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Predictors of tamponade and constriction in patients with pericardial disease undergoing interventional and surgical treatment

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ABSTRACT

Objective: The aim of our study was to define predictors of cardiac compression development including clinical, electrocardiographic, echocardiographic, chest-X-ray and perioperative parameters and their diagnostic value.
Methods: Overall 243 patients with pericardial disease, among them 123 with compression (tamponade, constriction) and 120 without signs of compression were included in the study. Clinical, laboratory, electrocardiographic, chest-X-Ray, echocardiographic and perioperative data were included in the logistic regression analysis to define predictors of tamponade/constriction development.
Results: Logistic regression analysis demonstrated large effusion (>20 mm) (OR 5.393, 95%CI 1.202–24.199, p = 0.028), cardiac chamber collapse (OR 31.426, 95%CI 1.609–613-914, p = 0.023) and NYHA class > 3 (OR 8.671, 95%CI 1.730–43.451, p = 0.009) were multivariable predictors of compression development. The model including these three variables allowed predicting compression in 91.7% of cases.
ROC analyses demonstrated that all three variables had significant diagnostic value with sensitivity of 75.6% and specificity of 74.2% for large effusion, low sensitivity and high specificity for cardiac chamber collapse (35% and 92%) and NYHA class (32.5% and 94.2%).
Conclusion: The independent predictors of compression development are presence of large effusion >20 mm, cardiac chamber collapse and high NYHA class. The model including all three parameters allows correctly predicting compression in 91.4% of cases. The diagnostic accuracy of each parameter is characterized by high sensitivity and specificity of large effusion, high specificity of cardiac chamber collapse and NYHA class.

1. Introduction

Tamponade and constrictive pericarditis (CP) are complications of pericardial diseases (PD), causing heart compression and accompanied by perioperative mortality in 4–14% of cases [1–5]. Several studies [2,4–8] reported that in patients with CP referred for pericardiectomy along with tuberculosis as the main etiological cause, there is an increase of idiopathic, postradiotherapy pericarditis and postpericardiotomy syndrome (PPTS). Therefore, early detection of compression and risk stratification seems to be important in prevention of constriction and tamponade.

Clinical tamponade signs like jugular vein distention, hepatomegaly, hypotension and paradoxical pulse, might not be evident in cases of localized effusion or low-pressure tamponade [1,9,10]. Several studies also demonstrated, that clinical signs were not suspected in >50% of cases with echocardiographic (echo) and cardiac catheterization signs of tamponade [11,12].

Imaging methods allow accurately diagnose tamponade and constriction, though several parameters need to be combined to identify correctly compression, as they differ by accuracy and some signs can accompany other structural heart diseases [13–15,18].

However, studies on predictors of tamponade and constriction development in PD are limited and few is known on factors determining development of compression. It is known that risk of tamponade increases three-fold when paradoxical pulse exceeds 10 mmHg [19], and in presence of low voltage QRS, accompanying large effusion [20]. Chronic large effusion might progress to tamponade in about 1/3 of cases, while in subacute large effusions risk of tamponade is high in cases unresponsive to medical treatment [13,21,22]. Constriction

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develops in tuberculous pericarditis accompanied by tamponade, despite full resolution of effusion after pericardiocentesis and in cases of acute pericarditis resistant to treatment with acetylsalicylic acid [6,23].

It was demonstrated that cardiac chamber collapse (CCol) and inferior vena cava (IVC) plethora lacked value in prediction of tamponade, while large pericardial effusion increased risk 78-fold [24]. Large effusion size (>500 ml) was shown to predict tamponade development with accuracy of 100% [25]. It should be also noted, that CCol could be observed in 1/3 of cases without clinical signs of tamponade [26].

Thus, evidence on factors predictive for development of compression in PD is limited, few is known whether combination of clinical, hemodynamic, X-Ray, ECG, and echo parameters may improve early identification of patients at risk of constriction and tamponade.

The aim of our study was to define predictors of cardiac compression development including clinical, electrocardiographic (ECG), echo, chest-X-ray and perioperative parameters and their diagnostic value.

2. Methods

We retrospectively analyzed records included in the prospective database of patients with PD admitted to the Scientific Research Institute of Heart Surgery and Organ Transplantation between 1997 and 2014.

2.1. Patients

Overall, 243 consecutive patients with PD referred for treatment in our center, were included in the study. All patients were divided into 2 groups according to presence of tamponade and constriction signs [1]: 123 patients with syndrome of compression and 120 patients without signs of compression.

