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Surgical embolectomy versus thrombolytic therapy in the management of acute massive pulmonary embolism: Short and long-term prognosis

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ABSTRACT

Objective: Comparison between surgical embolectomy and thrombolytic therapy in patients suffering from acute massive pulmonary embolism (AMPE).
Background: Prompt treatment of AMPE is necessary, although optimal management is a matter of debate.
Methods: Patients with AMPE were assigned to either thrombolytic therapy or pulmonary surgical embolectomy. Early and late mortality, systolic pulmonary artery pressure (SPAP), right ventricular diameter (RVD) and bleeding complications were evaluated.
Results: Seventy eight patients were treated with thrombolytic therapy and 30 patients underwent surgery. The difference between pre-intervention and third-day post-intervention in terms of RVD and SPAP was significantly greater in patients under surgical embolectomy ($P < 0.001$). There was a significant decline in RVD and SPAP in both groups during follow-up ($P < 0.001$). Mortality rate in the surgical embolectomy group was lower than the thrombolytic group although not significantly.
Conclusion: Early surgical treatment was associated with fewer complications in comparison to thrombolytic therapy.

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Introduction

Acute massive pulmonary embolism (AMPE) is life-threatening. Despite advances in diagnosis and therapy, AMPE is still associated with exceptionally high mortality and morbidity rates.[1,2] Patients presenting with AMPE are at high risk of circulatory collapse, medical and mechanical reanimation and late pulmonary hypertension.[3]

Prompt treatment should be undertaken when dealing with AMPE. However, optimal management remains debated, and there is no consensus regarding the best therapeutic method. Although in recent studies the results of surgical embolectomy have been satisfying, current guidelines suggest thrombolytic therapy as the treatment of choice. Therefore surgical management is placed in reserve for critically ill and high-risk patients, in whom thrombolysis is absolutely contraindicated or has failed.[4] Few studies have been conducted on short and long-term consequences of surgical embolectomy and thrombolysis. The rare incidence of acute massive pulmonary embolism and difficulty in matching patients has limited research on this issue. The present study aimed to analyze the short and long-term results in patients suffering from AMPE treated with either surgical embolectomy or thrombolytic therapy over an 8-year period.

Methods

A non-randomized prospective study was designed on patients referred with acute massive unilateral or bilateral pulmonary embolism. A number of parameters including early mortality, SPAP, RVD and bleeding complications were recorded during

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hospitalization. Thereafter, late mortality, SPAP, RVD and New York Heart Association (NYHA) class of patients were assessed during follow-up.

Selection of patients

A total of 120 patients with acute massive central or bilateral para-central pulmonary embolism (symptom onset less than 15 days) were included in the study. Due to the low incidence of the disease, all consecutive patients referring to Ghaem Hospital from May 2004 to November 2012 were enrolled. Massive PE was defined by occlusion of the pulmonary artery by more than 50% of its cross sectional area or occlusion of two or more lobar arteries or circulatory shock or moderate to severe right ventricular dysfunction confirmed by echocardiography.[3] Diagnosis was made by computed tomography (CT) scanning, transesophageal (TEE), or transthoracic echocardiography (TTE). Patients who refused any intervention or were not accessible during follow-up, were excluded from the study. All patients gave their informed written consent for inclusion in the study, and the protocol was approved by the local ethics committee. Upon indications, patients were assigned to either pulmonary surgical embolectomy (SE) or thrombolytic therapy (TT). A group of patients (TS) with failed responses to thrombolysis underwent rescue surgery and outcomes were analyzed separately.

Patients' data, including the initial manifestations, predisposing factors, co-morbidities, laboratory tests, ECG, diagnostic modalities used, site of thrombi and echocardiographic findings were recorded. Troponin I (Tp I) values were measured within 12 h of admission using a qualitative immunological assay (Tp I, Acon Inc.) and a quantitative ELISA (DiaPlus Diagnostics) with a detection limit of 0.5 ng/ml and 1.4 ng/ml, respectively. For practical reasons, both Tp I values exceeding the discriminator value were reported as positive. Patients' outcomes were evaluated by TTE and NYHA class. The patients were followed for at least 12 months.

