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CASE REPORT

The Adverse Reactions to Contrast Media During Percutaneous Coronary Interventions; Keep in Mind the Non-idiosyncratic Reactions

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Abbreviation List
CCU: Coronary care unit
ICM: Iodinated contrast media
IV: Intravenous
LCx: Left circumflex artery
PCI: Percutaneous coronary intervention
RCA: Right coronary artery

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ABSTRACT

Iodinated contrast media are of paramount importance in the field of modern interventional cardiology. Although iodinated contrast media have an overall good safety profile, severe or life-threatening reactions can also occur. Herein, we report the case of a 74-year-old female patient who presented with non-ST elevation myocardial infarction and underwent a successful percutaneous coronary intervention. Shortly after the procedure the patient developed the full-blown clinical picture of an iodinated contrast media adverse reaction that was timely recognized and treated. The two types of adverse reactions to iodinated contrast media during a percutaneous coronary intervention are being discussed. A high level of clinical suspicion is warranted to ensure prompt recognition and appropriate management.

INTRODUCTION

Organic radiographic iodinated contrast media (ICM) have been among the most commonly used agents in the modern era of medicine and have become of paramount importance in the field of interventional cardiology. Although ICM have a good safety record, with usually mild and self-limited adverse effects, severe or life-threatening reactions can also occur. Therefore, physicians should be able to immediately recognize and treat the severe reactions of ICM.[1,2]

CASE REPORT

We present the case of a 74-year-old female patient who was admitted to our cardiology department due to non-ST elevation myocardial infarction. She had a history of diabetes mellitus type II, arterial hypertension and hyperlipidemia and had recently developed angina and dyspnea. On admission, she complained of chest pain.

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The electrocardiogram at admission revealed a normal sinus rhythm with ST depression (2.5mm) and negative T waves in the inferior leads. Her blood pressure and oxygen saturation were within normal range. High-sensitivity troponin was elevated (164.0pg/ml, normal range 0-34.2pg/ml) and the patient was loaded with aspirin and ticagrelor and transferred to the Cath lab. She underwent coronary angiography via the transradial approach, which revealed a culprit lesion in the mid segment of the right coronary artery (RCA, 95% stenosis) (Figure 1A), and a stenotic lesion in the circumflex artery (LCx, 90% stenosis in the mid segment) (Figure 2A). Crossover to the femoral approach had to be undertaken because of anatomic variation of the brachial artery. Both lesions were successfully treated by deploying everolimus drug eluting stents (2.75×18mm at the RCA and 2.75×26mm at the LCx) establishing at the end a TIMI 3 flow (Figures 1B, 2B). At this point, the patient complained of dizziness and a metallic taste in her mouth combined with sudden drop in systolic blood pressure (down to 55mmHg) and bradycardia (45bpm). Intravenous (IV) atropine was administered up to a total dose of 3 mg, however hypotension and bradycardia persisted. Due to the severity of what seemed to be a vasovagal reaction, we promptly reviewed our angiographic results to ensure that there was no complication and we administered IV dopamine (15mcg/kg/min) followed by epinephrine (1:10,000 solution, 5mcg/min). The persistence of the reaction and the metallic taste guided our thought to a possible reaction to the contrast media. Epinephrine was up-titrated (to 15mcg/min), and 125mg of hydrocortisone were also administered. Eventually, the patient was hemodynamically stabilized and transferred to our coronary care unit (CCU). In CCU, the patient's condition deteriorated further; generalized edema developed and a drop in her hemoglobin of 4mg/dl occurred with no apparent hemorrhage. She received one unit of concentrated red blood cells. The next day a CT abdominal scan was performed excluding a retroperitoneal hematoma. During the exam we observed a sparkling appearance of the small intestine and the gallbladder, attributable to the ICM. The edema gradually subsided with diuresis. The patient was discharged 8 days later with no further complications and a hemoglobin value of 11mg/dl (admission value 12mg/dl).

