Diffuse cerebral oedema mimicking subarachnoid haemorrhage on computed tomography

Introduction

Computed tomography (CT) appearances of subarachnoid haemorrhage (SAH) on unenhanced scans are characteristic and include high density of the CSF spaces. There are several mimics including meningitis, diffuse cerebral oedema and intrathecal or high dose intravenous contrast media injection, described by the term pseudo-subarachnoid haemorrhage.

This case report describes the CT appearances in a patient with diffuse cerebral oedema mimicking SAH.

Discussion

SAH is a medical emergency and a CT scan is routinely performed when there is a clinical suspicion of SAH. Increased attenuation of the subarachnoid spaces and the dura are characteristic findings on unenhanced CT.

However, these imaging finding have also been described in diffuse cerebral oedema, meningitis and following intra-thecal contrast administration (Spiegel et al, 1986; Mendelsohn et al, 1994; Eckel et al, 1998; al-Yamany et al, 1999; Given et al, 2003).

Diffuse cerebral oedema is associated with increased intracranial pressure. This leads to relative displacement of low density CSF within the subarachnoid spaces. There is also dilatation and engorgement of the superficial venous channels resulting in increased attenuation of the subarachnoid spaces (Opeskin and Silberstein, 1998; Given et al, 2003). The attenuation of the adjacent brain parenchyma is decreased as a result of oedema, which further accentuates this appearance.

Given et al (2003) were able to measure the attenuation values within the basal cisterns in three patients. The attenuation value of the basal cisterns (29–33 HU) was lower than expected for SAH (60–70 HU) Pooling of contrast media within the engorged pial vessels has been noted by several other authors. This supports the theory that the CT appearance in diffuse cerebral oedema is mainly the result of vascular engorgement (Opeskin and Silberstein, 1998). Enhancement of the subarachnoid spaces following intravenous contrast administration can be a useful method to confirm the diagnosis on imaging.

Case Report

A 62-year-old man with a history of petrol consumption was admitted to the accident and emergency department of an adjacent district general hospital by paramedics. He complained of headache and difficulty in breathing. On admission his Glasgow Coma Scale was 10/15 which rapidly deteriorated to 4/15. An unenhanced computed tomography (CT) scan was performed immediately which showed obliteration of basal cisterns consistent with brainstem oedema. He was mechanically ventilated with inotropic support and then transferred to the intensive care unit of the authors' institution. Relevant past history included alcohol abuse, manic depressive psychosis and carbamazepine overdose.

Following transfer, sedation was withdrawn and the patient reassessed. He had fixed dilated pupils, no respiratory effort, absent gag reflex and response to pain. He was also hypernatraemic with a serum sodium level of 170 mmol/litre (normal range 133–144 mmol/litre). Toxicology for paracetamol and salicylates was negative.

A repeat unenhanced CT scan was performed at this stage. This showed high attenuation in the tentorium (*Figure 1a*), basal cisterns (*Figures 1b* and *c*) and in the sylvian fissures (*Figure 1d*). There was no history of injection of intravascular or intrathecal iodinated contrast media. This was initially interpreted as subarachnoid haemorrhage. On subsequent review, the diagnosis was changed to diffuse cerebral oedema.

In view of a failing brainstem and after discussion with the patient's family, treatment was withdrawn. A post mortem examination performed by the coroner showed no evidence of subarachnoid haemorrhage.

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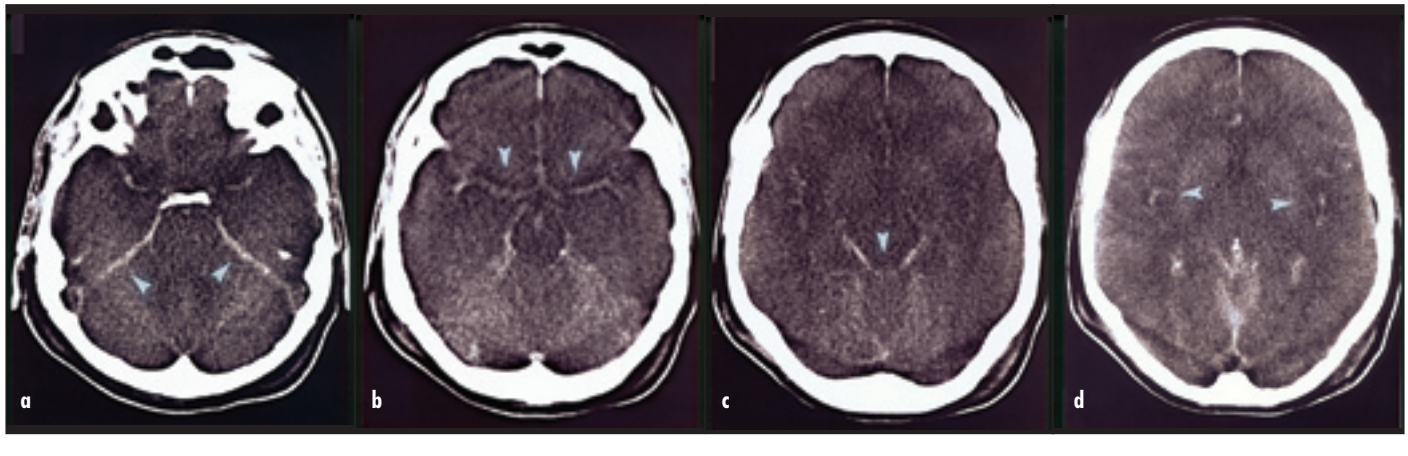
Figure 1. Unenhanced computed tomography of the brain showing increased density in (a) the tentorium (arrow), (b and c) in the basal cisterns (arrows) and (d) in the sylvian fissures (arrows). Window width 80, window level 40.