Echocardiography

Standard 2-dimensional (2D) and Doppler transthoracic echocardiography (TTE) were performed using the Vivid 3 and Vivid 7 (GE Vingmed Ultrasound, Horton, Norway) with a 3.2 MHz transducer in left lateral position in all patients before intervention, within three days after intervention and one year later. Echocardiography was performed by a qualified cardiologist while considering the American Society of Echocardiography (ASE) guidelines. The maximal tricuspid regurgitation (TR) velocity was recorded by continuous-wave Doppler from a standard view that yielded the highest peak velocity during end-expiration. This velocity reflects the pressure difference between the right ventricle (RV) and atrium (RA) during systole. Therefore, systolic RV pressure can be estimated by adding up RA pressure and the trans-tricuspid gradient obtained from tricuspid regurgitation velocity. Trans-tricuspid pressure gradient was calculated using the modified Bernoulli equation. RA pressure can be estimated by the respiratory collapse of the inferior vena cava (IVC) seen on 2D echocardiogram. When the diameter of IVC decreases by 50% or more with inspiration, RA pressure is usually less than 10 mm Hg, and those with less than 50% inspiratory collapse tend to have an RA pressure higher than 10 mm Hg. In the absence of pulmonic stenosis, RV systolic pressure is equal to systolic pulmonary artery pressure (SPAP). In this study, pulmonary hypertension (PH) was defined as SPAP >35 mm Hg. Right ventricular diameter (RVD) was measured in the apical 4-chamber view at one thirds level from the base of the RV. Care was taken to obtain the image demonstrating the maximum diameter of the right ventricle without foreshortening. Tricuspid annular plane systolic excursion (TAPSE) was measured in m-mode by placing the cursor through the free wall of the RV at the level of tricuspid annuls in the apical 4-chamber view.[5]

Medical intervention

Considering the local policy and lower expenses, streptokinase (SK) was used as the thrombolytic agent. Prior to initiating thrombolytic therapy, activated partial thromboplastin time (aPTT), prothrombin time (PT), blood group, hemoglobin (Hb) and platelet count were assessed for each patient. Streptokinase (SK) was not administered in patients who had previously received SK or had a recent streptococcal infection. Streptokinase was administered as an IV bolus of 250,000 IU and an infusion of 100,000 IU/hr for 12–24 h.[6]

Unsuccessful thrombolysis within the first 36 h was defined as both persistent clinical instability and residual echocardiographic RV dysfunction. Persistent clinical instability was prospectively defined as the presence of at least two of the following criteria: refractory cardiogenic shock; systemic arterial hypotension (defined as systolic BP of $\leq$90 mm Hg or a pressure drop of $\geq$40 mm Hg for $>$15 min if not caused by new-onset arrhythmia, hypovolemia, or sepsis); severe hypoxemia (i.e., room-air pulse oximetry of $\leq$90% or PaO_2 without oxygen therapy of $\leq$55 mm Hg); or tachycardia (heart rate $\geq$110 beats/min). Residual RV dysfunction was defined as pulmonary vascular obstruction $>$30% at the 10th day after thrombolysis on right heart catheterization or Multi detector CTPA.[7,8]

Surgical intervention

Indication for surgical embolectomy included a central or para-central massive PE with additional criteria such as cardio-respiratory arrest, right heart thrombi, large patent foramen ovale (PFO), unsuccessful thrombolysis or contraindication for thrombolytic therapy.[7,8] In cases of thrombolysis failure, SE was performed within 72 h of the initial thrombolysis.

Surgical embolectomy was performed through a median sternotomy using mild hypothermic cardiopulmonary bypass (CPB). The clot was extracted through a longitudinal arteriotomy in the main pulmonary artery extending into the right or left PA branches, under direct vision using a special forceps. The right atrium and ventricle were explored routinely and all clot material was carefully removed. Coronary artery bypass grafting (CABG) and closure of PFO was also performed when required. Three days after intervention, the patients received warfarin with an International Normalized Ratio range between 2 and 3.

Statistical analysis

Patient characteristics were described using mean with standard deviation, or absolute numbers with percentages as appropriate. Mean comparisons were performed using *t*-test for data with normal distribution, and Mann–Whitney U for non-parametric data. Fisher's exact test was used for the comparison of predisposing factors and co-morbidities between the two groups. The trend over time within a group was analyzed using repeated measure ANOVA followed by Scheffe test for mean comparison. The probability level of significance was considered as <0.05 for all tests. The analyses were performed using SPSS V.11.5 software for windows.

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