

Non-ionic contrast media neurotoxicity mimicking intracerebral hematoma

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Received: 9 August 2010 / Accepted: 13 August 2010 / Published online: 26 August 2010
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Dear Editor,

Neurotoxic events with contrast media are well known after angiography. In some cases, contrast media can destroy the blood–brain barrier (BBB) and extravasate to interstitial tissue. It can mimic subarachnoid hemorrhage (SAH) and also intraparenchymal hematoma both clinically and radiologically [1, 4, 5, 8]. Most of the time, this is a transient, local effect, and all symptoms will resolve as soon as contrast media excreted with glomerular filtration.

We are presenting a case who suffered non-ionic contrast media neurotoxicity after percutaneous coronary intervention (PCI). The clinical and radiological findings were mimicking SAH, intraparenchymal hematoma, and basal ganglial hematoma with disappearance of radiological findings and improvement of neurological deficits in 6 h.

Case report

A 69-year-old man with acute coronary syndrome underwent PCI. He started on 300 mg of acetylsalicylic acid, 600 mg of clopidogrel, and 70 U/kg of heparin sulfate. During PCI, 100 ml of iohexol, non-ionic contrast media, was used intraarterially. The patient suffered an acute headache with nausea and vomiting 30 min after PCI. On his neurological

examination after deterioration, he was stuporous and disoriented. His Glasgow Coma Scale was 11/15. He had hemiplegia on his left side. Computed tomography (CT) scan without contrast agent demonstrated diffuse hyperdense lesion in the right frontoparietal region and two hyperdense lesions located in the right parietal lobe and the right basal ganglia (Fig. 1a, b). CT was reported as intraparenchymal hematoma associated with SAH. Due to suspicion of intracranial hemorrhage, the initial treatment was stopped.

Surprisingly, after 6 h, the patient became awake. His neurological examination was returned totally normal. Control CT scan of the head obtained after improvement did not demonstrate any intracranial pathology and reported as normal (Fig. 1c, d). We reanalyzed the first CT scan and found the densities of lesions described was 140 to 160 Hounsfield units (HU), exceeding the values for blood (40–60 HU), which is compatible with contrast media. The patient was discharged after 3 days with full recovery.

Discussion

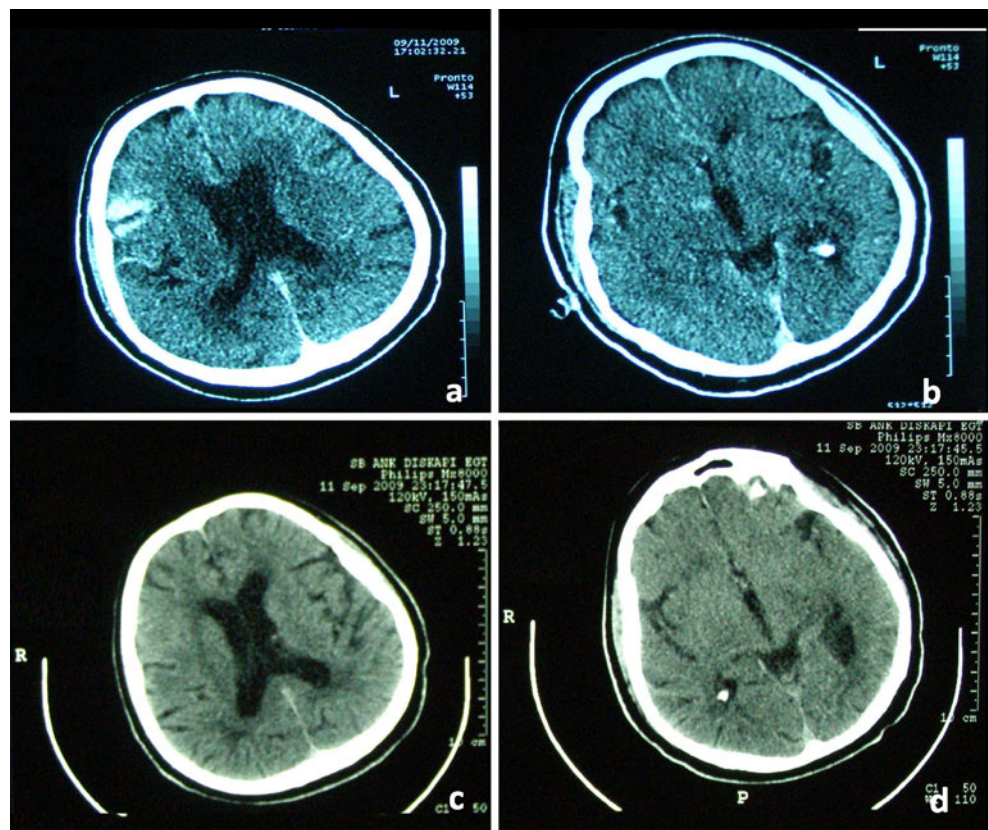
Dramatic shift in fluid due to the hyperosmolality of the contrast agents with efflux of water, shrinkage of cells, and opening of tight junctions causes the increase in the permeability of BBB [3]. Extravasation of contrast media to interstitial space causes direct stimulation and excitation of neural cells and can result to neurotoxicity [6, 7].