2.2. Clinical variables

The following demographic and clinical data were included in the analysis: age, gender, etiology of PD, NYHA class, duration of hospitalization; presence of inflammation, laboratory analysis; hemodynamic status — heart rate (HR), systolic (SBP) and diastolic (DBP) blood pressure, central venous pressure (CVP) where appropriate; cardiothoracic index (CTI), presence of pleural effusion and superior vena cava (SVC) dilatation/compression on chest X-Ray.

2.3. Echocardiography

Cardiac chambers` size and pericardial involvement (thickening, calcification, effusion size, tamponade and constriction signs) were estimated according to recommendations of American Society of Echocardiography and European Association of Cardiovascular Imaging [27,28] using transthoracic 2-dimensional (2D), 2D-guided M-mode and Doppler echocardiography (echo). The following echo data were included in analysis: left atrial (LA) size, right atrial (RA) enlargement, left ventricular (LV) end-systolic and end-diastolic dimensions, LV ejection fraction (LVEF, estimated by Simpson rule), RV dimension, mean pulmonary arterial pressure, signs of LA, RA, RV and LV collapse [29–31], IVC plethora [14], respiratory variations of tricuspid and mitral flows` [32], interventricular septal (IVS) and inter-atrial septal paradoxical movement, flat atrioventricular (AV) groove [16], constrictive E/a pattern of mitral flow [15,17,33], hepatic vein dilatation and flow reversal [15], and presence of fibrin detachments in pericardial cavity. We also measured presence and extent of pericardial thickening and calcification, extent and size of effusion. Based on above-mentioned echo data, pericardial compression was defined as tamponade and constriction [13,15,27–34].

2.4. Electrocardiography

ECGs were scanned and digitally analyzed as described previously [35] for presence of PR segment depression, low voltage QRS, QRS alternans, ST junction elevation/depression, and P-wave morphology abnormalities (notched P-wave, changes in amplitude — P mitrale and pulmonale patterns) [20,36,37]. Arrhythmias and conduction defects [38] were evaluated from ECGs, and whenever available Holter monitoring and telemetry records.

2.5. Pericardiocentesis and pericardiectomy

Pericardiocentesis with and without drainage, subxiphoid pericardiostomy and pericardiectomy were performed by standard technique [1,9,39,40]. During interventions and surgery, we assessed size of effusion obtained by drainage and intraoperatively, and its extent; during surgery we also evaluated extent and size of adhesions and calcifications.

2.6. Definitions

Based on above-mentioned data, pericardial involvement was classified as effusion, effusion with compression (tamponade), constriction and constriction with effusion, adhesive, adhesive with effusion and adhesive-effusive with signs of compression, as well presence or absence of compression (tamponade or constriction).

Extent and size of pericardial effusion, thickening and calcification of pericardium were classified based on echo and drainage (effusion)/intraoperative data. Effusions were categorized by extent as localized and diffuse; and by size as small (<100 ml by drainage/intraoperatively, <10 mm by echo for diffuse effusion and <5 mm for localized effusion), moderate (100–400 ml by drainage/intraoperatively, 10–20 mm by echo for diffuse effusion and <5–10 mm for localized effusion), and large (>400 ml drainage, >20 mm by echo for diffuse effusion and >10 mm for localized effusion), [1,34,41,42]. Pericardial thickening and calcifications were graded as localized and diffuse.

Presence of inflammation was judged on increased levels of C-reactive protein (CRP), sedimentation rate, and immune markers where applicable.

2.7. Outcomes

Outcomes were recorded based on hospital records and follow-up of patients. The following outcomes were included in the analysis: recurrent PD, heart failure, death and composite outcome.

2.8. Statistical analysis

Statistical analyses were performed using SPSS for Windows software (IBM, New York). Categorical variables are presented as number (percentage) and continuous variables as mean (SD). The normality of data distribution was assessed by Kolmogorov–Smirnov test. Comparisons between groups were performed using Chi-square test for categorical variables and unpaired t test for independent samples for normally distributed data and Mann–Whitney U test for abnormally distributed data. Logistic regression analysis (LRA) was performed to identify predictors of compression syndrome. The dependent variable was binary-presence or absence of cardiac compression; for selection of independent variables — variables with significance value < 0.1 on univariate analysis were included in the model. p value < 0.05 was accepted as a significant value for all tests. Diagnostic value of multivariable predictors of cardiac compression was defined using ROC analysis with assessment of area under the curve (AUC), 95% CI, p values, sensitivity and specificity.