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NEURO-IMAGES

Contrast-induced encephalopathy after abdominal CT examination

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Introduction

Contrast-induced encephalopathy (CIE) is a rare, acute and usually reversible neurological complication that occurs during or after intra-arterial administration of iodinated contrast medium (ICM) [1, 2]. CIE has been reported following various angiographic procedures, mainly coronary or cerebrovascular angiography whilst our patient developed this complication after intravenous and oral ICM administration [1, 2].

Case presentation

A 75-year-old man underwent duodenocephalopancreasectomy to remove an ampulloma. Ten days after surgery, an abdominal CT was performed to assess the presence of a pancreatic fistula. Blood tests revealed elevation of serum creatinine (1.9 mg/dl), azotaemia (112 mg/dl), glycaemia (137 mg/dl), GGT (114 U/L), LDH (289 U/L), RCP (41.3 mg/l), iron (250 ug/dl), WBC ($31.85 \times 10\text{^}3$/ul) and decrease of RBC ($4.3 \times 10\text{^}6$/uL); other laboratory findings were unremarkable. Were intravenously administered 120 mL of ICM (1.5 ml/Kg, Iopamiro 370 mg I/ml) followed by a saline flush; contextually, an oral water-based solution (50 mL, 1:1) of contrast medium (Gastrografin, 370 mg I/ml) was given. After CT study, the patient received intravenous hydration. Within 6 h of receiving ICM, he developed sudden and severe muscle weakness with complete loss of muscle strength of the lower face, arm, and leg on the left side (hemiplegia) associated with disorientation in time and space.

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Unenhanced head CT scan was urgently performed and showed a slight cortical–subcortical hyperdensity of the right rolandic gyrus which appeared swollen compressing the adjacent subarachnoid spaces (Fig. 1a). The morning after, neurological disorder was no longer evident and blood tests documented reduction of serum creatinine (1.2 mg/dl), azotaemia (102 mg/dl) and glycaemia (89 mg/dl). Then, control unenhanced head CT—performed 16 h after symptoms onset—resulted negative (Fig. 1b). The patient was discharged 1 month later without neurological symptoms after the resolution of the abdominal complication.

Discussion

CIE has been associated with various neurological presentations including cortical blindness, seizure, encephalopathy, focal neurological deficits and even a chemical meningitis from extravasation of ICM. Symptoms and neurological deficits occur early after contrast administration and typically are reversible, resolving within 24–48 h; symptoms lasting longer have also been observed while fatal encephalopathy has been rarely reported [1, 3–8].

CIE's pathogenesis can be explained on the basis of temporary disruption of the blood–brain barrier (BBB) by hyperosmolarity and chemotoxicity of ICM resulting in cerebral vasogenic oedema [1, 8, 9].

ICM can cause CIE dependently of their osmolarity or ionic states and there is no limit dose of ICM related to CIE, however larger doses are considered a risk factor [1].

Predisposing factors seem to be chronic hypertension, renal failure, history of stroke and impaired cerebral autoregulation [1, 3].

In our patient, renal impaired function may have reduced the clearance of ICM, which may have exacerbated the accumulated osmolality and neurotoxicity of contrast. Moreover, his disease states may have increased permeability of the BBB allowing ICM enter the brain more easily.

CT imaging may demonstrate diffuse cortical hyperattenuation similar to subarachnoid haemorrhage that, despite our

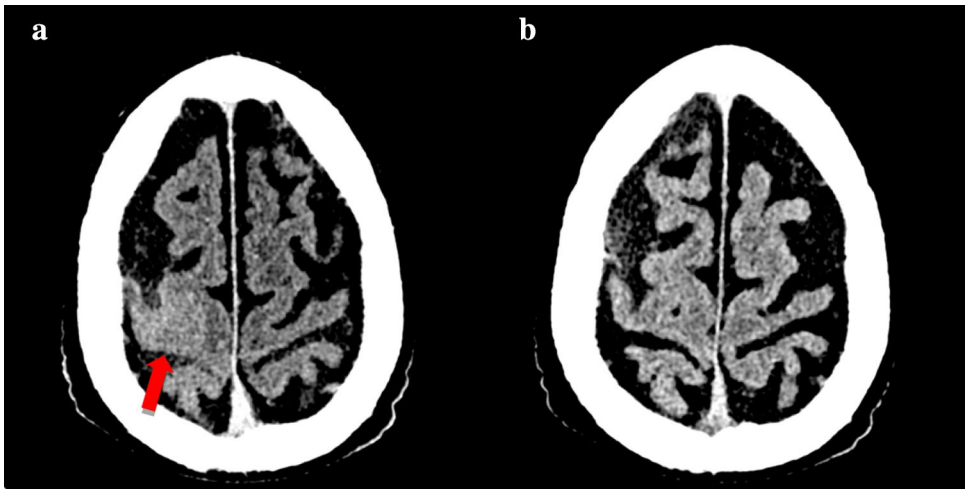

Fig. 1 Axial CT image depicting slight hyperdensity of the right Rolandic gyrus (**a**) no longer visible on control CT (**b**)

case, most commonly affects the parieto-occipital cortex; normal brain CT has also been described [3]. As a limitation of our case report, brain MRI was not performed given the resolution of the finding at control CT. Brain MRI frequently reveals the presence of vasogenic oedema with gyral swelling and increased signal on T2-weighted and FLAIR sequences, with increased intensity on DWI sequences and ADC maps [5].

Therapy is generally supportive, involving intravenous hydration while anticonvulsants can be used in cases of seizures [3].

Declaration

Conflict of interest Authors declare no conflict of interest.

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