DOI: 10.7860/JCDR/2019/39660.12454

Case Report

Obstetrics and Gynaecology Section

Systemic Thrombolytic Therapy for Pulmonary Embolism in Early Postoperative Period Following Laparoscopic Vaginal Hysterectomy

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ABSTRACT

A 51-year-old female underwent a laparoscopic-assisted vaginal hysterectomy for fibroid uterus. After an uneventful procedure, the patient developed symptoms of Pulmonary Thromboembolism (PTE). The PTE was managed with, systemic thrombolysis using tissue plasminogen activator (Tenecteplase), appropriate support of inotropes and anticoagulants. While there is less incidence of PTE in laparoscopic procedures, in particular, in gynaecology, there is a definite risk even in a patient with low-risk factors, which has been highlighted. Prompt diagnosis, aggressive therapy and adequate haemodynamic support go a long way to reduce mortality and morbidity from this life-threatening complication.

Keywords: Anticoagulants, Fibroid uterus, Tenecteplase, Venous thrombosis

CASE REPORT

A 51-year-old female patient was diagnosed as a case of fibroid uterus posted for elective Laparoscopic-Assisted Vaginal Hysterectomy and Bilateral Salpingo-Oophorectomy (LAVH-BSO). There was no history of medical illness or any significant family history and on examination, weight was 55 kg, heart rate-80/minute, BP-136/80 mmHg and airway examination was normal. Her pre-operative investigations were within normal limit.

After explaining the procedure, the risk involved with the procedure, a written informed consent was obtained from the patient and she was then shifted to Operating Room (OR). After the successful surgery, she was shifted out to postoperative Intensive Care Unit (ICU) with stable vitals.

On Postoperative Day 1 (POD-1), patient complained of giddiness and shoulder pain. On examination, patient was conscious, with PR of 120 bpm, BP 60/40 mmHg, RR 22/minute, SpO_2 85% with Hudson Mask providing oxygen at 6 L/minute. Chest revealed bilateral normal vesicular breath sound on respiratory examination, and for S1, S2 sounds in cardiovascular examination. One litre of 0.9% normal saline was given fast. With no improvement in vital signs, an arterial line was secured and noradrenaline at 0.01 mcg/kg/minute was started followed by dopamine at 2.2 mcg/kg/minute.

Patient became drowsy, with HR of 132 bpm, BP 80/60 mmHg, SpO_2 77% with oxygen at 10 L/minute and RR of 24/minute. Patient was intubated with endotracheal tube and was connected to the ventilator. The Electrocardiogram (ECG) showed sinus tachycardia, S1, Q3, T3, and ST depression in V4, V5, and V6. Inj. phenylephrine (200 micrograms), heparin 5000 IU and inj. morphine 2 mg were given, a minute apart. Emergency echo, D-Dimer, Creatine Kinase MB (CK-MB), Troponin T, Renal Function Test (RFT), CT-angiogram, and Arterial Blood Gas (ABG) were asked. Inj. dopamine (2.2 mcg/kg/minute), inj. noradrenaline (0.01 mcg/kg/minut), adrenaline (5 mL/hour) and Inj. morphine (2 mg) were started intravenously.

The ABG revealed mild acidosis; echo showed dilated Right Atrium (RA) and Right Ventricle (RV) with D-shaped septum. Severe Tricuspid Regurgitation (TR) and Pulmonary Arterial Hypertension (PAH) were seen along with Left Ventricular Hypertrophy (LVH) and normal systolic function. A provisional diagnosis of PTE was made and patient was advised CT-pulmonary angiogram. Left central line was secured, and inj. Dobutamine was started at 5 mL/hour along with heparin (800 IU/hour).

Troponin T-test showed value of 125 ng/L, CK-MB 16 IU/L, D-Dimer 8.1 mg/L, the CT-angiogram revealed dilated atrium, and hypodense filling defect at the origin of the bilateral descending pulmonary artery, more on the left than right, with extension into the left upper segmental bronchus. As diagnosis of acute PTE was confirmed Inj. Tenecteplase (30 mg) was given intravenously over 10 seconds. On POD-2, patient was started to wean from ventilator, two units of Packed Red Cells (PRC) were infused and heparin infusion was started.

