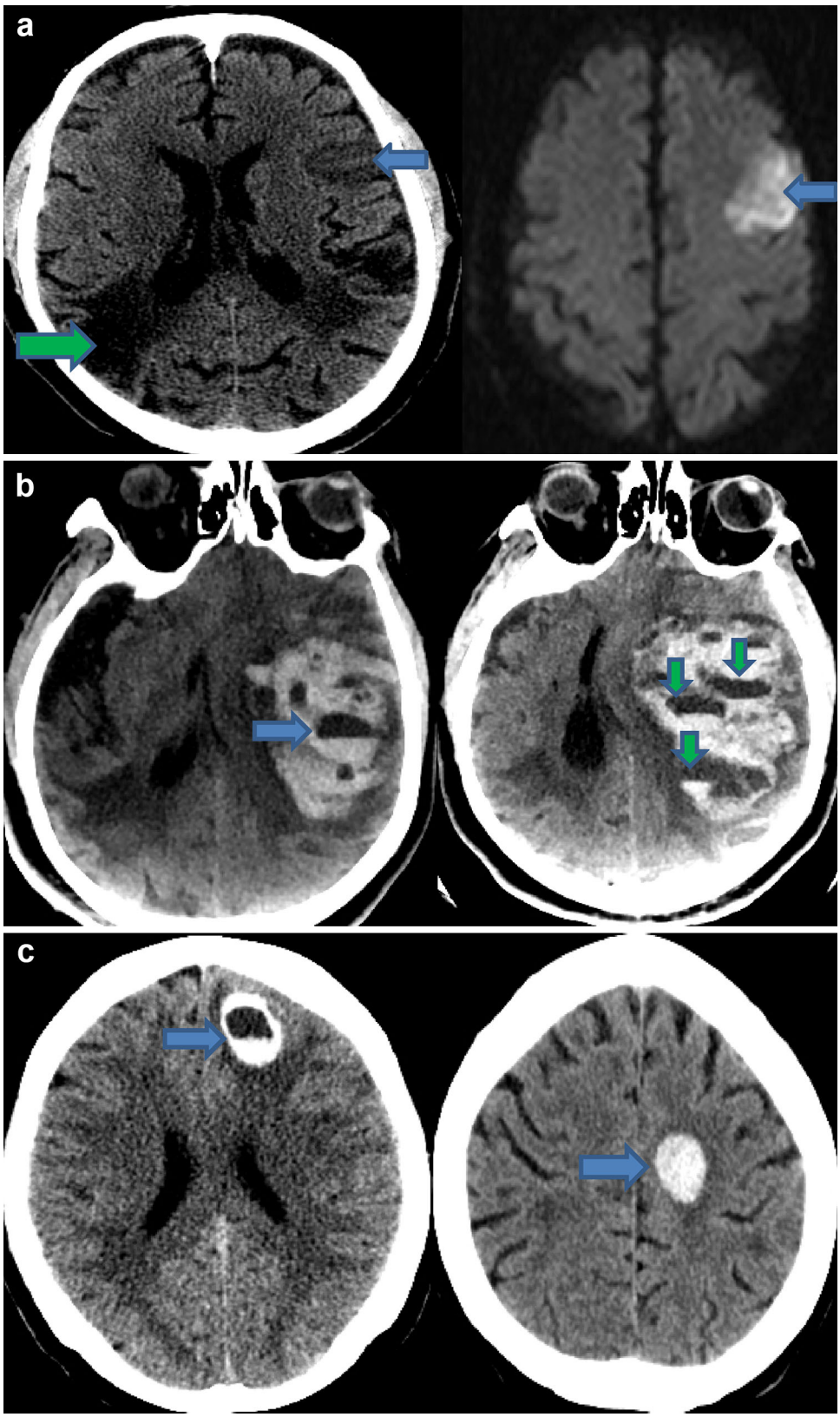

Fig. 1 Panel **a** first image is a noncontrast CT showing a subacute infarct in the left frontal operculum (*blue arrow*). Corresponding diffusion-weighted MR image at the same level confirms the subacute left frontal lobe infarct. An old infarct is also present in the right posterior temporal lobe (*green arrow*). Panel **b** first image in panel B is a noncontrast head CT of the same patient as in panel A, showing new hemorrhagic conversion in the left middle cerebral artery stroke bed, with at least one fluid level (*blue arrow*). Repeat noncontrast head CT 6 h later shows interval growth of the hematoma with multiple fluid levels (*green arrows*). Panel **c** first image in panel **c** is a 68-year-old woman with atrial fibrillation on warfarin, with a left frontal lobe hematoma and a fluid level (*blue arrow*). Her international normalized ratio (INR) was 3.4 at the time of imaging. The second image is a comparison case from a patient with normal coagulation parameters (INR 1.04) and a similar left frontal lobe hematoma (*blue arrow*). The second image represents the typical appearance of an intraparenchymal hematoma in a patient without coagulopathy (color figure online)

PT/INR is associated with a higher likelihood of blood-fluid levels, but many patients with fluid levels may only have mild to moderate PT/INR elevation [3]. As the coagulopathy is reversed, the bleeding stabilizes, fluid levels disappear, and the hematoma is eventually resorbed. Fluid levels in intracerebral hemorrhage have also been

reported in patients receiving fibrinolysis after acute myocardial infarction [4]. In patients with anticoagulant-associated intraparenchymal hemorrhage, a careful history of anticoagulant use should be elicited, coagulation parameters checked, and reversal agents administered promptly to stabilize hematoma size and promote clotting [5].

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Statement of human and animal rights All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards

Informed consent Informed consent was obtained from all participants included in this study.

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