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CASE REPORT

Post-traumatic myocardial infarction with hemorrhage and microvascular damage in a child with myocardial bridge: is coronary anatomy actor or bystander?

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ABSTRACT

We present the case of a 13 year old patient with myocardial bridge in left anterior descending coronary artery, who develops a myocardial infarction after a cardiothoracic trauma.

About 24 hours after admission for trauma, an Electrocardiogram (ECG) showed an ST-segment elevation on anterior-lateral leads and QS complex referable to anterior-septal infarction, and an increase in troponin T serum levels was noted. An impaired left ventricular ejection fraction with diffuse regional wall motion abnormalities involving the left ventricular apex and interventricular septum were seen at transthoracic echocardiography. Contrast enhanced cardiac magnetic resonance showed a widespread myocardial edema and necrosis at the level of left ventricular apex and interventricular septum. Intramural hemorrhage and signs of microvascular damage were found mainly at the mid-ventricular level of the antero-septal and anterior segments of myocardium. The coronary angiography revealed normal coronary arteries except for a myocardial bridge on distal part of left anterior descending coronary artery. A myocardial infarction with hemorrhage and microvascular damage was diagnosed, but the absence of a correspondence between site of the most severe myocardial injury and distal location of myocardial bridge was noted. Whether myocardial infarction and microvascular damage have been caused only by traumatic hit, or also by the contribution of myocardial bridge, is unknown. An intense constriction of left anterior descending coronary artery at the level of myocardial bridge could have determined thrombus formation with subsequent septal and distal embolization and myocardial infarction.

Key words: myocardial bridge, myocardial infarction, contrast-enhanced cardiac magnetic resonance, coronary arteries, thrombus, thoracic trauma.

Introduction

The myocardial bridge (MB) is a common anatomic variant defined as an intramural segment of a coronary artery that runs through the myocardium. The incidence of MB is higher at autopsy than at coronary angiography (86 % and 5%, respectively). This difference has been attributed to insensitivity of angiography to MB. (1,2)

MB is generally considered a benign condition that most commonly affects the mid portion of the left anterior descending (LAD) coronary artery. It is characterized by systolic compression of intramyocardial course of coronary artery, which remains clinically silent in the vast majority of patients. However, numerous clinical cases of this anomaly have reported the association of MB with myocardial infarction. (1,3)

We describe the case of a 13 year old patient with MB and a post traumatic myocardial infarction. We discuss the physiopathology of this event, the real role of MB in myocardial infarction after chest trauma.

Case report

A 13 year old male was referred to our

Emergency Department for traumatic cardiothoracic and head injury, occurred during a bike ride. Apart from a temporary loss of consciousness, at admission he was hemodynamically stable, suffering only from a mild chest pain. Computed tomography demonstrated a fracture of the first left rib, a hematoma in the interscalene groove and a small spleen contusion. The first electrocardiographic examination (ECG) described only a sinus tachycardia. The day after, while he was asymptomatic, ECG showed an ST-segment elevation on antero-lateral leads, QS complex in V2-V4 referable to antero-septal infarction associated to sinus tachycardia. Then, an increase in troponin T serum levels (4.5 ng/mL), which reached a peak value of 5.8 ng/mL afterwards, was noted. Noninvasive imaging assessment was performed by transthoracic echocardiography (TTE) and contrast enhanced cardiac magnetic resonance (CeMR). TTE demonstrated a moderately impaired left ventricular (LV) ejection fraction with diffuse regional wall motion abnormalities extending to the entire LV apex and interventricular septum (figure 1). CeMR showed a widespread myocardial edema, involving the entire LV apex and interventricular septum in T2-weighted STIR images (figure 2a). In the context of edematous myocardium, a hypointense rim of intramural hemorrhage could also be appreciated (between arrows). First pass sequences acquired just after gadolinium injection (figure2c) showed a subendocardial perfusion defect within LV anterior and septal walls mainly at the mid-ventricular and apical level (between arrows), whereas delayed enhancement images revealed a transmural ischemic necrosis of antero-septal mid-ventricular wall, which extended, as subendocardial hyper-enhancement, to the apex and mid-ventricular anterior wall (figure 2e). In addition, signs of microvascular damage were found inside the injured myocardium of antero-septal and anterior segments. As confirmation of the absence of any clinical signs of post-traumatic cardiac tampona-

