Successful management of acute coronary syndrome with cardiogenic shock, severe mitral regurgitation and multi-organ dysfunction: A case report

Abstract

Acute coronary syndrome (ACS) with cardiogenic shock has worst prognosis. Early revascularization has been shown to be beneficial in cases who present early; but many a times, patients present late after ACS and their hemodynamics is compromised by systemic inflammatory response syndrome and multi-organ dysfunction. We hereby present a case of anterior wall myocardial infarction with severe left ventricular systolic dysfunction (left ventricular ejection fraction (LVEF):27%) and severe mitral regurgitation (MR), who presented late after ACS and was in cardiogenic shock, pulmonary edema and had acute kidney injury on presentation. The patient was initially managed conservatively with dual oral antiplatelets, statins, glycoprotein IIb/IIIa inhibitors infusion (tirofiban), parentral anticoagulation, inotropes, diuretics, and mechanical ventilation. She was taken up for coronary angiography and percutaneous coronary intervention to left anterior descending artery and chronically occluded left circumflex artery during same admission after stabilization of hemodynamics. She tolerated the procedure well and her repeat echocardiogram (done after two weeks) showed LVEF of 50% and mild MR. This case highlights an alternative approach to manage ACS with cardiogenic shock, who presents late after acute event.

Key words: Acute coronary syndrome, Acute kidney injury, Anterior wall myocardial infarction, Cardiogenic shock, Glycoprotein IIb/IIIa inhibitors, Multi organ dysfunction syndrome, Mitral regurgitation,

Pulmonary edema

Background

Cardiogenic shock (CS) after acute coronary syndrome (ACS) carries poor prognosis with an estimated mortality of 50 to 60%, despite early revascularization and mechanical circulatory support.[1] Though the SHOCK (Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock) trial seems to justify and recommend early invasive strategy in ACS patients with CS,[2,3] there are many studies which question the safety and benefit of early intervention in this group.[4,5,6,7,8] Besides, this strategy may not be best for the late presenters where the hemodynamics has been further compromised with multi organ dysfunction and systemic inflammatory response syndrome (SIRS). Most of our ACS patients, in developing countries, have delayed presentation and such a cohort of ACS patients with CS & delayed presentation has not been represented well in randomized controlled trials. We hereby describe a case of ST elevation anterior wall myocardial infarction (AWMI) with CS and acute pulmonary edema in Killip class IV, who presented more than 72 hours after symptom onset and was managed successfully with initial conservative approach, followed by revascularization in same admission.

Case Presentation:

Fifty years old female, a known case of diabetes type 2, hypertension, chronic kidney disease (baseline creatinine: 2.1 mg/dL) and hypothyroidism presented with acute onset anginal chest pain of 3 days duration associated with class 4 dyspnea and decreased urine output. Her electrocardiogram (ECG) revealed sinus rhythm, ST elevation in V1 to V3 and ST depression in inferolateral leads s/o ST elevation AWMI (as shown in Figure 1a). At admission, she was in Killip class IV with blood pressure of 84/56 mm of Hg on inotropes, heart rate of 116/minute, respiratory rate of 28/minute, and saturation of 70% at room air. She had bilateral diffuse crackles, gallop rhythm and a soft pan-systolic murmur of grade 3/6 at apex. Echocardiogram revealed severe eccentric mitral regurgitation (MR) with severe

hypokinesia in left anterior descending artery (LAD) and left circumflex artery (LCx) and an left ventricular ejection fraction (LVEF) of 27%. Reverse transcription polymerase chain reaction (RT-PCR) for COVID-19 was negative. Blood investigations revealed leucocytosis (14900/mm^3), Troponin T of 14.8 pg/ml and BNP of 1232 pg/ml and deranged renal function with creatinine of 3.9 mg/dl and urea of 89 mg/dl. Arterial blood gas showed hypoxia with high anion gap metabolic acidosis (lactic acid 2.4 mmol/l). Chest X-ray showed white out lungs with bat wing appearance suggestive of diffuse pulmonary edema (as shown in figure 2a).