On POD-3, all ionotropiv tapered and stopped. Finally, patient was extubated and put on oxygen at 5 L/minute with mask. With the activated Partial Thromboplastin Time (aPTT) exceeding 180 seconds, heparin was stopped. Another two pints of PRCs were infused. A final venous doppler showed a negative study, while the echo revealed mild dilated right atrium/ventricle, mild TR/no significant PAH, normal LV function and concentric LV hypertrophy. Inj. fondaparinux 2.5 mg SC was given before the patient was shifted to ward. Post-discharge patient was on warfarin for three months and then stopped.

DISCUSSION

PTE is an infrequent but serious complication in gynaecological surgeries. Major causes of postoperative morbidity and mortality in PTE is caused by occlusion in pulmonary artery circulation consequent to a clot formed elsewhere, in particular, the deep veins of the leg. A PTE complicated by haemodynamic instability is defined as a Massive Pulmonary Embolism (MPE) [1]. Since its occurrence is frequently referred to critical care service, there is a need for critical care physicians and anaesthesiologist to remain on constant alert to handle eventualities.

Authors found an overall prevalence of postoperative venous thromboembolism of 0.5% in patients admitted for surgery. The four risk factors were identified associated with venous thromboembolism after hysterectomy: body mass index, abdominal route of surgery, surgical time, and gynaecologic cancer as the indication for surgery.

Overall, the rate of postoperative venous thromboembolism for women undergoing abdominal hysterectomy was 1.1% which was

higher than rates seen with laparoscopic hysterectomy (0.3%) and vaginal hysterectomy (0.2%). No difference was seen between vaginal and laparoscopic hysterectomy [2]. In patients who have new onset or worsening dyspnoea, chest discomfort and persistent hypotension without other obvious causes should be suspected for acute MPE. Rapid diagnosis and treatment of MPE within a short time can decrease mortality and clinical probability assessment is the first utmost important step in diagnosis a PTE [3].

As a standard therapy approach, a spiral CT scan should be done as an investigation of choice in haemodynamically unstable patients who are in shock because of its high sensitivity in detecting pulmonary vessel emboli. Echocardiography is performed as an alternative in patients who are critically ill. Numerous approaches to managing acute MPE exist. These approaches include systemic thrombolysis, catheter-directed therapy and surgical embolectomy. In life-threatening PTE it is important to dissolve the clot rapidly, which is why thrombolytic therapy is still widely accepted [4]. Shen L et al., reported a similar case of postoperative MPE treated satisfactorily with alteplase, tissue-type Plasminogen Activator (t-PA) thrombolysis that occurs following a major thoracic surgical resection on 6th POD [5]. Whereas in the present case thrombolysis had to be done on 1st POD so risk of bleed from surgical site was high.

Currently, the American Heart Association (AHA) and American College of Chest Physicians recommend systemic thrombolysis as first-line therapy in patients with a massive PTE and consideration of thrombolysis in patients with a sub-massive PTE [4,6]. For high-risk patients with absolute contraindications to thrombolytic treatment, catheter-directed therapy or surgical thrombectomy is recommended [7]. However, catheter-directed thrombolysis has no clear advantage over intravenous thrombolysis [8]. Therefore, authors decided to administer standard-dose thrombolytic, Tenecteplase, to the postoperative patient with MPE. While recent surgery is generally a contraindication for thrombolysis, the reason for this was partly because successful reports have demonstrated the feasibility of full-dose thrombolysis in similar cases [9,10]. Lampert J et al., reported a case of massive PE treated with systemic thrombolysis using alteplase in a patient with recent neurosurgery for an intracranial neoplasm [11]. Another reason for using tenecteplase was that surgical or catheter thrombectomy was not available in the hospital and the patient was critically ill.

The 2008 guidelines of the European Society of Cardiology's Task Force for the Diagnosis and Management of Acute Pulmonary Embolism indicate absolute contraindications to systemic thrombolytic therapy for hemorrhagic stroke or stroke of unknown origin at the time of PTE, Central Nervous System (CNS) neoplasms, major trauma or surgery within the preceding three weeks, ischaemic stroke within six months, gastrointestinal bleeding within the last month, and known active bleeding [12].

