A Fatal Case of Amiodarone-Induced Acute Respiratory Distress Syndrome in a Patient with Severe Left Ventricular Dysfunction Due to Extensive Anterior Acute Myocardial Infarction

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Case Report

A Fatal Case of Amiodarone-Induced Acute Respiratory Distress Syndrome in a Patient with Severe Left Ventricular Dysfunction Due to Extensive Anterior Acute Myocardial Infarction

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Abstract We here report a case of 71-year-old man with acute extensive anterior myocardial infarction, who was complicated with ventricular tachycardia (VT) even after successful percutaneous coronary intervention. As intravenous administration of nifekalant terminated VT, we started oral administration of amiodarone (day 1). We gave 400 mg of amiodarone a day for the first week and 200 mg a day from the second week. The patient was stable with normoxia by day 20, in spite of pulmonary congestion-like infiltrates on chest X-ray. On day 21, he was complicated with acute respiratory distress syndrome. Immediate discontinuance of amiodarone and high-dose pulse glucocorticoid therapy with intubation slightly improved the infiltrations on chest X-ray. However, glucocorticoid therapy induced hyperglycemia with an increase in plasma osmolality, complicated with hypoalbuminemia, and gastrointestinal bleeding. Despite treatment with a large amount of saline, high-doses of catecholamines, and blood transfusion, the patient died on day 28. It is sometimes difficult to diagnose congestive heart failure or amiodarone-induced pulmonary infiltrates in patients with severe left ventricular dysfunction.

Introduction

Amiodarone reduces the incidence of ventricular fibrillation or arrhythmic death among survivors of acute myocardial infarction with frequent or repetitive ventricular premature complexes. However, amiodarone is well-recognized to cause serious pulmonary adverse effects. Here, we report a fatal case of amiodarone-induced acute respiratory distress syndrome (ARDS) in a patient with severe left ventricular dysfunction due to extensive anterior acute myocardial infarction.

Case Presentation

A 71-year-old man with hypertension, hyperlipidemia, and diabetes mellitus had a sudden onset of chest pain. His primary physician diagnosed acute myocardial infarction based on ST elevation in leads II, III, aVF, and V1-6 with ventricular premature complexes on electrocardiography (ECG). On arrival at our hospital, the patient had a blood pressure of 94/64 mmHg and a heart rate of 90 beats/min. Left ventricular anterior and posterior wall
motion was severely reduced and left ventricular ejection fraction was 25% on echocardiogram. In the cardiac catheterization laboratory, the ECG showed non-sustained ventricular tachycardia (VT). Prior to coronary angiogram (CAG), we inserted intraaortic balloon pumping to stabilize hemodynamics. CAG showed total occlusions of 3 vessels; the proximal left anterior descending (LAD) coronary artery, the proximal left circumflex coronary artery with well-developed collaterals from high obtuse marginal branch, and the proximal right coronary artery with poorly developed collaterals from conus branch. Then, we performed percutaneous coronary intervention (PCI) to LAD, which was considered as culprit. During and even after successful PCI to LAD, sustained VT and ventricular fibrillation (VF) occurred. Intravenous lidocaine was not effective, and VT/VF was treated with an unsynchronized electric shock. As intravenous administration of nifekalant terminated VT/VF, we started oral administration of amiodarone on the next day (Day 1). We gave 400 mg of amiodarone a day for the first week and 200 mg a day from the second week (Fig. 1). The patient, who was complicated with renal dysfunction (serum creatinine level; 1.4–1.8 mg/dl), was hospitalized in the intensive care unit and stable with normoxia by day 20, in spite of mild pulmonary congestion-like infiltrations on chest X-ray (Fig. 2A: Day 1-14). Cardiac ultrasound did not show the improvement of left ventricular function. On day 21, he complained of dyspnea with oxygen saturation of 91%, when chest X-ray and computed tomography scan indicated ARDS (Fig. 2A: Day 21 and Fig. 2B). The plasma concentration of amiodarone/desethylamiodarone was 154/275 ng/ml on that day. Discontinuance of amiodarone and high-dose pulse glucocorticoid therapy with intubation slightly improved the infiltrations on chest X-ray (Fig. 2A: Day 24). However, glucocorticoid therapy induced hyperglycemia (serum glucose level; 1,239 mg/dl) with an increase in plasma osmolality (412 mOsm/kg*H2O), hypoalbuminemia (1.6 g/dl), and gastrointestinal bleeding (hemoglo-

