Catheter-based therapy for acute pulmonary embolism: Lifesaving in a clinical dilemma!

Sir,

22-year-male, (body mass index 34) with Guillain-Barre Syndrome was on treatment with plasmapheresis. He suddenly became hemodynamically unstable on the 5th day of hospitalization. Arterial blood gas (ABG) showed hypercapnia and later hypoxia. Electrocardiograph showed sinus tachycardia. Echocardiography revealed hypokinesia of the right ventricle and moderate tricuspid regurgitation with pulmonary artery systolic pressure (PASP) of 45 mmHg. The patient continued to be hypoxemic, hypotensive, and suffered a cardiac arrest. In view of deteriorating status, the patient was taken for pulmonary angiography with the intention to diagnose as well treat pulmonary embolism in the same setting. After confirming the diagnosis, mechanical fragmentation was initiated in the embolus followed by intralesional urokinase [Figure 1]. Over the next 24 hrs, hemodynamic parameters gradually improved with the resolution of shock and reduction in pulmonary artery pressure to 27 mmHg.

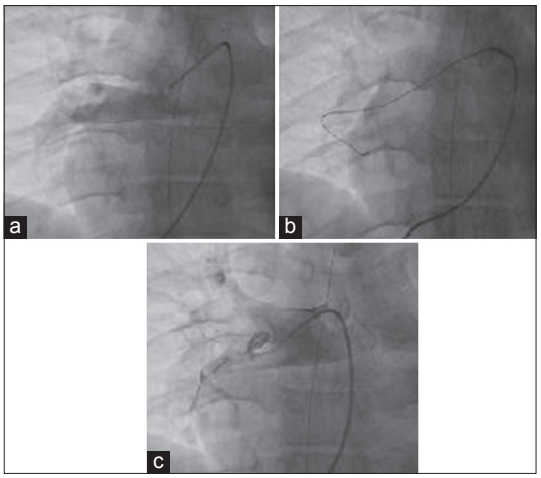

Figure 1: Pulmonary angiography (a) total cut-off of right pulmonary artery (RPA) and clot in left PA (b) mechanical breakdown and intrapulmonary urokinase administration (c) postprocedural pulmonary angiography revealing restoration of pulmonary flow in PA and its branches

The true incidence of Pulmonary embolism (PE) is unknown because the diagnosis of PE is more often missed than confirmed due of varying clinical presentations. Provisional diagnosis of pulmonary embolism is usually based on the clinical setting and the pretest probability guides the choice of confirmatory tests.

This case was at high risk for pulmonary embolism as he was obese with quadriplegia and had been immobilized for almost 1-week. ABG showed respiratory acidosis which in not a common finding in patients of PE, but our patient had neuromuscular paralysis of respiratory and accessory muscles which precluded him from hyperventilation and resulted in respiratory acidosis as the first change in ABG. Thus, the presence of respiratory acidosis on ABG does not exclude the possibility of PE as quoted in one of the recent articles rather blood gas must never be analyzed alone but should be correlated to the clinical scenario.[1]

Acute pulmonary embolism is pulmonary embolism resulting in hypotension, shock or need for cardiopulmonary resuscitation (CPR) and is recognized as the third major cause of death among the hospitalized patients. Hence, immediate attention and treatment is crucial. The American college of chest physicians consensus statement have laid down recommendations for management of PE but not more than two-thirds of the patients receive systemic thrombolysis because it is contraindicated (absolute or relative) in a large number of clinical conditions.[2] Since the first description of embolectomy catheter in 1971, the clinical application of catheter-based interventions in the management of acute PE are still very infrequent. There are no randomized controlled trials to compare its efficacy with systemic thrombolysis, with or without anticoagulation. In a recently published meta-analysis, only 594 cases of acute massive PE were treated with catheter-directed therapy (CDT), with a success rate reported to be about 86%.[3]

Although our patient was a candidate for systemic thrombolysis, but refractory arterial hypotension and the need for CPR led to the selection of CDT as the modality of choice. Two hour period required for infusion of full dose of tPA and about 20% risk of major hemorrhage following its infusion also led to the selection of CDT as the first line therapy.

The catheter-based fragmentation leads to mechanical debulking which can be clinically seen as an immediate improvement in hemodynamics, resolution of shock, and reduction in PASP. However, breakdown of the clot provides a large exposed area for subsequent thrombolysis. The thrombolytic infusion following debulking results in decreasing the incidence of chronic PE and pulmonary hypertension. FDA

has approved urokinase (4400 IU/kg intravenous over 10 min, followed by 4400 IU/kg/h for 12 h) and streptokinase (250,000 IU intravenous over 30 min, followed by 100,000 IU/h for 24 h). It is a plasminogen activator with the additional quality of a catalytic agent that renders fibrinogen less clottable by thrombin. Furthermore, in terms of cost and availability it was chosen as the preferred agent.

This case highlights that clinicians should have a low threshold for diagnosing PE in acutely bed ridden patients. In a selected group of patients with neurological disorders where systemic thrombolysis is contraindicated, combination of mechanical fragmentation and local thrombolysis may be a promising option for reduction in mortality.

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DOI: 10.4103/0972-5229.158297