She was managed with mechanical ventilation, inotropic support along with dual oral antiplatelets (DAPT), statins, glycoprotein IIb/IIIa infusion (tirofiban), parentral anticoagulation and gastric mucosal protection. Her hospital stay was complicated with sepsis and worsening of renal functions which was managed as per the guidelines. She responded to the treatment; renal function tests improved (creatinine 4.7 to 2.0 mg/dl), pulmonary edema resolved (as shown in Figure 2b) with decrease in severity of MR. After hemodynamic stabilization, she was taken up for coronary angiogram which revealed triple vessel disease with diffuse 80% stenosis in left anterior descending (LAD) artery and chronic total occlusion (CTO) of left circumflex artery (LCx) and right coronary artery (RCA) (as shown in Figure 3 a,b,c and Video 1,2,3). She was offered coronary artery bypass grafting (CABG) as first option for revascularization, however as the patient and relatives were unwilling for open heart surgery, she was taken up for angioplasty to LAD and LCx and planned for staged ischemia driven revascularization of RCA. LAD lesion was stented with long tapered drug eluting stent (DES) 3.0 x 2.5 x 50 mm (Biomime Morph, Meril life sciences^{TM}, India) with TIMI III flow (as shown in Figure 3d). LCX CTO was successfully crossed with Gaia II (ASAHI^{TM} Intech, Aichi, Japan) wire using microcatheter (as shown in Figure 3e,f) followed by Sion Blue guide wire (ASAHI^{TM} Intech, Aichi, Japan). Lesion preparation was initially

done with 1.25 x 8 mm semi-complaint balloon, followed by a 2.0 x 20 mm non-compliant (NC) balloon. Two overlapping DES 3 x 32 mm and 2.75 x 20 mm were deployed from proximal to distal LCX and post dilated with 3.0 x 15 mm NC balloon at 14 to 16 atmosphere with TIMI III flow both in LAD and LCX. (as shown in Fig 3g,h). The procedure was uneventful with a total fluoroscopy time of 22 minutes, procedural time of 56 minutes and radiation dose of 524 mGy. Total contrast used was 130 ml. She was continued on DAPT and statins. Two weeks post procedure, her echocardiogram revealed improved LVEF OF 50 % with mild MR (as shown in Figure 4).

Discussion:

CS complicates 7-10% cases of ACS and is associated with significant mortality. With better medical care and revascularization, the mortality in ACS with CS has reduced from 70-80% to 40-50%.[1,9,10] Though the SHOCK trial revealed improved survival upto six years with early revascularization, patients in the trial presented to medical set up at much shorter time, such that median time from ACS to shock was 5-6 hours, medial time to intervention after randomization was 1-2 hours. However in real world scenario, especially in developing countries, median time to appropriate care in best of the centers is at least 24 hours.[11] Besides >30% patients with ST elevation myocardial infarction (STEMI) in developing countries scenario are late presenters.[12] A delayed presentation in STEMI is associated with extensive necrosis and worse outcomes.[13] In present COVID era, the delay in presentation of ACS to hospitals is further increased due to fear of contracting viral infection and due to decreased availability of public transport. Since unprepared health care system was overwhelmed with the pandemic, the delay in revascularization was further augmented by unmet needs of logistics and skilled manpower.[14,15] The index patient had chest pain for 3 days before presentation to our center, and additional four hours were required for screening the patient for COVID-19. Though all efforts were made to provide maximum supportive

care at earliest medical contact, the activation of catheterization services was often delayed due to obvious logistical reasons.[14,15]

ACS with left ventricular dysfunction is the most common cause of CS. A loss of more than 40% of functional myocardium is required to produce CS.[16] Besides mechanical complications like ventricular septal rupture, free wall rupture, and papillary muscle rupture or dysfunction may also contribute to CS after ACS.[17] The patients with CS who survive to discharge have similar outcomes compared to those who have stable hemodynamics, so it is important that their early survival be improved.[18] A long standing shock produces a state of SIRS and MODS further compromising already depressed cardiac mechanical function and leads to a vicious cycle. The late presenters with CS, usually have acidosis, inappropriate vasodilation resistant to vasopressors, compromised renal and lung functions; besides being in a state of inflammation and hypercoagulation. Sepsis was suspected in 18% of the SHOCK cohort and those with culture positive sepsis had twice the risk of death.[19] The most accepted theory of septicemia in these patients is decreased perfusion of intestinal tract which enables transmigration of bacteria into bloodstream.[20]

Though early revascularization and mechanical circulatory devices have reduced the mortality in patients of ACS with shock from 80 – 90% to below 50%;[21,22] the mortality in late presenters is still very high and the consensus on management is far from reached. Early invasive management in such patients may further compromise the already depressed cardiac and renal function without any additional advantage.[4-8] Any intervention in such a state has a very high baseline risk of mortality and failure. For same reasons, this subgroup of patients have always been excluded from trials studying the role of early revascularization in ACS.[23] Pre-hospital ambulance thrombolysis or primary percutaneous coronary intervention (within 2 hours of symptoms onset) can remarkably decrease the incidence of cardiogenic shock[24], but for those patients presenting late to the health care facilities, medical stabilization offers a

plausible approach with equal rates of in-hospital mortality.[1] The medical stabilization in mechanical complications like ventricular septal rupture after ACS has been well described.[25]