In patients with meningitis, the breakdown of the blood–brain barrier and leak of proteinaceous CSF into the subarachnoid spaces results in increased attenuation on CT. The presence of cerebral oedema exaggerates this finding. However, significant imaging changes caused by increased CSF proteins are only seen in very severe cases of meningitis (Mendelsohn et al, 1994).

This case report highlights the presence of mimics of SAH on unenhanced CT of which clinicians should be aware of and prevent more invasive tests being carried out on these critically ill patients. BJHM

al-Yamany M, Deck J, Bernstein M (1999) Pseudo-subarachnoid haemorrhage: A rare neuroimaging pitfall. *Can J Neurol Sci* **26:** 57–9

Eckel TS, Breiter SN, Monsein LH (1998) Subarachnoid contrast enhancement after spinal angiography mimicking diffuse subarachnoid haemorrhage. *Am J Roentgenol* **170:** 503–5

Given CA 2nd, Burdette JH, Elster AD, Williams DW 3rd (2003) Pseudo-subarachnoid haemorrhage: a potential imaging pitfall associated with diffuse cerebral oedema. *Am J Neuroradiol* **24:** 254–6

Mendelsohn DB, Moss ML, Chason DP, Muphree S, Casey S (1994) Acute purulent leptomeningitis mimicking subarachnoid haemorrhage on CT. *J Comput Assist Tomogr* **18:** 126–8

Opeskin K, Silberstein M (1998) False-positive diagnosis of subarachnoid haemorrhage on computed tomography scan. *J Clin Neurosci* **5:** 382–6

Spiegel SM, Fox AJ, Vinuela F, Pelz DM (1986) Increased density of tentorium and falx: a false positive CT sign of subarachnoid haemorrhage. *Can Assoc Radiol J* **37:** 243–7

Giant rectal villous adenoma presenting with hypokalaemia

A 64-year-old man was admitted to hospital following a collapse. He had a history of fatigue and muscle weakness over the last 6 months. He also complained of diarrhoea associated with the passage of large amounts of mucus. General examination was unremarkable. Laboratory tests revealed the presence of hypokalaemia. A computed tomography scan revealed a markedly distended rectum with a large enhancing mass present within suggestive of a giant villous adenoma. Colonoscopy and biopsy confirmed the diagnosis. Following correction of the patient's hypokalaemia, the mass was removed surgically. A 10 cm giant villous adenoma was confirmed at histology and the patient made a full recovery.

Adenomatous polyps are, by definition, neoplastic. They can be either sessile or pedunculated (Bond, 2001). Adenomas are divided into three subtypes: tubular, tubulovillous, and villous. Villous adenomas are encountered least frequently, accounting for only 5% of all adenomas. Villous adenomas may be large in size and are associated more often with dysplasia. Villous adenomas have a risk of malignant transformation of between 15 and 25% (Bond, 1993). Although they may occur anywhere within the colon, they occur more frequently in the rectum and rectosigmoid than elsewhere. They generally are sessile structures that appear as velvety or cauliflower-like projections (Zauber and Winawer, 1997) as in this case. The most common presenting symptom is with bleeding or anaemia. Other symptoms include diarrhoea, constipation and flatulence (O'Brien et al, 1990). Villous adenomas rarely cause a secretory diarrhoea syndrome as in this case. In such cases, the tumour usually is located at the rectosigmoid or rectum and is often at least 3–4 cm in diameter. Stool volumes of 350–3000 ml have been reported leading to hypovolaemia and metabolic imbalances such as hypokalaemia.

Treatment is by surgical removal following electrolyte correction. BJHM

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Bond JH (1993) Polyp guideline: diagnosis, treatment, and surveillance for patients with nonfamilial colorectal polyps. The Practice Parameters Committee of the American College of Gastroenterology. *Ann Intern Med* **15**(119): 836–43

Bond JH (2001) Colon polyps and cancer. *Endoscopy* **33:** 46–54

O'Brien MJ, Winawer SJ, Zauber AG (1990) The National Polyp Study. Patient and polyp characteristics associated with high-grade dysplasia in colorectal adenomas. *Gastroenterology* **98:** 371–9

Zauber AG, Winawer SJ (1997) Initial management and follow-up surveillance of patients with colorectal adenomas. *Gastroenterol Clin North Am* **26:** 85–101

Figure 1. Axial contrast-enhanced computed tomography scan through the lower pelvis reveals a markedly distended rectum (arrow) with an enhancing frond-like mass in keeping with a giant villous adenoma.