FIRST CASE OF INTRAMURAL HEMATOMA AND ACUTE PULMONARY EMBOLISM FOLLOWING PACE-MAKER IMPLANTATION: A CASE REPORT.

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Abstract

Bleeding complications after pacemaker implantation pose risks, including infection and prolonged hospital stay. A case involving intramural aortic hematoma (IMH) arising from subclavian vein access during implantation and concomitant acute pulmonary embolism (PE) is presented. In the present case, IMH probably resulted from subclavian artery vasa vasorum trauma during vein puncture and guidewire advancement, leading to IMH and hemothorax. PE possibly stemmed from a prothrombotic state caused by intervention and IMH. A conservative management with seriate CT scans was chosen due to hemodynamic stability and high surgical risk, with IMH resolution confirmed on follow-up.

INTRODUCTION

Bleeding related events after pacemaker (PM) implantation are associated with prolonged hospital stay, increased risk of device infection, patient discomfort and may be potentially life threatening. Pocket, pleurae and pericardium are known sites of bleeding following PM implantation. Acute aortic syndrome (AAS) are defined as a range of severe, painful and potential life-threatening abnormalities of the aorta. They include aortic dissection, penetrating aortic ulcer and intramural aortic hematoma (IMH). IMH is defined as a bleeding contained between the media and the intima of the aortic wall caused by the rupture of vasa vasorum. AAS are life-threatening conditions with an high mortality rate which is reduced only when diagnosed early and treated by surgeons with considerable expertise. We report a case of IMH which complicated a PM implantation with concomitant accidental finding of pulmonary embolism (PE) which was managed conservatively.

CASE

A 67-year-old female presented to the emergency department with epigastric pain, fatigue and vertigo lasting 3 weeks. The electrocardiogram (ECG) at admission revealed absence of atrial activity with junctional escape rhythm at 46 bpm. The patient had no reversible causes of AV block and no history of beta-blocker assumption. Bedside echocardiography revealed preserved ejection fraction with no wall motion abnormalities and no significant valve heart disease. According to last EHRA guidelines on cardiac pacing, permanent PM implantation was selected as treatment of choice. The patient was therefore brought to the electrophysiology laboratory: due to absence of cephalic vein, left subclavian vein was chosen as site of access. At the first attempt of vein puncture the patient experienced transient back pain while advancing the guidewire, thereby the needle and the wire were immediately removed. Vital signs remained stable. The following puncture was successful and passive fix ventricular leads were placed in the apex of the...
right ventricle and in the right atrial appendage. Postoperatively, the patient reported dyspnea and chest pain worsening during deep inspiration. Chest X-ray was immediately performed and revealed correct lead positioning but also the appearance of left pleural effusion (FIGURE 2). Blood exams showed a mild reduction in hemoglobin (from 12.3 to 10.1 g/dl). Therefore, a chest CT scan (FIGURE 3) was ordered to exclude active bleeding sources. Images showed a 49 mm pleural effusion and a 9 mm pericardial effusion localized at the periaortic recess associated with thickening and hyper density of the left portion of the aortic arch up to the origin of the left subclavian artery. No active bleeding sources were found. Those findings were compatible with IMH and hemothorax. Additionally, pulmonary embolism (PE) in the right inferior lobar artery was detected. Upper and lower limb ultrasonography did not reveal deep venous thrombosis. No signs of hemodynamic instability were present and bed-side echocardiography didn’t show right ventricular failure nor dilation. Pulmonary Embolism Severity Index (PESI) risk score was low (class II). Due to the presence of bleeding complications, anticoagulant therapy at optimal dosage could not be started to treat PE. Therefore, an inferior vena cava filter was placed by Interventional Radiologist and 0.4 ml subcutaneous low molecular weight heparin (LMWH) was administered every 12 hours. Hemothorax was treated by placing a thoracic drainage for two days. After multidisciplinary consultation with Cardiothoracic and Vascular Surgeons, considering hemodynamic stability and IMH was treated conservatively. CT scans at two-weeks showed a stationary condition of the IMH and gradual resolution of the pleural effusion and pulmonary embolism whilst plasmatic hemoglobin remained stable without need of red blood cells transfusion. The patient was discharged after three weeks, asymptomatic and in stable clinical condition. LMWH was continued at home and was followed-up weekly. At one-month CT-scan performed from discharge, IMH appeared completely resolved.

