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# Spontaneous Hemopericardium Post-Thrombolysis in Stroke

**Mohammad S N**

**Department of Critical Care Medicine, Sakra Hospital, Bengaluru**

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**Corresponding author: Mohammad S N**
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**Abstract**

Use of intravenous (IV) tissue plasminogen(tPA) as a fibrinolytic therapy for acute ischemic stroke is a standard acute phase treatment. The common complication of tPA is hemorrhage, especially intracranial. But there are other rare sites of bleeding. We present one such rare case of a patient developing spontaneous hemopericardium after IV tPA for acute ischemic stroke.

**Keywords:** coagulopathy, hemopericardium, Ischemic stroke, tissue plasminogen activator

## Introduction

All eligible acute ischemic stroke patients should be thrombolysed within 3 hours of last known normal and to a more selective group of eligible acute stroke patients, (based on ECASS III exclusion criteria) within 4.5 hours of last known normal.[(1)] tPA is the most commonly used agent for thrombolysis, given its neurological benefits at 3 months and long term.[(2)]But, complications are also associated with the use of tPA.

Hemorrhage is one such serious complication of tPA. However, the coagulation factors that may potentially increase the risk of bleeding, after tPA, are not well understood. But, an early transient coagulopathy associated with elevated International normalized ratio (INR) in stroke patients treated with tPA has been reported. [(3)] The term post- tPA coagulopathy is used for an early transient coagulopathy associated with documented elevated INR> 1.5 within 24 hours of tPA infusion without a known cause. [(3)]

## CASE REPORT:

A 61-year-old gentleman, known case of hypertension, presented to Emergency department with sudden onset of left sided weakness since 1.5 hours with progressive weakness leading to inability to walk or hold objects by left hand. No history of slurred speech, chest pain, loss of consciousness or dyspnea.

Patient was hemodynamically stable, with a GCS-15/15. Pupils were bilaterally equal and reactive to light. Power on left upper limb was 0/5 and left lower limb was 2/5.

An MRI brain (stroke protocol) was done (figure-1), which suggested an acute right frontal and parieto-occipital- right Middle cerebral artery (MCA) territory patchy infarcts.

Neck Doppler done suggested bilateral multiple plaques in carotid bulbs and proximal Internal Carotid Artery without significant stenosis. The right vertebral artery was hypoplastic with non- visualization of distal vertebral artery Va segment.

Electrocardiogram showed ST depression in leads 1, aVL and tall T waves in chest leads, Troponin I-0.02ng/ml, INR -1.2

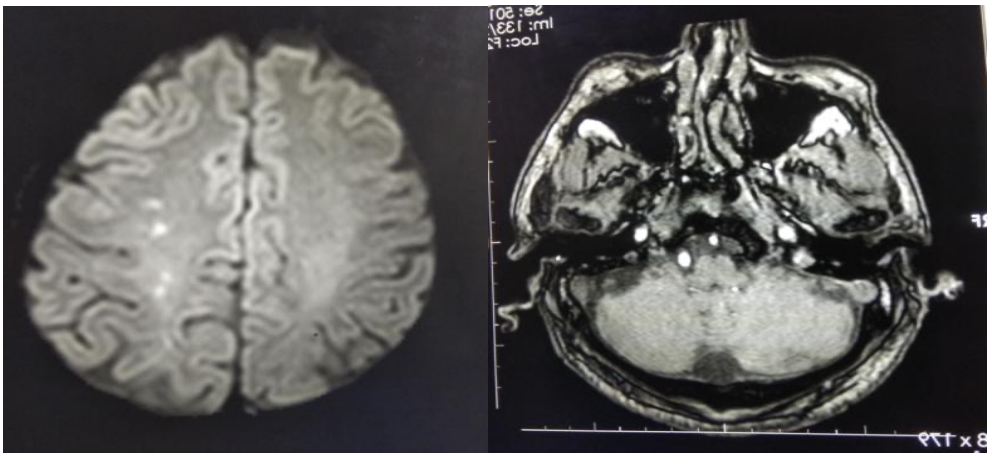

**Figure 1: DWI MRI Images showing acute right MCA territory infarcts**

As the patient presented within the window period for thrombolysis, he was administered IV tPA after obtaining consent. Gradually, left lower limb power improved to 3/5 over 3 hours and patient remained hemodynamically stable. At the end of 3 hours, patient developed hypotension (Blood Pressure-74/40mm Hg). Hypotension was managed with fluid resuscitation. Pupils were bilaterally equal(2mm) and reactive to light and there was no further worsening of neurological deficit.