In the presented case, the patient who underwent PCI suffered from a rapid deterioration of neurological condition. We obtained a CT scan as urgently as possible, and CT scan demonstrated Fischer grade 4 SAH. Hence, we stopped all anti-aggregant and anticoagulant therapy which are extremely vital for the patient with acute coronary syndrome. After a while, the patient became neurologically and radiologically

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Fig. 1 **a, b** Cranial CT scan revealing diffuse hyperdense lesion in the right frontoparietal region and two hyperdense lesions located in the right parietal lobe and the basal ganglia. **c, d** Control CT scan of head obtained after 6 h later revealing no hyperdense lesions or intracranial pathology



normal. Misdiagnosis of intracranial hemorrhage caused an inappropriate withdrawal of anti-aggregant and anticoagulant treatment. Exact diagnosis is very important in such cases because the patient may suffer from important morbidity or even mortality by the withdrawal of accurate therapy. Radiodiagnostic modality, especially the density of lesion, can be very beneficial for differential diagnosis between contrast media extravasation and intracranial hemorrhage; while blood has a density range between 40 and 60 HU, contrast media have higher attenuation values [1]. The clinical course of patient can also be helpful to physicians. Rapid clinical and radiologic recovery is not expected if the patient suffers from SAH. A small amount of SAH can disappear within 24 h in CT scan [2]. On the other hand, both neurotoxic effects and radiologic manifestations of contrast agents recover and disappear as soon as renal clearance of contrast media is completed.

In conclusion, there is an important diagnostic dilemma between intracerebral hemorrhage and contrast neurotoxicity; each diagnosis has different treatment options. Misdiagnosis can cause the death of patient. Therefore, a high degree of suspicion and exact diagnosis based on radiologic evidences, especially density values, are very critical for accurate diagnosis.

Conflicts of interest None.

References

1. Chattopadhyay S, Srinivasan M, Thomas P (2008) Postangiographic contrast enhancement mimicking acute subdural hemorrhage in a patient with severe occipital headache and neurological symptoms: a case report. *J Med Case Reports* 23:119
2. Hayman LA, Pagani JJ, Kirkpatrick JB, Hinck VC (1989) Pathophysiology of acute intracerebral and subarachnoid hemorrhage: applications to MR imaging. *AJR Am J Roentgenol* 153:135–139
3. Junck L, Marshall WH (1983) Neurotoxicity of radiological contrast agents. *Ann Neurol* 13:469–484
4. Ko DY (2000) Contrast agent neurotoxicity presenting as subarachnoid hemorrhage. *Neurology* 54:1014–1015
5. Korn-Lubetzki I, Rosenmann D, Steiner-Birmanns B (2008) Reaction to intravenous contrast media mimicking intracerebral hemorrhage after percutaneous coronary intervention. *Med Sci Monit* 14:142–144
6. May EF, Ling GS, Geyer CA, Jabbari B (1993) Contrast agent overdose causing brain retention of contrast, seizures and parkinsonism. *Neurology* 43:836–838
7. Oftedal SI, Kayed K (1973) Epileptogenic effect of water-soluble contrast media. An experimental investigation in rabbits. *Acta Radiol Suppl* 335:45–56
8. Sharp S, Stone J, Beach R (1999) Contrast agent neurotoxicity presenting as subarachnoid hemorrhage. *Neurology* 52:1503–1505