Absolute contraindications to anticoagulation were considered relative in the setting of an MPE with unstable haemodynamic, in a patient who was proving refractory to other therapeutic interventions. For hospitals that lack surgical intervention capabilities, medical thrombolysis is the best treatment option [12]. Complications from this approach include an increased risk of serious bleeding, overall incidence of major haemorrhage is reportedly around 12%, in particular, there is a 1-3% incidence of intracranial haemorrhage that can be fatal in up to 50% of cases [13,14].

CONCLUSION

Early diagnosis, treatment strategy and haemodynamic support of the patient are the only major predictors of survival in Massive Pulmonary Embolism (MPE). Delaying appropriate therapies increases mortality In comparison with early medical thrombolysis or surgical intervention, both strategies are effective with a slightly increased survival probability with thrombolytic therapy.

REFERENCES

[1] Bagaria V, Modi N, Panghate A. Incidence and risk factors for development of venous thromboembolism in Indian patients undergoing major orthopaedic surgery: results of a prospective study. Postgrad Med J. 2006;82:136-39.
[2] Swenson CW, Berger MB, Kamdar NS, Campbell DA, Morgan DM. Risk factors for venous thromboembolism after hysterectomy. Obstetrics & Gynecology. 2015;125(5):1139-44.
[3] Arcasoy SM, Kreit JW. Thrombolytic therapy of pulmonary embolism: a comprehensive review of current evidence. Chest. 1999;115:1695-707.
[4] Jaff MR, McMurtry MS, Archer SL, Cushman M, Goldenberg N, Goldhaber SZ, et al. Management of massive and submassive pulmonary embolism, iliofemoral deep vein thrombosis, and chronic thromboembolic pulmonary hypertension. Circulation. 2011;123:1788-830.
[5] Shen L, Li Y, Hernandez-Arenas LA, Jiang G, Yang J. Successful treatment of a pulmonary embolism with low dose of tissue plasminogen activator after thoracic surgery. Am J Emerg Med. 2016;34(11);2259.e5-59.e6.
[6] Kearon C, Akl EA, Ornelas J, Blaivas A, Jimenez D, Bounameaux H, et al. Antithrombotic therapy for VTE disease: CHEST guideline and expert panel report. Chest. 2016;149:315-52.
[7] Agnelli G, Becattini C. Acute pulmonary embolism. N Engl J Med. 2010;363:266-74.
[8] Verstraete M, Miller GA, Bounameaux H, Charbonnier B, Colle JP, Lecorf G, et al. Intravenous and intrapulmonary recombinant tissue-type plasminogen activator in the treatment of acute massive pulmonary embolism. Circulation. 1988;77:353-60.
[9] Klaus S, Bahlmann L, Mierisch C, Heringlake M, Schmucker P. Early postoperative thrombolytic therapy after laparotomy. Resuscitation. 2001;50:353-55.
[10] Sayeed RA, Nashef SA. Successful thrombolysis for massive pulmonary embolism after pulmonary resection. Ann Thorac Surg. 1999;67:1785-87.
[11] Lampert J, Bikdeli B, Green P, Baldwin MR. Systemic thrombolysis in a patient with massive pulmonary embolism and recent glioblastoma multiforme resection. BMJ Case Rep. 2017;2017. pii: bcr-2017-221578.
[12] Cox JC, Jablons DM. Operative and perioperative pulmonary emboli. Thorac Surg Clin. 2015;25:289-99.
[13] Kanter D, Mikkola KM, Patel S, Parker JA, Goldhaber SZ. Thrombolytic therapy for pulmonary embolism. Frequency of intracranial hemorrhage and associated risk factors. Chest. 1997;111(5):1241-45.
[14] Levine MN, Goldhaber SZ, Califf RM, Gore JM, Hirsh J. Hemorrhagic complications of thrombolytic therapy in the treatment of myocardial infarction and venous thromboembolism. Chest. 1995;108(4):364S-73S.

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FINANCIAL OR OTHER COMPETING INTERESTS: None.

Date of Submission: **Sep 17, 2018**
Date of Peer Review: **Oct 27, 2018**
Date of Acceptance: **Nov 10, 2018**
Date of Publishing: **Jan 01, 2019**