UNIVERSITÀ DEGLI STUDI DI TORINO

INTRAMURAL LEFT ATRIAL HEMATOMA:

A COMPLICATION OF PRIMARY CORONARY ANGIOPLASTY

INFERIOR MYOCARDIAL INFARCTION

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Introduction

An intramyocardial dissecting hematoma following a myocardial infarction is a rare condition, and even more uncommon is a localisation in the left atrium. There is uncertainty on the prognosis and most appropriate management of this complication, often a result of coronary artery perforation during percutaneous coronary intervention (PCI) (1). Although the dissecting hematoma usually forms almost exclusively in the myocardium adjacent to the culprit coronary lesion, in unique cases it can expand to other areas of the heart, compressing nearby structures and impeding blood flow. We therefore describe an intramural left atrial hematoma following a coronary perforation during PCI in a patient with inferior myocardial infarction.

Case report

This 77-year-old lady was admitted for acute inferior ST-segment elevation myocardial infarction. Her medical history was silent, besides being a smoker until few years before and reporting mild untreated hypertension. The coronary angiography performed four hours from the onset of the symptoms following bolus intravenous haeparin (5000 UI) and abciximab (0,25 mg/Kg), showed a thrombotic occlusion of the origin of the right coronary artery. Percutaneous aspiration was unable to completely remove the thrombus, and caused distal embolisation with TIMI 2 coronary flow. Significant lesions of the distal, posterior inter-ventricular, and postero-lateral portions of the right coronary artery were then treated with balloon angioplasty. However, the last inflation in the postero-lateral branch was complicated by a type 2 perforation, managed with prolonged (20 minutes) balloon inflations (Figure 1). At the intra-operative echocardiogram examination a 1.5 mm circumferential pericardial effusion was detected, without symptoms or signs of pericardial tamponade and, after documentation of satisfying final angiographic result, the patient was transferred to the Coronary Care Unit.

The three-hour control echocardiogram disclosed an unmodified pericardial effusion and a novel undefined mass with ipo-echogenic centre at the atrio-ventricular groove facing the lateral wall of the right ventricle. During the examination the patient developed right arm plegia and mouth deviation, thus brain and chest computed tomography scans were immediately performed. While the brain scan failed to describe notable lesions, the chest scan described extravasation of the contrast medium in the pericardium and presence in the

left atrium wall of an abundant and heterogeneous cluster of contrast medium mixed with clotted blood (Figure 2).

Although the hemodynamic state had hitherto remained stable, few hours later a sudden hypotensive episode required repeat echocardiography. The trans-thoracic examination showed an echogenic mass in the left atrium wall. The following trans-oesophageal investigation described a mass extensively occupying the left atrium and obstructing the left ventricular inflow (Figure 3).

Given the likely brain embolism episode and the undefined cardiac mass, associated with hemodynamic instability, she was referred to the cardiac surgeons for imperative intervention. The median sternotomy showed a pericardial effusion and an extrinsic compression on the left atrium by a massive intramural haematic infiltration; moreover, at surgical exploration an haemorrhagic right ventricle effusion extending toward the inferior diaphragm wall, the atrio-ventricular groove, the left atrial wall and the pulmonary vein ostia was evident.

The hematoma was drained with atrial decompression achieving a fairly stable hemodynamic state albeit with intravenous inotropic support.

After five days of critical hemodynamic stability, and repeat echocardiograms showing a progressively decreasing intra-atrial mass, the patient suddenly died in pulseless electrical activity. Autopsy was performed, showing that a massive pulmonary embolism due to deep venous thrombosis occurring in the hemiplegic limb had caused death.

Discussion

The present lady unfortunately incurred in multiple complications following a PCI for myocardial infarction, presenting simultaneously coronary distal embolisation, coronary perforation, dissecting hematoma and stroke. In front of this convergence of medical complications the left atrial intramural hematoma has been managed with an urgent surgical approach. Few similar cases can be found in literature, most of the patients undergoing surgical intervention in the first hours after the complication because of progressive hemodynamic deterioration (2, 3) and only one managed with a conservative successful approach (4). In the present case the decision has been dictated by the hemodynamic instability itself. In the present case the clotted blood collected in the restricted dissecting plane of the left atrial wall potentially affected pulmonary venous and transmitral flows obstructing left ventricular inflow. Furthermore the neurological symptoms

(right arm plegia and mouth deviation) related to the transition, at some point, of the clotted blood included in the broad dissection plane to the left blood stream.

Besides the doubts regarding the therapeutic options the diagnosis of this complication proved extremely challenging and resource consuming (5). The rapid evolution of the trans-thoracic and trans-oesophageal echocardiograms, showing a voluminous left atrial mass almost obliterating the left atrial chamber or icons easily mistakable with an intracavitary mass (especially if related to stroke symptoms), may result exceptionally confusing. The chest tomography scan images, on the other side, heightened the understanding of the anatomic evolution of the areas of the heart progressively involved. In fact the most plausible pathophysiological explanation of the present case of intramural left atrial hematoma is that following the subintimal dissection of the distal segment of the postero-lateral vessel the initial bleed was limited due to the fact that the cardiac tissues and the visceral pericardium contrasted the propagation. However over time the systemic pressure of the vessel pushed the blood through available areas of least resistance. The propagation gradually continued through the right ventricle myocardium creating a long plane dissection crossing the atrio-ventricular groove creating the large left atrial hematoma.

In conclusion, a left atrial intramural hematoma is a rare but deadly complication that can occur following PCI in inferior and right-ventricle myocardial infarctions. The diagnosis has to be promptly made; even though the best approach needs to be determined a conservative treatment, in stable hemodynamic conditions, possibly seems rewarding.

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Figures legend

Figure 1. This 77-year-old woman underwent coronary angiography disclosing thrombotic occlusion of the origin of the right coronary artery (A). Thrombus aspiration was unable to completely remove the thrombus and provoked a distal embolisation with a final TIMI 2 flow. The treatment with percutaneous balloon angioplasty only of the postero-lateral portion of the right coronary artery was complicated with a type 2 perforation (B) sealed by prolonged balloon inflations (C). Diffuse contrast medium effusion in the atrio-ventricular groove and left atrium are evident at the end of the procedure (D).

Figure 2. A chest computed tomography was performed for the evidence of an undefined mass with ipo-echogenic centre at the atrio-ventricular groove. The scan described extravasation of the contrast medium in the pericardium and presence in the left atrium wall of an abundant and heterogeneous cluster of contrast medium mixed with clotted blood.

Figure 3. The trans-thoracic echocardiogram described an echogenic mass in the left atrium wall (A), while the trans-oesophageal examination a mass extensively occupying the left atrium (B).