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Case Report

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Acute encephalopathy associated with anaphylactic shock caused by angiographic radiocontrast media: An unusual case

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Abstract

Radiocontrast media have been used with increasing frequency for centuries. Radiocontrast media may have the spectrum up to anaphylaxis as a side effect. The case presented here is of a patient with acute encephalopathy, associated with the anaphylactic shock of angiographic radiocontrast media. A 67-year old male patient with a diagnosis of abdominal aortic aneurysm (AAA) was referred for coronary and peripheral angiographic evaluation before the AAA operation. After the angiographic examination was completed, the patient developed complaints of dyspnea, stridor, flushing and eruption. The patient recovered totally after a successful medical intervention was performed for anaphylaxis. Decorticated posture gradually developed, and the patient lost consciousness again. After cranial computed tomography, diffusion magnetic resonance imaging and electroencephalography were performed, levetiracetam treatment was started to the patient after the patient had a tonic-clonic convulsion. The patient recovered completely after a seizure with antiepileptic treatment without. Nonconvulsive status epilepticus must be considered after the allergic reaction, and anti-epileptic agents should be taken into consideration in addition to preventing hypoxia and hypoperfusion.

Keywords: radiocontrast media, anaphylaxis, hypoxia, nonconvulsive status epilepticus

1. Introduction

The first iodinated radiocontrast media (IRM) was introduced in 1929, and IRMs are today used in >50 million radioscopic examinations per year (1). IRMs can be classified into two groups, ionic and non-ionic. These are both structurally based on tri-iodinated benzene rings. Non-ionic monomers have much lower osmolality. Hyper osmolality is known to have a role in the pathogenesis of physiological reactions. Hypersensitivity reactions (HR) can be classified as 1) allergic-like, 2) pseudoallergic, and 3) anaphylactoid reactions (1).

Because of mast cell activation in anaphylactic reactions, cerebral hypoperfusion due to vasoconstriction in brain vessels (Kounis-like syndrome) or directly caused by systemic hypotension can cause brain disorders and encephalopathy (2).

The case presented here is of a patient with acute encephalopathy, which could not be detected with imaging methods, associated with the anaphylactic shock of angiographic radiocontrast media.

2. Case Report

A 67-year old male patient with a diagnosis of abdominal aortic aneurysm (AAA) was referred for coronary and peripheral angiographic evaluation before the AAA operation. We performed abdominal computed tomography angiography (CTA) for AAA diagnosis. There was no allergic reaction during this CTA, and we detected the diameter of the infrarenal abdominal aorta was 45 mm, increased in compliance with aneurysmatic enlargement, and we observed mural thrombus areas reaching a thickness of 20 mm observed on CTA (Fig. 1 a-b).

With radial artery access, coronary angiography indicated non-critical coronary arterial disease and 70-80% lesion on bilateral superficial femoral arteries. We detected an infrarenal AAA (Fig. 1c-d) on peripheral angiography. Both CTA and coronary angiography examinations were performed with the iohexol which was a non-ionic low-osmolar IRM. After completing the angiographic examination, the patient was taken to the recovery room and followed for bleeding. The patient developed complaints of dyspnea, stridor, flushing and eruption. The patient lost consciousness during this time. We urgently administered methylprednisolone 40 mg + diphenhydramine 50 mg + adrenaline 1 gr intra-venosus to the patient, who was then admitted to the intensive care unit. The patient's aspiration and consciousness improved. There was no motor sensorial deficit, but decorticated posture gradually developed, and the patient lost consciousness again.

An anaesthetist and neurologist examined the patient, and respiratory support was provided. The patient underwent cerebral CT (CCT) at 6-hour intervals, but we observed no pathology other than chronic ischaemic changes (Fig. 2a). There was no pathology on diffusion magnetic resonance

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imaging (DMRI) (Fig. 2b). As there was no ischaemic or hemorrhagic lesion on CCT or DMRI, we performed electroencephalography (EEG) for non-convulsive status epilepticus. We determined moderate-heavy ground rhythm pathology on EEG (Fig. 2c).

Almost 24 hours after the angiography, the patient had a tonic-clonic convulsion detected by a doctor. After this convulsion, we started levetiracetam treatment as the recommendation of the neurology clinic. On the post-convulsion control DMRI and flair sequences, we observed cortical diffusion restriction at the vertex level of the frontoparietal in the right hemisphere, which could represent a postictal signal change (Fig. 2d).

