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Acute Thrombocytopenia After Infusion of an Iodinated Contrast Medium: A Case Report

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Abstract

lodinated contrast medium, administered during several diagnostic tests may be frequent cause of anaphylactic reactions or rashes. Thrombocytopenia after injection of iodinated contrast medium is a most rare adverse event, but several published reports have shown that this complication is more common than previously thought. In patients with acute drug induced thrombocytopenia, antibody tests are often negative. Thrombocytopenia due to iodinated contrast medium is transient, but the patient is made sensitive to the drug; further examinations should therefore always be performed in the absence of iodinated contrast medium.

Keywords: Thrombocytopenia; Iodinated contrast medium; Platelet disorders

Case Report

Iodinated Contrast Medium (ICM), administered during several diagnostic tests such as angiography, angioCT or CT scan, may be frequent cause of anaphylactic reactions or rashes [1-3]. Thrombocytopenia after injection of ICM is a most rare adverse event, but some studies have reported it [4-8]. Several hypotheses have been suggested about the mechanism that induces this syndrome, but none was confirmed such as the real cause of thrombocytopenia. Patient with a history of drug-induced thrombocytopenia may show antibody tests negative [4-9]. We report a case of two subsequent episodes of severe thrombocytopenia due to ICM.

A 73-year-old man with bilateral carotid stenosis, severe right hip osteoarthritis and iliac atheromasia was admitted to our hospital for evaluation of Aortic Abdominal Aneurysm (AAA). At the time of hospitalization the medications were telmisartan associated with hydrochlorothiazide, rosuvastatin, indobufene and oxycodone. Laboratory data included a platelet count of 214×103 µL, INR and aPTT in range. The day after admission, the patient underwent an angioCT with a previous injection of 20 ml iodinated contrast medium, immediately post ICM infusion the patient presented acute epigastric pain and mild cyanosis spontaneously resolved after few minutes. His platelet count was 28×10³ μL, decreased to 7×10³ μL six hours later, but both, INR and aPTT, remained within normal limits. No major or minor bleedings were found. On the basis of these findings, the physicians excluded a Disseminated Intravascular Coagulation (DIC). No other causes of thrombocytopenia were evident: the patient was not treated with heparin and the other drugs, he assumed, could not be correlated with a decrease in platelets. Only the administration of the ICM could therefore have caused this acute thrombocytopenia. His platelet count increased spontaneously and reached 181×10³ μL by the fifth day after procedure. Subsequent checks showed that platelet value has always been within normal range.

Four years before, the patient had a similar episode of thrombocytopenia attributed to ICM infusion. Still in that case, the patient was admitted to hospital for a diagnostic evaluation of AAA. CT scan was performed with and without ICM infusion, but immediately after ICM injection the patient presented an abdominal pain, followed by a severe decrease of platelet count, from $208\times10^3\,\mu\text{L}$ at admission, to $3\times10^3\,\mu\text{L}$. His platelet count increased quickly after corticosteroid therapy and platelet infusion and remains within normal range, until the new recent event. Following this first episode, corticosteroids and

anti-histamines were administered still in the last case, but they were not sufficient to prevent thrombocytopenia.

Discussion

Thrombocytopenia was generally considered a non common adverse event following injection of iodinated contrast medium, but several published reports have shown that this complication is more common than it was previously thought [4-7].

Our patient developed two episodes of abdominal pain and severe thrombocytopenia immediately after ICM injection. In the first case the patient was immediately treated with corticosteroids and transfused with platelets to prevent the risk of intracranial or intrapulmonary haemorrhage, the event was resolved within a short time. Following this previous experience, corticosteroids are still administered before the last radiographic procedure, but they were not sufficient to avoid acute thrombocytopenia. If thrombocytopenia is drug-induced, such in our case, there are no evidences about the real efficacy of corticosteroids [4-9]. In the past, few cases of platelet decrease due to disseminated intravascular coagulation after ICM injection have been reported [7-10], but in our case laboratory tests excluded it. The platelet count is in fact rapidly increased after discontinuation of the ICM infusion. The exact mechanism of acute thrombocytopenia due to contrast medium is still not entirely known. A rapid onset, followed by a proliferation of megakaryocytes in the marrow, indicate destruction or a consumption of peripheral platelets, these are some events that characterize the thrombocytopenia. Some drugs or chemical agents may cause an immune-mediated platelet destruction [7], but several hypotheses have been proposed such as mechanism that causes specific antibody induction: drug induced conformational change of surface glycoprotein IIb/IIIa, drug induced autoantibody reacting with autologous platelets,

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J Clin Case Rep ISSN: 2165-7920 JCCR, an open access journal soluble drug that induces a binding of a drug induced antibody to membrane protein, formation of immune complex mediated by PF4, chemical agent and specific antibody [4]. In patients with acute drug induced thrombocytopenia, antibody tests are often negative [4-9].

Thrombocytopenia due to iodinated contrast medium is transient, but the patient is made sensitive to the drug; further examinations should therefore always be performed in the absence of ICM.

Disclosures

The authors declare no conflict of interest relative to this paper.

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