

NSAIDS-induced anaphylaxis precipitating acute coronary vasospasm

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We report a case of NSAID-induced anaphylaxis precipitating coronary artery vasospasm leading to myocardial infarction in the presence of normal coronaries. Furthermore, we have also discussed the possible mechanisms for this. Attending clinicians should be aware of this potential complication when treating anaphylaxis, especially in patients with known or presumed coronary artery disease. *European Journal of Emergency Medicine* 15:48–50 © 2008 Wolters Kluwer Health | Lippincott Williams & Wilkins.

European Journal of Emergency Medicine 2008, 15:48–50

Keywords: anaphylaxis, coronary vasospasm, myocardial infarction, NSAIDS

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Received 17 August 2006 Accepted 10 September 2006

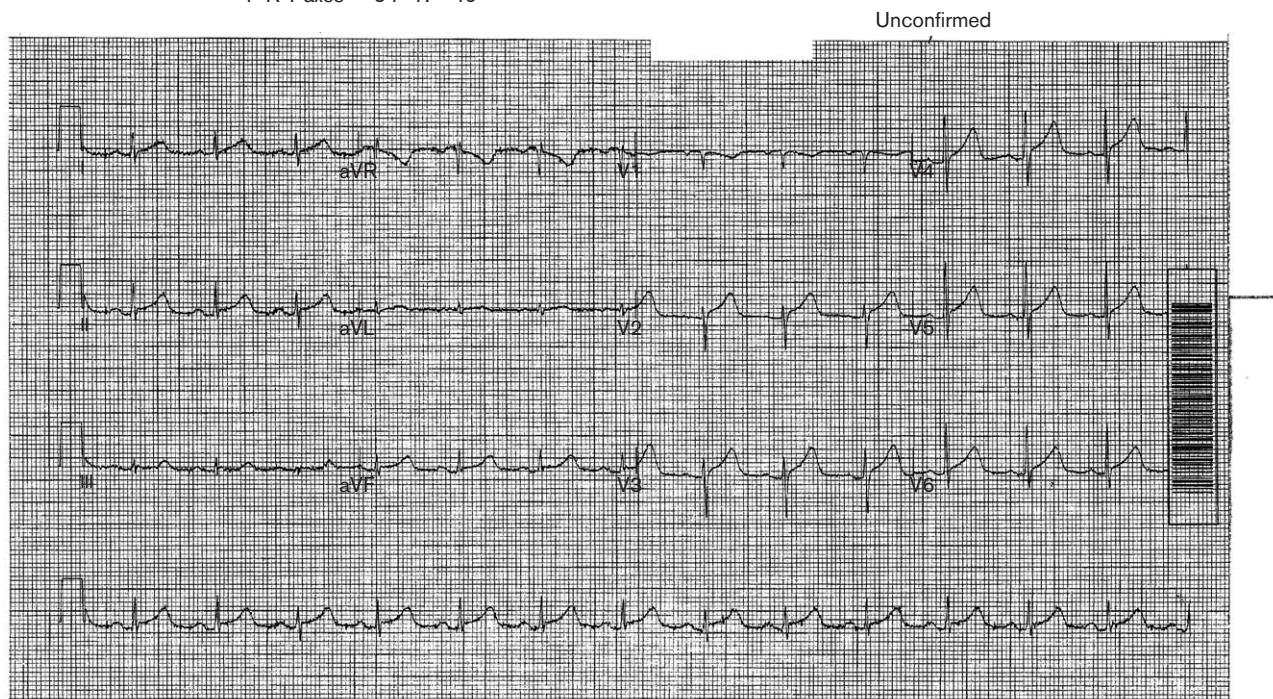
Introduction

Drug-induced anaphylaxis is a relatively common condition seen in most Emergency Departments (ED). We report a rare and potentially life threatening complication

of NSAID-induced anaphylaxis precipitating coronary vasospasm resulting in myocardial infarction in an individual with normal coronaries. We also discuss the possible mechanisms.