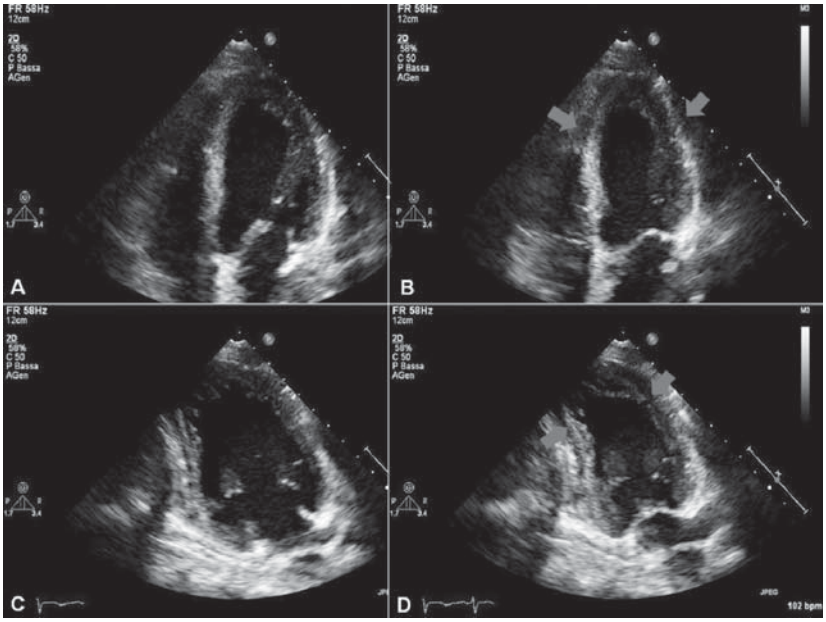

Figure 1. Two dimensional transthoracic echocardiographic diastolic (a, c) and systolic (b, d) frames acquired at admission in 4 (a, b) and 2 (c, d) chamber view and demonstrating a moderately impaired left ventricular ejection fraction with akinetic apex and hypokinetic mid-ventricular septum and anterior walls (between arrows).

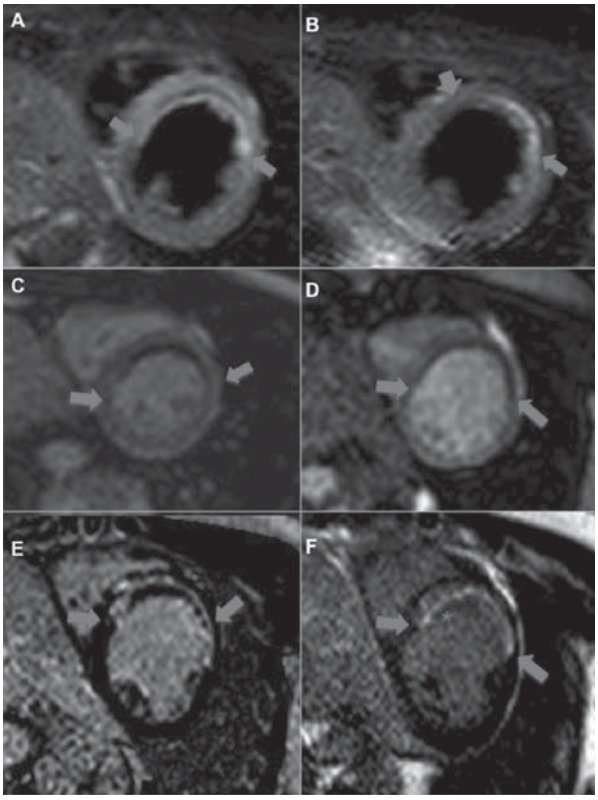

Figure 2. Cardiac magnetic resonance images in short axis plane, showing a mid-ventricular slice acquired by T2-weighted STIR (a, b) first pass (c, d) and delayed enhancement (e, f) sequences during the sub-acute phase of myocardial injury (a - e) and at 3 months follow-up (b - f).

