ECHOCARDIOGRAPHY GUIDED THERAPY FOR MASSIVE INTRA-OPERATIVE PULMONARY EMBOLI DURING ARTERIO-VENOUS FISTULA/GRAFT THROMBECTOMY

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Abstract

Various techniques' are currently employed for thrombectomies of the arterio-venous (AV) fistula/ graft to restore flow. Sub massive or massive pulmonary emboli's have been reported following such procedures both intra-operatively and post-operatively. The hemodynamic responses depend not only on the size of the emboli's but also on the pre existing cardiac or pulmonary reserve of the patient. Rapid intra-operative echocardiography by anesthesiologist not only will help the clinicians with diagnosis but also can guide treatment plan as well as prognosis.

Conflict of interest: None

Key words: Pulmonary Emboli, Echocardiography, Hypotension, Intra-operative, Hemodialysis, Fistula

Introduction

Hemodialysis graft or fistula occlusion is a common event that occurs frequently in patient with end stage renal disease. As a result, the vast majority of dialysis patients with AV graft/ fistula thrombosis undergo surgery for thrombectomy[1-3]. Surgical approach and techniques include, thrombolysis[4], suction thrombectomy[5], balloon thrombectomy[6], mechanical thrombectomy with surgical devices[7] or combinations of these methods. Restoration of flow can be achieved with balloon angioplasty and in some instances endovascular stents[8-9]. Embolization could occur during extraction process and result in mild to severe cardio-respiratory complications[10].

Intra-operative echocardiography is not only a valuable tool for the anesthesiologist to diagnose pulmonary emboli (PE) but also help the surgical team to make appropriate treatment plan.

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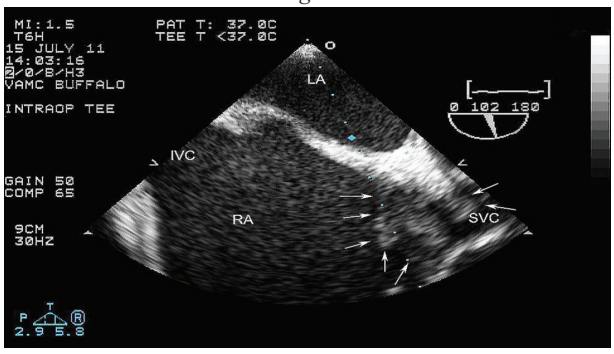
Case Report

We report a case of a 58 year old male with past medical history significant for end stage renal disease (ESRD), hypertension and liver disease secondary to hepatitis C infection. Surgical history was significant for multiple hemodialysis and AV graft/fistula thrombectomies. He was admitted to the hospital with the chief complain of non functioning AV fistula. Vascular team evaluations revealed AV fistula thrombosis and patient was scheduled for thrombectomy of AV fistula. Prior to surgery, bilateral lower extremities venous doppler ultrasound did not detect deep venous thromboses. He was brought to the operating room for attempted thrombectomy of the AV fistula. Vital signs on arrival to the operating room were blood pressure of 100/60 mm/Hg, heart rate of 110 beat /min, oxygen saturation of 97% and axillary temperature of 36.5 centigrade. On arrival to the operating room, he was placed on 100% oxygen via face mask. He was induced with propofol/ fentanyl/oxygen/air and trachea was intubated without difficulty. He was maintained on desflurane/air/ and 50% oxygen. He was placed on controlled mechanical ventilation with respiratory rate of 14 beat /minute, tidal volume of 600 ml, positive end expiratory pressure of 5 cmH_2O and end tidal CO2 of 25 mm/ Hg with pulse oximetery reading of 97% saturation. Arterial blood gas analysis showed pH = 7.32, PaCO2 = 35 mm/Hg, PaO2 = 120 mm/Hg. Operation begun with a longitudinal incision above the antecubital crease and dissection was carried down until fistula was identified. The basilic vein appeared to be pulsatile. An attempt was made to puncture the fistula and passed the wire in a retrograde fashion but unsuccessful. The fistula was then opened with a #11 blade and manually large amount of thrombus was evacuated from the basilic vein. A fogarty catheters was also passed through the basilic vein with return of brisk bleeding. A left brachio-basilic fistulogram was performed. The basilic vein appeared to be widely patent. A short period after thrombectomy patient vital signs became unstable with a drop in arterial oxygen saturation to low 60% with low end tidal CO_2 below 10 mm/Hg. Systolic blood pressure dropped below 40 mm/Hg. Patient was immediately placed on 100% oxygen and vasopressors and rapid infusion of crystalloids were started. Vasopressin infusion was started at 0.1 units per minute and norepinephrine at10 mcg/min to maintain a mean arterial pressure of 65 mm/Hg. Transesophageal echocardiography was performed intra-operatively and confirmed the presence of large pulmonary emboli in pulmonary artery [Fig. 1, 3] and superior vena cava [Fig. 2]. It was decided to start thrombolytic therapy with the tissue plasminogen activator (tPA) 100 mg IV over 2 hours. Patient was immediately transferred to MICU and his post operative course was complicated with respiratory failure with mechanical ventilation and hypotension requiring vasopressor therapy. He gradually recovered with supportive care and was transferred to floor a week later.