Fig. 1

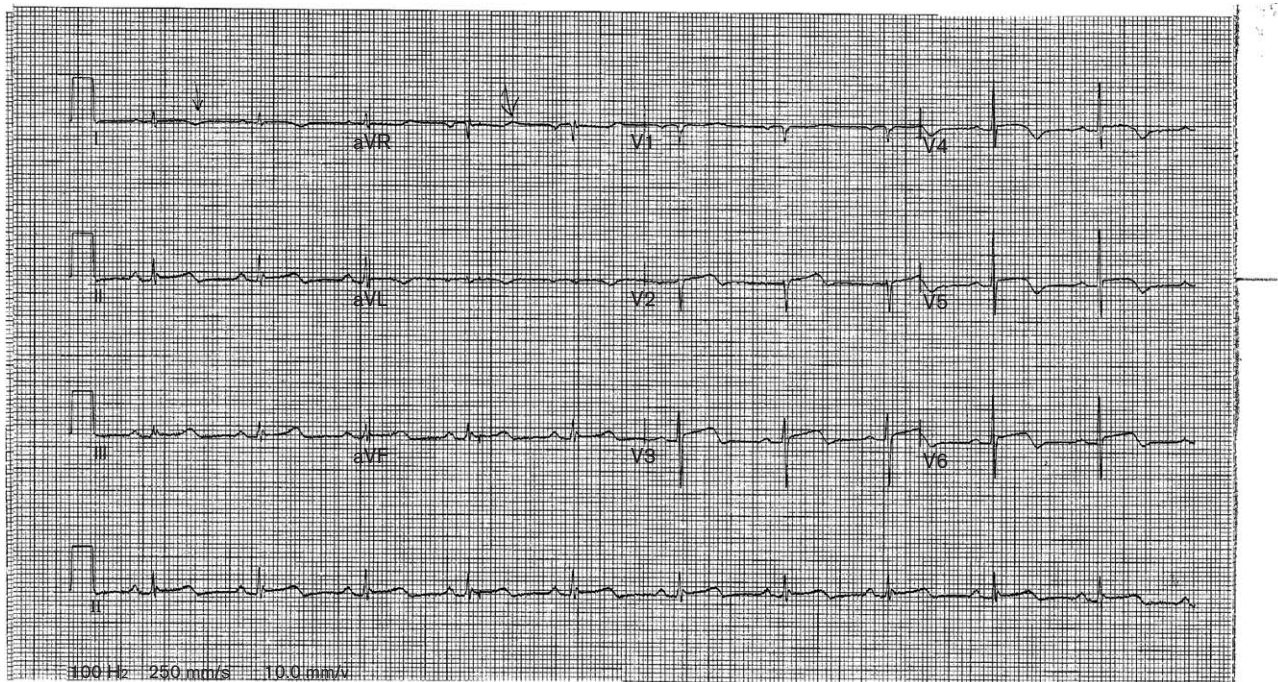
Vent. rate 82 bpm
PR interval 188 ms
QRS duration 70 ms
QT/QTc 392/457 ms
P-R-T axes 54 47 49



Baseline ECG on arrival.

Fig. 2

Vent. rate 63 bpm
 PR interval 194 ms
 QRS duration 68 ms
 QT/QTc 498/509 ms
 P-R-T axes 76 70 98



ECG 12 h later showing antero-lateral T inversion.

Case report

A 79-year-old Chinese woman with a background history of diabetes mellitus, hypertension and past history of anaphylaxis requiring endotracheal intubation and ventilatory support. She developed wheezing and periorbital swelling shortly after receiving intramuscular Diclofenac by the general practitioner (GP) for pain relief of her osteoarthritic knee. She did not have any syncopal episode or chest pain. She was conveyed by the paramedics to our ED who administered bronchodilators via nebulizer *en route* to our ED. No hypotension was documented by the paramedics.

On arrival at our facility an hour later she was not in distress with good resolution of her bronchospasm. Her vital signs were stable (blood pressure 140/75 mmHg, heart rate 83/min) and saturation in room air was 98%. The attending doctor administered antihistamine in the form of oral loratidine 10 mg and oral prednisolone. No adrenaline was administered to the patient.

Baseline ECG (Fig. 1) done did not show any abnormality. Initial troponin T was 0.06 µg/l. Chest radiograph and

electrolytes were also unremarkable. The patient remained stable throughout her stay in the ED and upon admission to the ward. A repeat ECG (Fig. 2) 12 h later showed antero-lateral T inversion that evolved into deep arrowhead T inversion 1 day later (Fig. 3). Troponin T increased to 0.82 µg/l and creatine kinase and MB 264 U/l and 24 µg/l, respectively. She subsequently underwent a coronary angiogram that showed normal coronaries with good ventricular function (visual ejection fraction).

The presumptive diagnosis was coronary vasospasm causing myocardial infarction precipitated by NSAIDS anaphylaxis.

