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Intracranial contrast enhancement mimicking intracranial hemorrhage after coronary angiography

Wewnątrzczaszkowe wzmocnienie kontrastowe naśladujące krwotok śródczaszkowy po koronarografii

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Abstract

A 34-year-old male patient was diagnosed with acute coronary syndrome and referred to our clinic for coronary angiography. After a complex percutaneous coronary intervention he developed non-sustained neurologic symptoms compatible with intracranial hemorrhage. Follow-up cranial images revealed rapid clearance of the hyperdensity involvements, consistent with direct neurotoxicity of the contrast agent. The patient's symptoms resolved and he was discharged without any neurologic sequalea

Key words: contrast agent, neurotoxicity, coronary angiogram

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Case report

A 34-year-old male patient was diagnosed with acute coronary syndrome and referred to our clinic for coronary angiography. Baseline cardiac examination on admission was normal and electrocardiography revealed ST segment depressions in the inferolateral leads. Transthoracic echocardiography revealed a regional hypokinesia in the right coronary artery (RCA) territory with an estimated left ventricular ejection fraction of 50%. Laboratory tests were normal expect an elevated level of troponin I (0.4, reference 0.000–0.016 ng/mL). Clopidogrel, acetylsalicylic acid, beta-blocker, statin, angiotensin-converting enzyme, nitrate and heparin was initiated. Coronary angiography revealed a significant atherosclerotic stenoses in the proximal and mid segment of RCA. It was decided to perform percutaneous coronary intervention. After post-dilatation with a non-compliant balloon TIMI 2 flow was observed and intracoronary bolus of tirofiban followed by intravenous infusion was administered. During this complex procedure we approximately injected 400 mL of iohexol (Omnipaque®, GE Healthcare Co., Ireland Cork, Ireland) and 15,000 IU of unfractionated heparin. After 4 hours, the patient complained of drowsiness, severe headache, nausea, and vomiting. The patient's pupil reflexes were prompt and isocoric, blood pressure was 100/60 mm Hg and heart rate was 80 beats per minute. Complete blood count revealed a hemoglobin level of 14.7 g/dL and platelet count of 233,000/μL platelets. The prothrombin time (international normalized ratio) was 1.28, the activated partial thromboplastin time was 140.3 seconds, serum creatinine was 0.89 mg/dL, aspartate aminotransferase and alanine aminotransferase were 26 U/L and 50 U/L respectively. Emergent non-contrast brain computed tomography (CT) was performed. Diffuse intracranial hyperdensity in the transverse sinuses and dura along the

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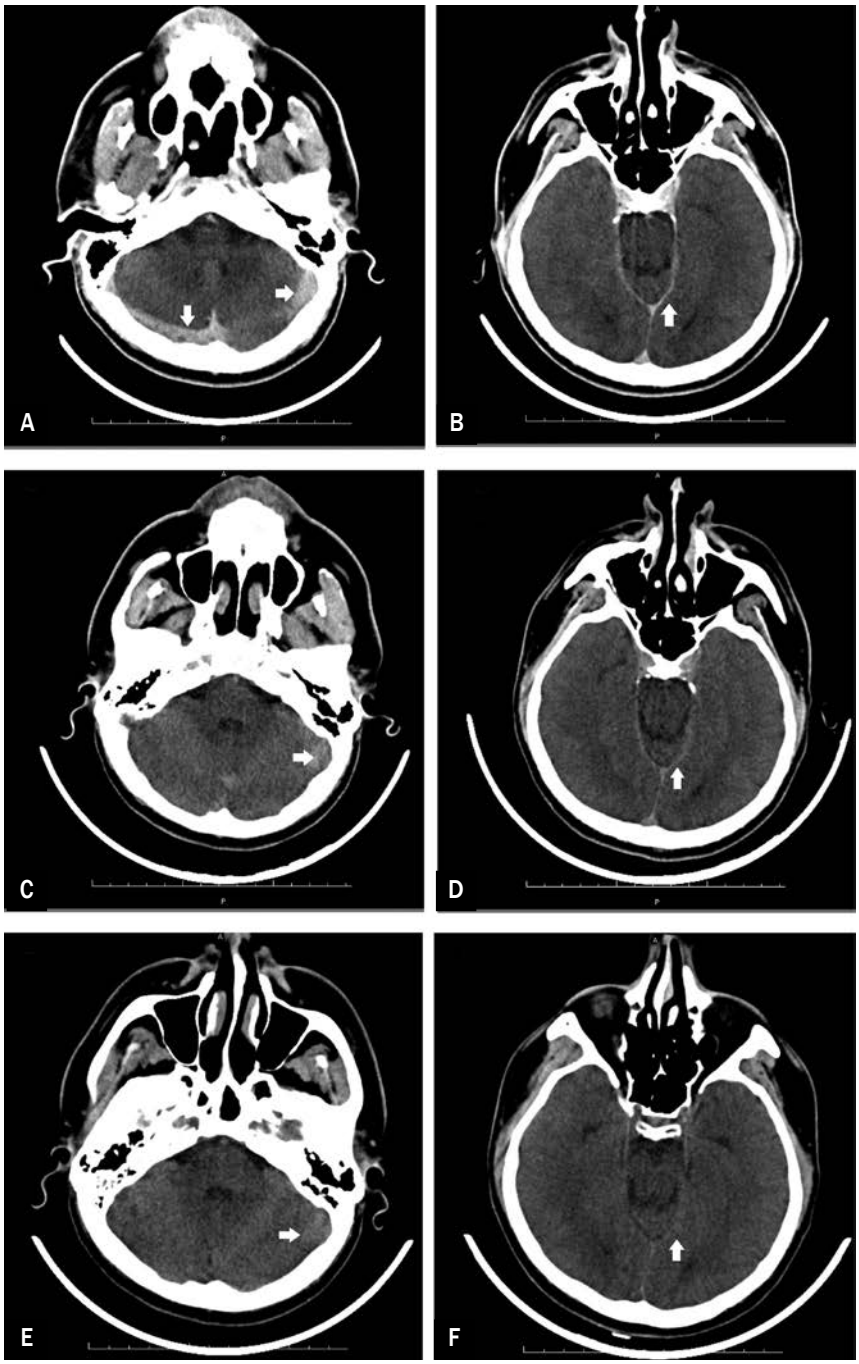