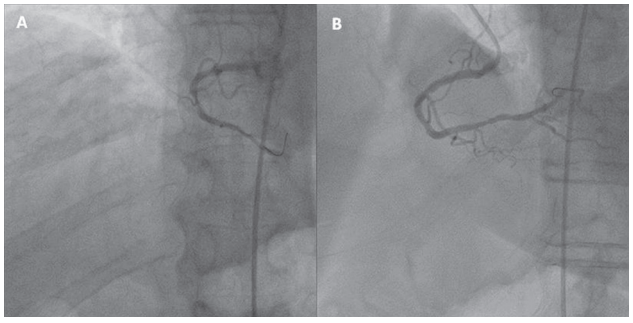

FIGURE 1. A: Coronary angiography of the RCA (left projection). B: RCA after stent deployment. RCA: Right coronary artery.

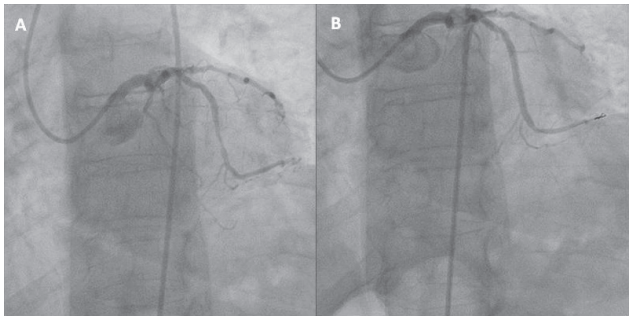

FIGURE 2. A: Coronary angiography of the LCx (right caudal projection). B: LCx after stent deployment. LCx: Left circumflex artery.

DISCUSSION

Adverse reactions to ICM are classified as idiosyncratic (also termed anaphylactoid) and non-idiosyncratic (also termed non-anaphylactoid or physiochemotoxic).[3] Idiosyncratic reactions typically occur within 20 minutes after the ICM injection, are dose-independent, have the same manifestations as anaphylactic reactions and can be classified as mild, moderate, and severe. Non-idiosyncratic reactions, on the other hand, cover a spectrum of cardiovascular reactions, such as bradycardia, low blood pressure, vasovagal reactions, arrhythmias or ischemia, and extend to non-cardiac manifestations, i.e. neuropathy, extravasation, nausea, and vomiting as well as delayed reactions.[4,5] Sensation of warmth and a metallic taste in mouth are indications of a non-idiosyncratic reaction to the contrast media.

Non-idiosyncratic cardiovascular reactions during a percutaneous coronary intervention (PCI) can be misinterpreted as a complication of the procedure per se or as a simple vasovagal reaction. Persistence of the symptoms, even though the procedure has been successfully completed, can be an alarming sign of an ICM adverse reaction.[6] Our patient developed a non-idiosyncratic reaction to ICM, manifested as a severe vasovagal reaction escalating to shock, generalized edema, and anemia. To our knowledge this is a rare report of a non-idiosyncratic reaction to ICM after a PCI.

Non-idiosyncratic reactions can manifest in various ways. ICM exerts a direct negative inotropic effect on the myocardium and can precipitate bradycardia and peripheral vasodilatation. Autonomic manifestations, such as nausea, vomiting, and diaphoresis, or mental status changes may also occur.[3] Untreated, these effects can lead to cardiovascular

collapse and death. Moreover, ICM can lower the threshold of ventricular arrhythmias and induce cardiac arrhythmias, provoke fluid shifts leading to an intravascular hypervolemic status and pulmonary edema, precipitate angina or lead to Kounis syndrome.[5]

The similarity of the cardiovascular and anaphylactic reactions to ICM can create perplexity in recognizing the true nature and severity of an adverse reaction, thus, leading to overtreatment or undertreatment of symptoms.[2-4] The aim of our case is to point out that ICM adverse reactions during a PCI, particularly non-idiosyncratic ones, are extremely rare and can go undetected or be misidentified as PCI related complications.[6] Physicians should keep in mind that what manifests as a simple vasovagal reaction can be a reaction to the contrast media, with prolonged hemodynamic compromise. On the other hand, acute hypotension or even shock is not always a complication of the PCI per se, but the cause may well be the contrast media. Therefore, familiarization with the presenting symptoms of ICM adverse reactions, vigilance, and prompt management are required.

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