Abstract: P665

Unusual case of anterolateral papillary muscle rupture

Authors:
IA Guelli1, D Morrone1, A Pizzuto1, A Canu1, L Villanova1, G Terllizzese1, R Pedrinelli1, E Orsini1, 1Dipartimento di patologia chirurgica, medica, molecolare e dell'area critica, Sezione malattie dell'apparato cardiovascolare - Pisa - Italy,

Topic(s):
Echocardiography: Valve Disease

Citation:
European Heart Journal - Cardiovascular Imaging (2019) 20 (Supplement 1), i393

Case Report: A 62-year-old male, with diabetes mellitus type 2, was admitted to the emergency room (ER) with chest pain and sweating. At the physical examination, heart rate was 98 beats/min and blood pressure was 100/80 mmHg. The EKG revealed inferior-lateral STEMI with involvement of posterior wall and right ventricle. Trans-thoracic echocardiographic examination (TTE) showed akinesia of the basal and mid inferior wall and akinesia of the basal infero-lateral and basal infero-septal wall, with mild reduced left ventricular systolic function (estimated ejection fraction of 48 %). The patient was promptly deferred to the cath lab and angiography was performed showing a mid tract occlusion of the right coronary artery (RCA). Percutaneous coronary intervention with stenting of the RCA was performed, with evidence of no reflow treated with adenosine and abciximab with poor angiographic result (TIMI 2). The post-procedural EKG, performed within 4 h showed persistence of ST elevation. Laboratory tests showed increased Troponin Hs (2732 ng/ml) and CK-Mb level (19 ng/ml). Trans-thoracic echocardiographic examination (TTE) was unchanged in terms of kinesis but moderate-severe mitral regurgitation with posterior leaflet tethering was also founded. Following days the patient was hemodynamically stable with a decrease in markers of myocardial necrosis therefore was discharged on day eight post-MI. After two days from discharge, he presented to the ER for sudden onset of dyspnea and profuse sweating, patients was in acute pulmonary edema and suddenly developed signs of cardiogenic shock (systolic blood pressure 80 mmHg, with signs of hypoperfusion). At this time TTE revealed anterolateral papillary muscle rupture with severe eccentric mitral regurgitation (See GIF). He was promptly transferred to the cardiothoracic surgery operating room (OR) after starting norepinephrine and diuretics. Upon arrival in the OR, intraoperative transesophageal echography (TEE) confirmed anterolateral papillary muscle rupture with severe regurgitation jet. Valvular replacement with mechanical valve prosthesis was therefore performed and the patient was transferred to the intensive care unit. However, the patient suffered cardiorespiratory failure on day 2, requiring intubation with mechanical ventilation; the patients deceased due to septic shock on day 7.

Conclusions: We presented an unusual case of anterolateral papillary muscle rupture due to an RCA lesion. Anterolateral papillary muscle has a dual blood supply (left anterior descending and circumflex coronary arteries) and therefore, is more likely to be protected following an acute myocardial infarction in a single-vessel territory. The posteromedial papillary muscle is only supplied by the posterior descending coronary artery. We believe that, in this clinical case, an overwhelming right predominance of the blood supply to the anterolateral papillary muscle led to this catastrophic outcome.
Case Report: A 62-year-old male, with diabetes mellitus type 2, was admitted to the emergency room (ER) with chest pain and sweating. At the physical examination, heart rate was 98 beats/min and blood pressure was 100/80 mmHg. The EKG revealed inferior-lateral STEMI with involvement of posterior wall and right ventricle. Trans-thoracic echocardiographic examination (TTE) showed akinesia of the basal and mid inferior wall and akinesia of the basal infero-lateral and basal infero-septal wall, with mild reduced left ventricular systolic function (estimated ejection fraction of 48%). The patient was promptly deferred to the cath lab and angiography was performed showing a mid tract occlusion of the right coronary artery (RCA). Percutaneous coronary intervention with stenting of the RCA was performed, with evidence of no reflow treated with adenosine and abciximab with poor angiographic result (TIMI 2). The post-procedural EKG, performed within 4 h showed persistence of ST elevation. Laboratory tests showed increased Troponin Hs (2732 ng/ml) and CK-Mb level (19 ng/ml). Trans-thoracic echocardiographic examination (TTE) was unchanged in terms of kinesis but moderate-severe mitral regurgitation with posterior leaflet tethering was also found. Following days the patient was hemodynamically stable with a decrease in markers of myocardial necrosis therefore was discharged on day eight post-MI. After two days from discharge, he presented to the ER for sudden onset of dyspnea and profuse sweating, patients was in acute pulmonary edema and suddenly developed signs of cardiogenic shock (systolic blood pressure 80 mmHg, with signs of hypoperfusion). At this time TTE revealed anterolateral papillary muscle rupture with severe eccentric mitral regurgitation (See GIF). He was promptly transferred to the cardiothoracic surgery operating room (OR) after starting norepinephrine and diuretics. Upon arrival in the OR, intraoperative transesophageal echography (TEE) confirmed anterolateral papillary muscle rupture with severe regurgitation jet. Valvular replacement with mechanical valve prosthesis was therefore performed and the patient was transferred to the intensive care unit. However, the patient suffered cardiorespiratory failure on day 2, requiring intubation with mechanical ventilation; the patient deceased due to septic shock on day 7.

Conclusions: We presented an unusual case of anterolateral papillary muscle rupture due to an RCA lesion. Anterolateral papillary muscle has a dual blood supply (left anterior descending and circumflex coronary arteries) and therefore, is more likely to be protected following an acute myocardial infarction in a single-vessel territory. The posteromedial papillary muscle is only supplied by the posterior descending coronary artery. We believe that, in this clinical case, an overwhelming right predominance of the blood supply to the anterolateral papillary muscle led to this catastrophic outcome.