de, no pericardial effusion was found. Despite the imaging appearance of sub-acute myocardial infarction in territories supplied by LAD coronary artery, the subsequent coronary angiography revealed the absence of any thrombus, stenosis or dissection in the proximal and mid portion of LAD and showed an MB only on distal part of LAD. An antiplatelet and beta-blocker therapy was adopted, and about one week later TTE showed a progressive improvement of global and regional contractile function with recovery of ejection fraction (last value was 55%). The troponin T levels were normalized and the ECG showed sinus rhythm, T waves inversion in antero-lateral leads with Q-waves in V1-V3 leads. CeMR was repeated 3 months later (figures 2b-f). A mild persistence of myocardial edema (figure 2b) was noted, while perfusion defect (figure 2d) extended similarly to that at baseline. Areas of late hyper-enhancement appeared slightly reduced in transmurality, reaching > 50% of wall thickness within the mid-ventricular antero-septal segment and no more than 50% along the anterior and apical walls (figure 2f, between arrows). Similarly, a small area of microvascular damage persisted at 3 months follow-up, mainly at the level of mid-ventricular antero-septum (figure 2f).

Discussion

Myocardial contusion and hematoma due to blunt chest trauma are extensively reported in literature. Post-traumatic myocardial infarction, due to direct injury of coronary arteries, such as dissection, rupture and atherosclerotic obstruction, has been already characterized at CeMR. (4) Chest trauma in childhood often determines a ventricular septal defect or left ventricular aneurismal formation. (5) However, the occurrence of post-traumatic hemorrhage and microvascular damage with ischemic pattern has not been seen at CeMR yet. MB has been widely demonstrated to induce myocardial injury through several mechanisms. (1) In the present case, the absence of a strict correspondence between site of the most severe myocardial injury (mainly mid-ventricular septal wall) and distal location of MB let one suspect that myocardial infarction and microvascular damage could be caused only by traumatic hit. On the other hand, in presence of MB, an intense and prolonged constriction of LAD, lasting for both diastolic and systolic phase of cardiac cycle, might have determined thrombus formation inside the proximal and mid-ventricular portion of LAD, with subsequent septal and distal embolization. Similarly to the unknown pathophysiology, the best treatment option is unclear. In children, usually the choice of percutaneous treatment of MB is excluded due to the fact that stent might become disproportionate in respect to increasing LAD. Only in adulthood, if the pharmacological treatment fails, the possibility of surgical intervention as bypass grafting, or endovascular intervention with a stenting on LAD, could be evaluated. (3)To the best of our knowledge, this is the first case of myocardial infarction with intramural hemorrhage and microvascular damage occurred in a child with myocardial bridge of LAD.

REFERENCES

1. Möhlenkamp S, Hort W, Junbo GE, Erbel R. Update on Myocardial Bridging. Circulation 2002;106:2616-22.
2. Dursun I, Bahcivan M, Durna K, Ibrahimov F, Erk NH, Yasar E, et al. Treatment strategies in myocardial bridging: a case report. Cardiovasc Revasc Med 2006;7:195-8.
3. Gomberg-Maitland M, Micheal C, Kim A. A stratified approach to the treatment of a symptomatic myocardial bridge. Clin Cardiol 2002;25:484-6.
4. Malbranque G, Serfaty JM, Himbert D, Steg PG, Laissy JP. Myocardial infarction after blunt chest trauma: usefulness of cardiac ECG-gated CT and MRI for positive and aetiologic diagnosis. Emerg Radiol 2011;18:271–4.
5. Studer MA, Jefferies JL, McKenzie ED, Ing FF. Traumatic cardiac rupture and left ventricular aneurismal formation in childhood. Am J Cardiol 2008;101:413–4.