DISCUSSION

This report presents the case of a pacemaker implantation complicated by IMH, hemothorax and PE. In this case, IMH could be the result of the bleeding between the tunica media and the adventitia of the subclavian artery. Possibly, the vasa vasorum of the subclavian artery were damaged during vein puncture, causing IMH and subsequent blood spreading to mediastinum with following hemothorax. The bleeding was self-limiting because no sheath was inserted and the guidewire was immediately retired when the patient experienced back pain. Pulmonary embolism may be the result of a prothrombotic and inflammatory state caused by the intervention and IMH. Indeed, no signs of peripheral thrombi were found at US examination of upper and lower limbs. The patient was treated with the placement of a thoracic drainage, inferior vena cava filter and low dose LMWH. Despite IMH represents a potential life-threatening condition with the need of prompt intervention by expert surgeons, in our specific case it was managed conservatively due to hemodynamic stability and concomitant high surgical risk. Aortic complications are reported in literature following PM implantation and they all occurred due to injury of aortic wall by atrial lead. The first one was reported by Kashani et al. in 2004. In this case, aortic wall perforation was caused by excessive tissue penetration by the screw at the tip of the lead or perforation of the leaf body by the positioning stylet during manipulation whereupon the stylet traversed the atrial and the aortic wall, causing cardiac tamponade which was treated with thoracotomy and decompression. Kaljusto et al. also described an aortic complication following implantation. In their report, an epicardial perforation by the atrial lead caused an ulceration of the ascending aorta. These lesions caused a type A aortic dissection and cardiac tamponade requiring surgery. Moreover, Sticco el al. described the first case of delayed aortic wall perforation with subsequent cardiac tamponade occurring 2 weeks after PM implantation caused by pacing lead puncture of right atrial wall and subsequent injury of the adjacent ascending aorta. Finally, the last aortic complication reported in literature was described in 2014 by Di Marco et al. and occurred due to right coronary aortic sinus perforation by right atrial lead. However, in the present report the aortic complication did not affect the ascending aorta. IMH distribution was like a type B dissection and therefore was judged of less severity. Furthermore, the iatrogenic lesion did not affect the whole aortic wall, but only the media and the adventitia. Finally, the bleeding was self-limited and did not require invasive treatment. Nonetheless, life-threatening conditions may have ensued if the bleeding persisted or if it involved the ascending aorta. Cardiac tamponade, pseudo-aneurysm formation, aortic dissection, acute aortic regurgitation and thus acute
heart failure are possible outcomes of the progression of IMH. Therefore, early diagnosis and treatment of IMH are crucial in the post operative period. PE is a described complication which can occur after PM implantation. Indeed, lead implantation may cause DVT which in turn increases the risk for PE. In this case no signs of DVT were found at ultrasonography, therefore an alternative etiology should be suspected. It could be possible that PE was present and asymptomatic at presentation and became symptomatic after the procedure. Indeed, at presentation the patient had slightly increased d-dimer values (399 mg/mL) and vital signs (like blood pressure, respiratory rate and oxygen saturation) were normal. An alternative hypothesis is that the blood leak from the subclavian artery puncture caused a local inflammatory reaction, thus favoring a prothrombotic state at the site of puncture, favoring the formation of a thrombus within the subclavian vein. The introduction of the lead may have then dislodged the thrombus, causing PE.

CONCLUSIONS

Acute aortic syndromes are life-threatening conditions and represent a very uncommon complication after pacemaker implantation. According to our knowledge, we report the first case of IMH following PM implantation. In the present case, IMH may have been caused by traumatic injury by the needle or the guidewire to vasa vasorum of aortic wall which also caused left hemothorax. Concomitant PE complicated the clinical course making necessary inferior vena cava filter placement due to impossibility of continuing adequate anticoagulation. Due to hemodynamic stability and high surgical risk, a conservative management with periodical CT scans and blood cell counts was chosen, with complete documented resolution of IMH at follow-up.

FIGURE 1. 12-lead ECG at admission shows junctional rhythm at 46 bpm with narrow QRS and 1:1 V:A conduction ratio (retro conducted P-waves best seen in inferior leads)
FIGURE 2. Chest X-ray performed after implantation shows correctly placed Pacemaker leads with new-onset left pleural effusion.

FIGURE 3

FIGURE 3. Panel A and C: acute phase; Panel B and D: 2 months follow-up. A: high attenuation and thickening of the aortic wall (about 3-3.5 mm) associate with fat stranding of the periaortic anterior fat (dotted arrows). In B and D (2 months follow-up) the aortic wall is normal and the fat attenuation is almost normalized.

BIBLIOGRAPHY
KEY TEACHING POINTS:

- Bleeding related events after pacemaker (PM) implantation are associated with prolonged hospital stay, increased risk of device infection, patient discomfort and may be potentially life threatening.

- Acute aortic syndromes are life-threatening conditions with an high mortality rate which is reduced only when diagnosed early and treated by surgeons with considerable expertise.

- IMH may be caused from traumatic injury by the needle or the guidewire to vasa-vasorum of aortic wall and has never been described in literature as possible complication following PM implantation.

- Pulmonary embolism complicated the management of IMH due to the requirement of systemic anticoagulation and was treated with underdosed low molecular weight heparin and inferior cava vein filter placement.

- A conservative management of both conditions is possible, especially when surgical risk is high, hemodynamic conditions are stable and an adequate follow-up with seriate CT-scans and expert radiologists is available.