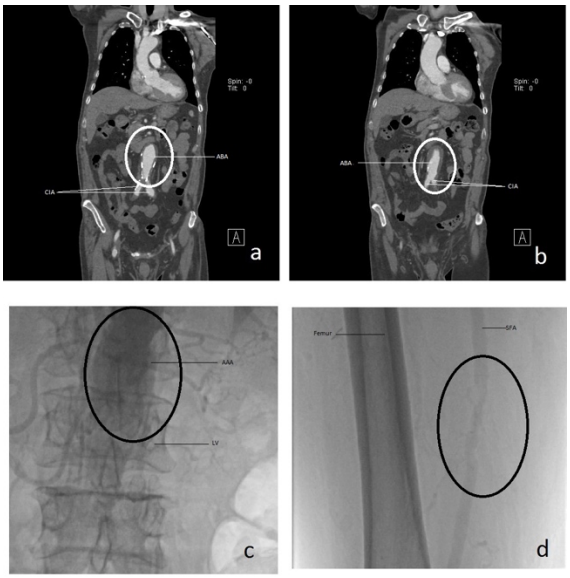

Fig. 1. Aneurysmatic dilatation and atherosclerotic calcification detected on CT(a-b), and conventional angiography (c), 70-80% peripheral lesion detected on conventional angiography (d). (ABA: Abdominal aorta, CIA: Common iliac artery, SFA: superficial femoral artery, LV: lumbar vertebra)

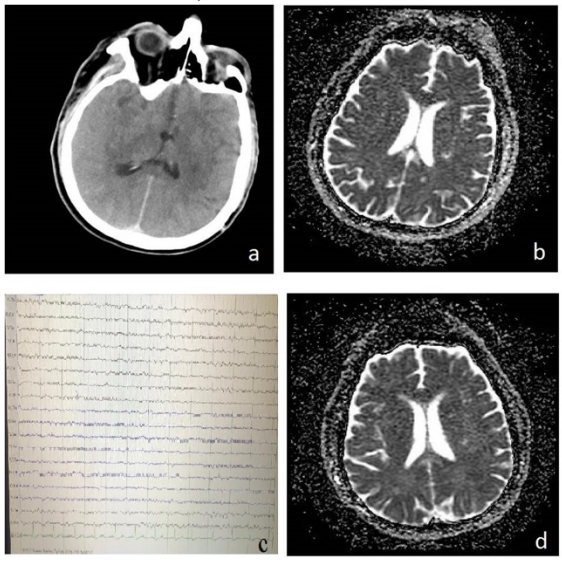

Fig. 2. No acute pathology on CCT (a) and DMRI (b), but there was moderate-heavy ground rhythm pathology on EEG (c), after convulsion, cortical diffusion restriction was observed at the vertex level of the frontoparietal in the right hemisphere, which could represent a postictal signal change (d)

The decorticated posture and unconsciousness improved after 6 hours of levetiracetam treatment. Although quadri-paresis continued, the patient responded to verbal stimuli. The quadri-paresis improved at 72 hours after angiography.

3. Discussion

Hyper sensitivity reactions (HR) are serious allergic reactions, which may be a response to any substance and sometimes lead to death due to anaphylactic shock and systemic hypoperfusion. HRs due to IRM are not dependent on the dose of IRM. HRs can be classified as acute and late phase reactions (1).

Symptoms include urticaria, erythema, bronchospasm, and shock. The action on first encountering a substance may result in sensation and lead to a weak-reaction, which can then result in a serious acute-reaction on the second encounter with the same substance. IRM-activated mast cells and basophils, complementary and kinin systems mediate all of these reactions (2).

HR is a systemic disorder due to anaphylaxis, hypotension and systemic perfusion and can affect all organs. Although rare, encephalopathy is the most threatening complication. The reasons for brain injury in HRs can be cerebral hypoxia caused by hypoperfusion because of anaphylactic shock or cerebral hypoxia caused by local allergic vasculitis secondary to HR (3).

Kounis syndrome (KS) is associated with HR, caused by acute myocardial injury (4). Furthermore, Kounis-like syndrome (KLS) has been described for other organs, meaning that this vascular phenomenon is not restricted to coronary injuries. As ischemia-sensitive organs, cerebral arteries and mesenteric arteries can be affected by KLS (5,6). There may be ischemic lesions or findings on MRI or CCT in KLS in relation to the duration of ischemia.

Reports in the literature about encephalopathy after HR and anaphylaxis include papers by Peláez et al. with the association of amoxicillin-clavulanic acid (7), Ding et al. presented formocresol (3), Speach et al. hymenoptera venom (8), Schabitz et al. diclofenac (9), and Arishima et al. an insect bite (10). In these cases, ischaemic lesions were present after the events on imaging tests.

The current case differs from the literature as there was no ischaemic lesion on CT or MRI, but there were ischaemic findings on EEG, and clinical improvement was obtained after anti-convulsive therapy after a convulsion, which may have been due to this clinic being nonconvulsive status epilepticus after a KLS.

The pathogenesis in this case may be associated with temporary vasoconstriction without sequelae because of early intervention for anaphylaxis and hypotension, and to KLS of the cerebral vessels. This situation with no-ischaemia on CT or MRI may have been due to nonconvulsive status epilepticus, and this situation can be masked by the heavy ground rhythm

pathology on EEG.

Even when a patient has previously been exposed to a drug with no problems, the allergic side-effects of each drug should be considered for each repeated procedure. In some cases, there may be an unexplained loss of consciousness with non-diagnostic imaging tests or EEG findings can be masked by hypoxia. In such a situation, nonconvulsive status epilepticus must be considered after the allergic reaction, and anti-epileptic agents should be taken into consideration in addition to preventing hypoxia and hypoperfusion.

Informed Consent

Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

Conflict of Interest

The authors declared no conflict of interest.

Acknowledgments

None to declare.

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