Early atrial fibrillation during acute posterior myocardial infarction: an interplay of ischemia of sinus node artery and atrial arrhythmogenesis.

ABSTRACT

Atrial arrhythmias and especially atrial fibrillation in the early phase (first 12 hours) of acute myocardial infarction (AMI) are rare. They are more common in the later stages of AMI and the most of the times as a sign of heart failure. The pathogenesis seems to be an underlined coronary ischemia. Among the causes an ischemia of sinus node artery (SNA) is previously described. SNA arise the most of the times from the initial part of right coronary artery while the remaining from the LCx or in a small proportion of them have double origin from RCA and LCx one. In this case we describe a case of atrial fibrillation in the early phase of AMI promptly resolved in the next one hour after successful percutaneous coronary angioplasty (PTCA) treatment of the culprit lesion in the LCx at site of the origin of SNA.

KEY WORDS: early atrial fibrillation, myocardial infarction, sinus node artery ischemia.
INTRODUCTION

Paroxysmal atrial fibrillation (PAF) in the early phase (first 12 hours) of AMI is rare. The most of the times this is a complication of the late stages particularly when heart failure is present. The cause of of PAF at this period of time during AMI is debated along time. In a recent retrospective study of 5946 patients the overall incidence of a new onset atrial fibrillation was 1.6% (AF terminated within 24 hours) and it was more prevalent among patients with inferior myocardial infarction (MI) (24.2%) [1].

Previous studies have shown that ischemia of sinus node artery (SNA) is present in this setting and may be the cause of atrial arrhythmias [2]. SNA is a small ramus of the right coronary artery (RCA) or of the circumflex branch of left coronary artery (LCx) and more rarely it has a double origin from the RCA and LCx [3,4]. Ischemia has produced either by an occlusion of the main artery just before the origin of SNA or at the site of the SNA origin [2,5]. In this case report we describe a rare case of PAF during inferior-posterior wall MI in a patient with double origin of SNA, due to a sub-occlusion of the branch originated from the LCx, at the site of culprit lesion.

CASE PRESENTATION

A 74 year-old man with a history of hypertension, dislipidemia, hyperuricemia was admitted to the emergency room (ER) complained for chest pain and dyspnea. The chest pain started 40 minutes before his arriving. The electrocardiogram (ECG) showed atrial fibrillation (AF), 100 bpm and posterior wall ST segment deviation myocardial infarction (STEMI) (figure 1). At focused echocardiography the ejection fraction (EF) was 50%, an inferior and postero-lateral walls akinesia was noted, there was minimal mitral regurgitation, no pericardial effusions and the right ventricle had preserved global kinesis. No past history of arrhythmias was known. Emergency coronarography was performed via radial approach. Before coronary angiography an oral loading dose of 600 mg of clopidogrel plus 300 mg of aspirin was administered together with 4000 UI of intravenous (IV) unfractionated heparin. The coronary angiography showed a subocclusion of LCx (culprit lesion) and a subcritical lesion of left anterior descending artery (visual estimation 60%) (figure 2a, 2b). Another ramus for sinus node originated from the initial part of RCA was observed at angiography of the right coronary artery (figure 3). Angioplasty and stenting with drug eluting stent (DES) of LCx was performed and a final TIMI flow 3 was obtained, without complications (figure 4).
Sinus rhythm was restored after one-hour without any other AF episode during hospital stay (figure 5). At third day, ECG (figure 6) and echocardiographic evaluation in intensive care unit (ICU) was performed. Left ventricle had preserved EF and no segment akinesia was observe. Persistence of sinus rhythm on ECG was also observed. The peak of troponin T was 3861 ng/L. Intravenous coagulation with unfractionated heparin was performed until 72 hours after PCI. Patient was discharged the fifth day after admission. The pharmacological therapy at discharge was: aspirine 100 mg/day, clopidogrel 75 mg/day, bisoprolol 1.25 mg/day, and atorvastatin 40 mg/day. An Holter ECG monitoring was also performed two months after discharge and no recurrence of AF were shown (figure 7).

Figure 1. Atrial fibrillation with ST segment depression in the anterior and lateral leads.