A bedside 2D Echocardiography (figure 2) was suggestive of concentric Left Ventricular (LV) hypertrophy, grade 1 LV Diastolic dysfunction, mild pericardial effusion around Right Ventricle, Right Atrium, Apex(0.9cm) and anterolateral wall(0.6cm) with Ejection Fraction 0.55, non- collapsing Inferior venacava and no regional wall motion abnormality.

After around half an hour, patient developed a second episode of hypotension (70/30 mm Hg) requiring vasopressor support and drop in GCS(E2V3M3) with reactive and normal pupils. There was no obvious source of external bleeding and peripheries were cold and clammy. A CT Brain was planned but had to be deferred as the patient was hemodynamically unstable.

A repeat bedside 2D Echocardiography done suggested further significant increase in the size of pericardial effusion with cardiac tamponade physiology-Right Atrial, Right Ventricular collapse.

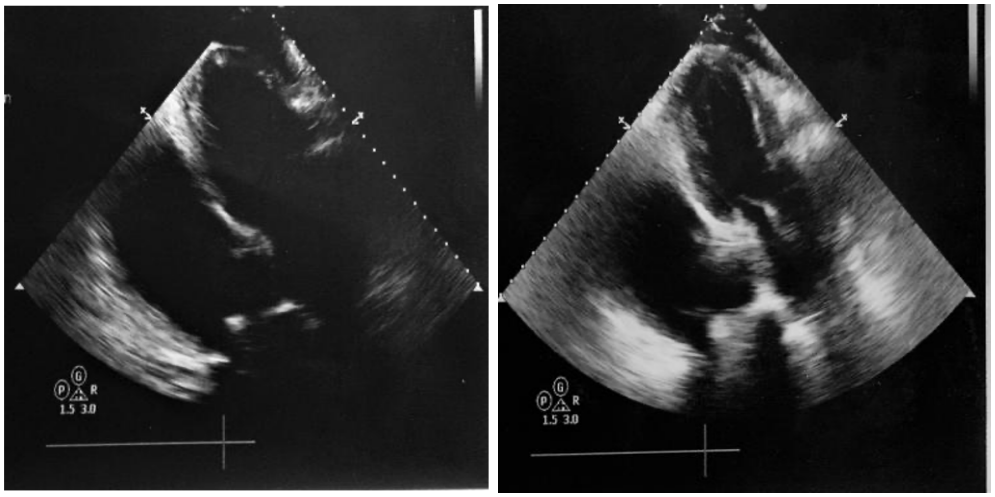

**Figure 2: 2D Echo showing a pericardial effusion.**

An emergency fluoroscopy guided pericardiocentesis(figure-3) was done by the Cardiology team, under Fresh frozen plasma cover (repeat INR-1.96) and around 380ml of hemorrhagic pericardial fluid was drained. During the procedure, patient developed severe respiratory distress requiring endotracheal intubation, ventilatory support and increased vasopressor support.