Discussion

Profound myocardial depression has been reported in patients after severe anaphylactic reactions following induction of anesthesia [1], contrast medium, protamine sulphate, bee stings [2], centipede sting, adrenaline (Epi-pen) [3] and even skin disinfectant [4].

The mast cell activation and the subsequent release of mediators such as histamines represent a central patho-

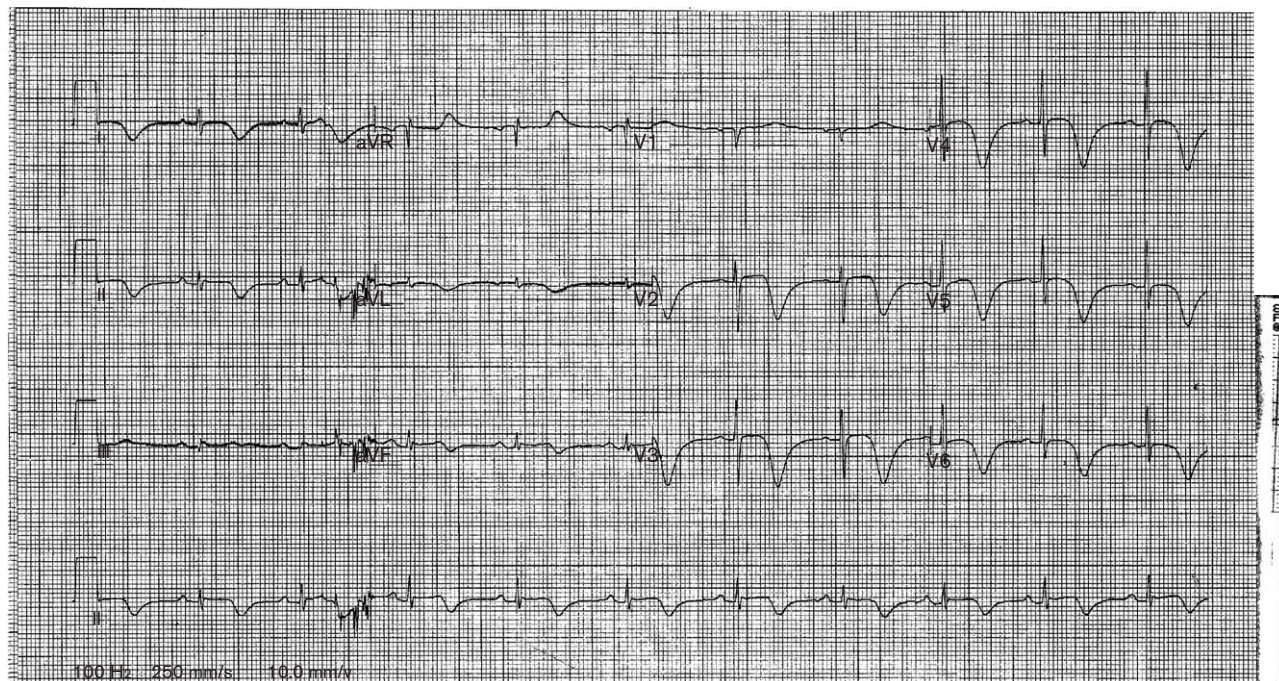
Fig. 3

Vent. rate 70 bpm
 PR interval 176 ms
 QRS duration 76 ms
 QT/QTc 528/570 ms
 P-R-T axes 57 65 170

Technician:

Referred by:

Unconfirmed



ECG one day later showing deep arrowhead T inversion.

genetic event in the induction of anaphylactic reactions. Histamine receptors have been isolated from coronary arteries and myocardium. Histamine has been shown to cause coronary vasoconstriction and consequent angina and myocardial injury. Alterations of coronary flow are also determined by the action of other mediators such as Cys-LTs-C4, LT-D4 and LTE4 [5–7].

Some authors have suggested that coronary hypoperfusion may result from the hemodynamic collapse that occurs in anaphylactic shock. Our patient was, however, not in shock and no adrenergic medicine was given. The only vasoactive substance administered was salbutamol and being a β -agonist was unlikely to cause the vasospasm.

This case highlights the potential complication of myocardial injury caused by coronary vasospasm precipitated by NSAIDs anaphylaxis. Clinicians should be aware of this possible complication when treating an anaphy-

lactic reaction especially in patients at risk for atherosclerosis. Early recognition of ECG changes is essential for diagnosis and treatment of coronary artery spasm.

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