Figure 2. Angiography of left coronary artery. A critical occlusion of LCx before the origin of the obtuse marginal branch was observed (culprit lesion) (A). At the beginning of the lesion a ramus for sinus node blood supply was present (arrows) (A). Subcritical lesion of left anterior descending artery (visual estimation 60%) was also present (B).
Figure 3. Angiography of the right coronary artery.
Another ramus for sinus node originated from the initial part of RCA (arrows).

Figure 4. A coronary angioplasty of LCx culprit lesion was performed (A, B). A final TIMI flow III of the main vessel with a better visualization of the branch for sinus node was observed (arrows) (C).

Figure 5. Sinus rhythm restoration one hour after PTCA.
Figure 6. Sinus rhythm persists 3 days after PCI.

Figure 7. A strip of holter monitoring 2 months later show persistence of sinus rhythm.

DISCUSSION

PAF in the early phase of AMI is almost rare and most of the times it is due to SNA ischemia [1]. Previous studies in experimental animal models demonstrated a direct link between atrial myocardial ischemia and PAF when submitted to selective occlusion of atrial coronary branches [6]. Moreover early PAF in the acute phase of MI is an arrhythmic phenomenon associated in particular with inferior MIs [1,2]. The same was observed by Hod et al. [7] who had shown that 3% of patients with MI developed AF within 3 hours of the onset of chest pain and all these cases were associated with inferior MI. In all the above cases an occlusion of an atrial branch was observed (5 cases form the LCx and in two cases from RCA) [7]. Also, in a case report, Blanton Jr et al. demonstrated the rapid resolution of an early AF after PTCA of a LCx occlusion proximally to an huge atrial branch [8].
In our case, a concomitant double origin of a SNA was observed and PTCA of LCx artery’s culprit lesion was performed with subsequent sinus rhythm restoration. Previous studies reported that a double origin of the SNA was rare, but when present, the arteries most commonly origin from the RCA and LCX with a pooled prevalence of 3% [3]. Moreover, in a previous study of Alasady et al [9] has shown that about fifty percent of PAF cases during the early phase of AMI had reduced circulation to the sinoatrial nodal branch originating from the right coronary or left circumflex arteries compared to only 2.4% of controls and that atrial branches involvement is an independent determinant of PAF in the early phase of AMI.

In the present case, impaired perfusion of the SNA branch from the LCx main vessel sub-occlusion (culprit lesion) was observed. A prompt flow restoration was achieved by PTCA with drug eluting stent placement of the proximal LCx. A TIMI flow III of the main vessel was observed (fig 4) with subsequent rapid resolution of PAF.

Accordingly, PAF appearance in the early phase (first 12 hours) of an inferior AMI setting, may be due to a relationship between an ischemic and atrial arrhythmic substrate. The above findings are consistent with those of Hod et al. [7] which suggest that left atrial ischemia is the primary cause of early atrial fibrillation. Moreover, the same result was also supported by Kyriakidis et al. [2]. On the other hand Kondo et al. [10] in a letter mention that atrial ischemia during AMI may play a key role in the development of PAF.

In the present case sinus node function was restored one hour after successful PTCA of LCx artery at the site of origin of SNA, with sinus rhythm evidence at ECG (figure 5). The above finding seems to be due to a temporary alteration of arrhythmogenic substrate of sinus node function which is resolved after restoration of blood supply in the SNA. The aforementioned occasional arrhythmic event seems do not have any further implications for the patient. This speculation is further corroborated from the results of an ECG Holter monitoring (figure 7) performed 2 months later which did not show any atrial arrhythmias.

Because up until now we have not any modality (including scintigraphy and echocardiography) to confirm atrial ischemic damage, further studies are necessary to evaluate the association between atrial and/or SNA ischemia and early PAF.
CONCLUSIONS

In conclusion, this case show a patient with PAF and double origin of SNA from LCX and RCA in the context of posterior AMI. An abrupt flow's impairment of the branch of SNA, due to thrombus sub-occlusion of this artery at the site of its origin from LCX artery, may cause an episode PAF. This episode was resolved one hour after successful PTCA, so practically after removal the ischemic burden. Similar episodes are frequent in the context of inferior AMI with proximal LCx and/or RCA involvement, at the site of or before the origin of SNA branch and seem do not have any further arrhythmic complications in the short-term follow up (2 months).

Disclosure statement

The authors did not report any potential conflict of interest.
REFERENCES