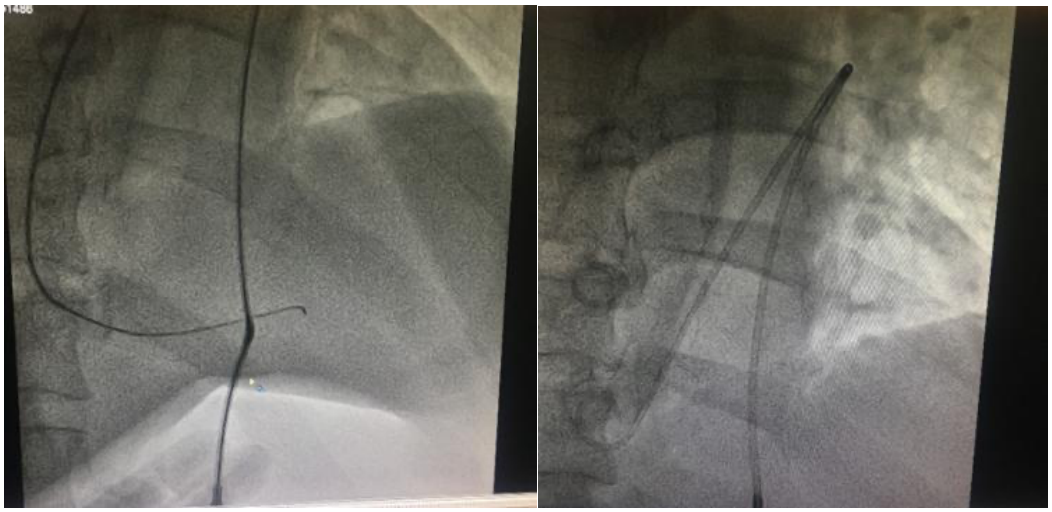

**Figure 3: Fluoroscopy guided pericardiocentesis**

Patient had developed refractory shock and couldn't be salvaged despite all aggressive measures.

**Discussion:**

tPA derivatives are the most commonly used thrombolytic drugs because of their relatively high selectivity for activating fibrin bound plasminogen, which cleaves to render plasmin. Plasmin in turn breaks down the fibrin molecules leading to dissolution of clots. It may also degrade coagulation factors like fibrinogen, factors V and VIII and generate breakdown products which interfere with fibrin polymerization.

Normally, circulating alpha2- antiplasmin inactivates plasmin, but therapeutic doses of tPA generate enough plasmin to overwhelm the alpha2-antiplasmin.

Though tPA is relatively selective for clot associated fibrin, it can produce bleeding complications through its action on non-fibrin bound plasminogen. Intracerebral hemorrhage is much more commonly seen than other sites of systemic bleeding, post-thrombolysis with tPA.

In the National Institute of Neurological Disorders and Stroke (NINDS) trials, symptomatic intracerebral hemorrhage within 36 hours after the onset of stroke occurred in 6.4% of patients who were given tPA, within 3 hours of onset of symptoms, with computed tomography (CT) documented hemorrhage vs 0.6% in patients who received placebo. As against this, the incidence of major systemic hemorrhage is approximately 1.6% of tPA treated patients. Sites of systemic hemorrhage reported are gastrointestinal hemorrhages, urinary tract hemorrhages and fatal cardiac tamponade to name a few. [(4)]

In our case, the patient had developed a spontaneous hemopericardium followed by a cardiac tamponade. This was attributable to post- tPA coagulopathy, an under recognized early transient coagulopathy associated with a documented elevated INR>1.5 within 24 hours of tPA without a known cause. [(3)]Our patient had a normal INR-1.2 pre-thrombolysis and an

INR of 1.96 done about 4 hours post-thrombolysis (repeated as we suspected hemopericardium since the 2D Echo done suggested a mild pericardial effusion), thus fulfilling the criteria. The pericardial effusion was not explainable by any other cause as we had ruled out ischemic heart disease (normal Troponin-I- 0.02ng/ml with no regional wall motion abnormality on 2D Echo), pulmonary embolism (no right atrium or right ventricular dilatation, no pulmonary artery hypertension on 2D Echo) and there was no history of trauma. The suspicion of hemopericardium was substantiated by the fact that hemorrhagic fluid was drained during pericardiocentesis.

In the case reports published [5,6,7] in the past, patients either had a recent ischemic heart disease or undetected myocardial or pericardial disease. What sets our case apart is that our patient neither had any ischemic heart disease nor anything suggestive of a pericardial disease. Thus, we concluded that this was a case of spontaneous hemopericardium which developed post- thrombolysis with tPA, in a case of ischemic stroke.

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