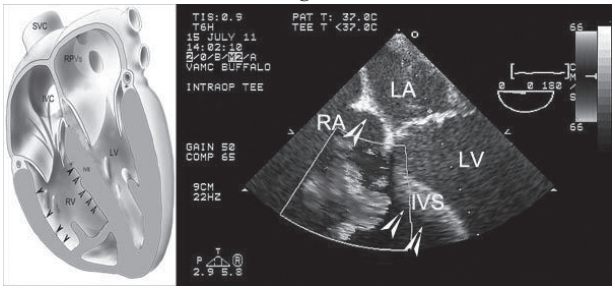
Fig. 1



TEE demonstrates several large emboli are observed in the superior vena cava. Arrows point to emboli.

TEE = Tranesophageal echocardiography, RA = Right Atrium, IVC = Inferior vena cava, LA = Left Atrium, SVC = Superior Vena Cava

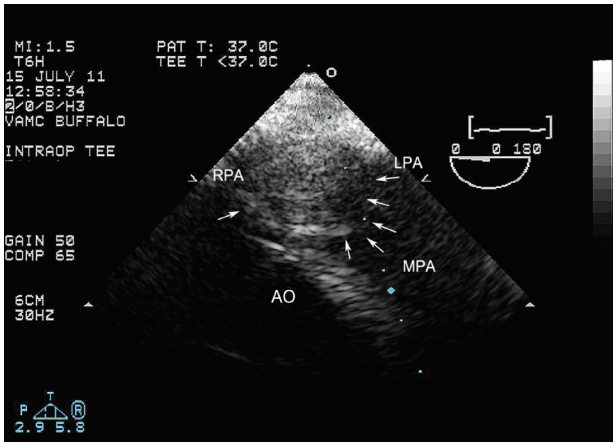
Fig. 2



TEE features of an acute PE includes: Right ventricular dilation, Tricuspid regurgitation, Ventricular septal wall bulging (arrow), Right atrial dilation

TEE = Trans-esophageal echocardiography, RA = Right atrium. LA = Left atrium, LV = Left ventricle, IVS = Intra-ventricular septum

Fig. 3



TEE = Transesophageal echocardiography indicate large emboli in the pulmonary artery (arrow)

AO = Aorta, RPA = Right pulmonary artery, LPA = Left pulmonary artery, MPA=Main pulmonary artery

Discussion

The United States annual renal report in 2008 indicated that a total of 112,476 patients started ESRD therapy, and the ESRD population reached 547,982 including 382,343 dialysis patients[11]. The vast majority of patients had interventional procedures for thrombosis of hemodialysis fistula/ graft in the USA[12-13]. To restore flow, catheter intervention techniques have become the primary mode of restoring flow in about 80% of cases[14]. Complications arising from such interventions include bleeding, PE, vein rupture, cerebral embolism and arterial embolism[10,15].

The true incidence and rate of successful lysis and outcome of massive intra-operative PE following such procedures remains largely unknown. Massive pulmonary embolism remains the most feared complication intra-operatively. Thrombolytic therapy remains the treatment of choice and surgical management is reserved for high risk patient[16]. Catheter embolectomies have also been reported and are currently limited to centers with specialized training and dedicated staff[17].

In general, standard diagnostic modalities utilized for evaluation of PE includes ventilation perfusion scan of lung[18], contrast enhanced computed tomography of lung with PE protocol[19] and echocardiography. Transesophageal (TEE) ot transthoracic echocardiography remains the most valuable tools in the operating room and can be useful in providing therapeutic and prognostic information[20-21]. Echocardiographic finding useful in diagnosis of PE include: Right ventricular dysfunction and dilation, dilated pulmonary artery, and reduced left ventricular size[22]. A recent study by Aymard et al suggested that right ventricule (RV) to left ventricule (LV) ratio of > 1.5 should be considered as the cut off value for allocating patients to surgical embolectomy[23]. TEE in our patient not only confirmed the presence of a saddle embolus in pulmonary artery [Fig. 3] and several smaller PE's in the superior vena cava (SVC) but also an RV/LV ratio of 0.9 favoring thrombolytic therapy. Thrombolysis with tPA in combination with heparin were initiated at the end of the operation after surgical homeostasis was achieved[24-26].

Other clues for supporting diagnosis of PE, includes the presence of a wide arterial to end tidal CO2 gradient 27 as well as hemodynamic instability. Echocardiography can play a valuable role and is a useful diagnostic test for early diagnosis, risk stratification, and management of patient with large PE.

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