![Fig. 1](image-url)
Fatal amiodarone-induced ARDS in AMI

Fig. 2  A. Chest X-ray. Day 1–14; Mild pulmonary congestion–like infiltrations. Day 21; Acute respiratory distress syndrome (ARDS). Day 24; ARDS, which is slightly improved by high-dose pulse glucocorticoid therapy. B. Lung computed tomography scan showed ARDS on day 21.

bin; 7.5 g/dl). Despite treatment with a large amount of saline, high-doses of catecholamines, and blood transfusion, the patient died on day 28.

Discussion

ARDS was first described in the 1960s, which is not a primary disease but a complication of severe and progressive forms of systemic inflammatory responses. It has been reported that the risks of development of ARDS are sepsis syndrome, multiple emergency transfusions, multiple trauma, and aspiration of gastric contents, which may be associated with the activation of neutrophils in pulmonary or systemic circulation, although the present case was not exposed to such conditions but amiodarone therapy.

It is generally believed that low-dose amiodarone therapy is safe. However, some reports have demonstrated that low-dose therapy can possibly induce pulmonary toxicity, whereas lung infiltrations, which are not fatal, seem to be caused at least several months after the administration. Therefore, it is rare that relatively low-dose amiodarone induced fatal ARDS in 3 weeks, as shown in the present study. Moreover, the present case might be possibly complicated with lung injury from the very early phase of the administration of amiodarone (Fig. 2 A; Day 1–14). We have to recognize that amiodarone could be associated with fatal lung toxicity even in early phase of administration, although it is sometimes difficult to diagnose congestive heart failure or amiodarone-induced pulmonary infiltrations in patients with severe left ventricular dysfunction.

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(和文抄録)

広範前壁急性心筋梗塞による重症左室機能低下例でアミオダロンにより急性呼吸促迫症候群を惹起し、救命し得なかった症例

麻生飯塚病院循環器科

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71歳、男性。広範前壁急性心筋梗塞による心源性ショック状態で、非持続性心室頻拍が発症していた。まず大動脈内バルーンパンピングを挿入し、血行動態を安定化させた。冠動脈造影上、左冠動脈前下行枝、回旋枝、右冠動脈の近位部が完全閉塞していたが、回旋枝および右冠動脈は側副血行路にて栄養されていた。責任病変は左冠動脈前下行枝と考え、インターベンションを施行した。インターベンション中、心室頻拍/心室細動となり、電気的除細動を繰り返した。左冠動脈前下行枝領域の虚血解消で心室頻拍/心室細動が改善せず、シンビット点滴にて心室頻拍/心室細動は消失した。血行動態が安定し、大動脈内バルーンパンピング挿入のまま集中治療室へ入室し経過観察した。入院翌日よりアミオダロンを開始し、20日後では酸素化も良好だったが、21日後、急性呼吸促迫症候群の所見を認め、ステロイドパルス療法を開始した。胸壁上、徐々にはあるが、急性呼吸促迫症候群所見は改善傾向に向かった。しかしながら、ステロイドパルス療法による非ケトン性高浸透圧性昏睡、低アルブミン血症、消化管出血による軽度の貧血を合併し、輸血およびアルブミン投与も無効で、救命し得なかった。アミオダロンによる肺障害は重篤な合併症の1つであるが、本症例のように重症左室機能障害を有する症例では、肺うっ血と早期のアミオダロンによる肺障害の鑑別が困難なことがあり、十分に留意する必要がある。