Figure 1A, B. Non-contrast brain computed tomography demonstrating diffuse intracranial hyperdensity in the transverse sinuses and dura (arrows) (**C, D**). Non-contrast brain computed tomography at the 4th hour demonstrating apparent reduction in the hyperdense involvements (arrows) (**E, F**). Non-contrast brain computed tomography at the 24th hour demonstrating almost total clearance of the contrast agent and near normal brain computed tomography (arrows)

tentorium was observed (Figure 1A, B). We immediately discontinued acetylsalicylic acid, clopidogrel and tirofiban, and administered intravenous sodium valproate, mannitol and protamine sulfate. Neurology and neurosurgery consultations were requested. After 4 hours the patient's symptoms were completely recovered. Control non-contrast brain CTs at the 4th and 24th hours showed a decrease in the hyperdense involvements (Figure 1C–F). Acetylsalicylic acid and clopidogrel were reinitiated and patient was discharged on the 4th day without any neurologic or cardiac sequela. On the 1-month visit, he was free of any complaints and brain CT was totally normal.

Contrast-induced neurotoxity (CIN), a very rare complication of contrast agents with an incidence of 0.06% [1]. It

can sometimes mimic an acute intracranial hemorrhage. It is a transient and rapidly reversible entity [2]. Contrast--induced neurotoxity especially occurs when ionic, and high osmolar agents are used [3]. In general, non-ionic contrast agents have a lower incidence of neurotoxic events than ionic contrast agents. Non-ionic contrast agents, such as iodixanol, can cause transient cortical blindness, confusion and amnesia without focal deficits, whereas ionic contrast agents have been reported to cause more severe complications, including seizures and motor and speech deficits. Although it is hard to discriminate between contrast agent leakage associated neurotoxity and intracranial hemorrhage clinically, hyperdense signal on both T1- and T2-weighted magnetic resonance imaging can be helpful in the diagnosis of intracranial hemorrhage. In this case non-sustained neurologic symptoms and rapid clearance of the hyperdensity on follow-up images were consistent with direct neurotoxicity of contrast agent. To avoid such adverse effect, contrast agent dosage and concentration should not exceed those recommended by the manufacturer for coronary angiography.

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Conflicts of interest

There are no conflicts of interest.

Streszczenie

Mężczyznę w wieku 34 lat z rozpoznaniem ostrego zespołu wieńcowego skierowano do ośrodka autora w celu wykonania koronarografii. Po złożonej przezskórnej interwencji wieńcowej u chorego wystąpiły objawy neurologiczne sugerujące krwotok wewnątrzczaszkowy. Kontrolne obrazowanie czaszki ujawniło szybką eliminację ognisk hiperdensyjnych świadczącą o bezpośredniej toksyczności środka cieniującego. Objawy ustąpiły całkowicie bez żadnych powikłań neurologicznych i chorego wypisano ze szpitala.

Słowa kluczowe: środek kontrastowy, neurotoksyczność, angiogram wieńcowy

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