Management of patients with CS and MODS requires intensive care with support of failing organs including lungs and kidneys.[26] The role of parentral antiplatelets in emergency PCI has been studied in emergency settings when oral absorption of drugs is doubtful. Intravenous infusion of glycoprotein IIB/IIIA inhibitors and stenting were independently associated with improved outcomes in CS.[27] We used Tirofiban infusion (in renal modified dosages) in our patient prior to angioplasty which might have influenced the outcome, as we were not confident about the adequate absorption of oral drugs during the initial phase of admission. The patient was taken up for angiography and angioplasty only after hemodynamic stabilization and could achieve optimal technical results. This case highlights the challenges faced during management of ACS patients with CS, who present late after the symptom onset. This is one of those patients where we did not pursue the urgent coronary angiography & revascularization; rather we improved the hemodynamics conservatively followed by definite management. The existing literature and guidelines on management of patients who present late after myocardial infarction and have CS, are highly insufficient and require further studies.

Ethical approval

Ethics committee approval was not sought since all the treatment was according to acceptable norms.

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Legends

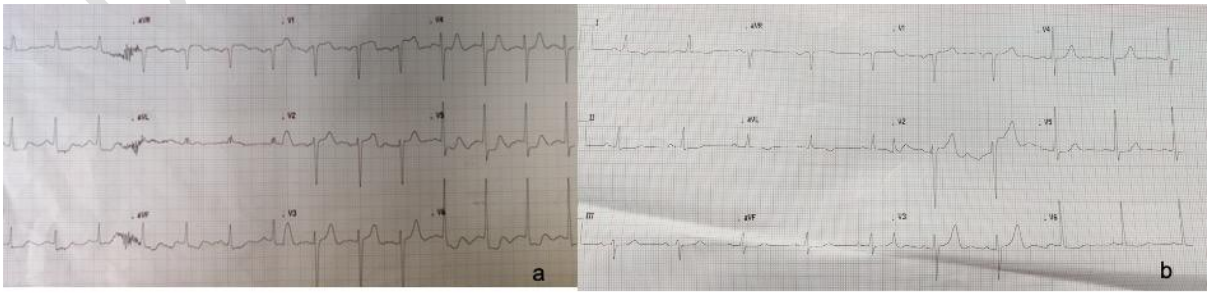

Figure 1a: Electrocardiogram (ECG) showing ST elevation anterior wall myocardial infarction

Figure 1b: Resolution of ECG changes post PCI

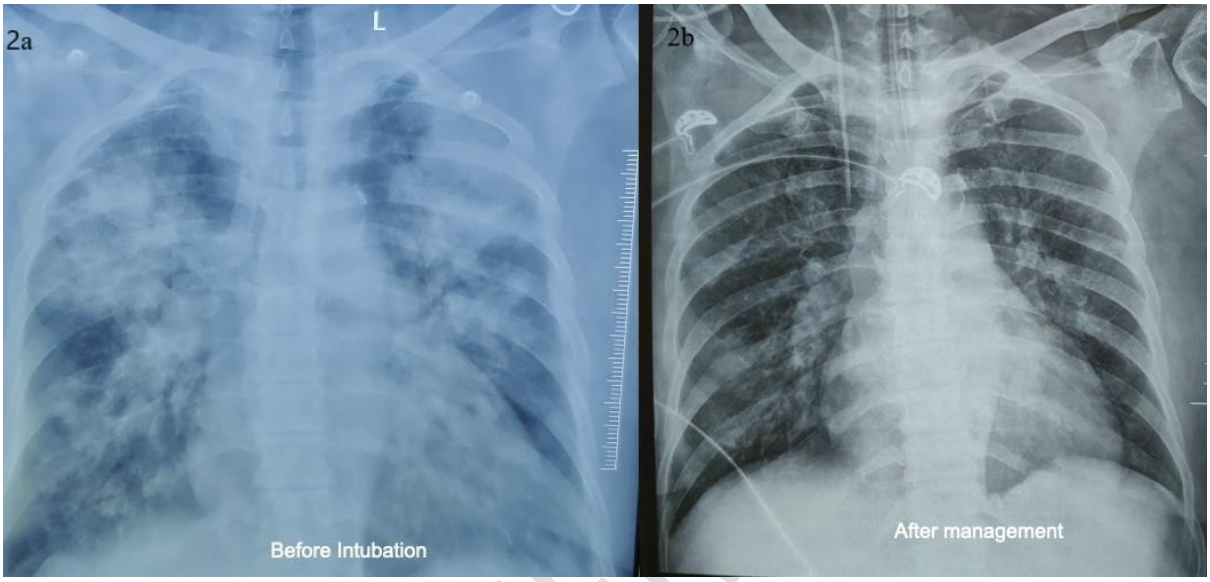

Figure 2a: Chest X ray showing diffuse infiltrates with bat wing appearance

Figure 2b: Chest X ray showing radiological resolution following diuretics and mechanical ventilation.

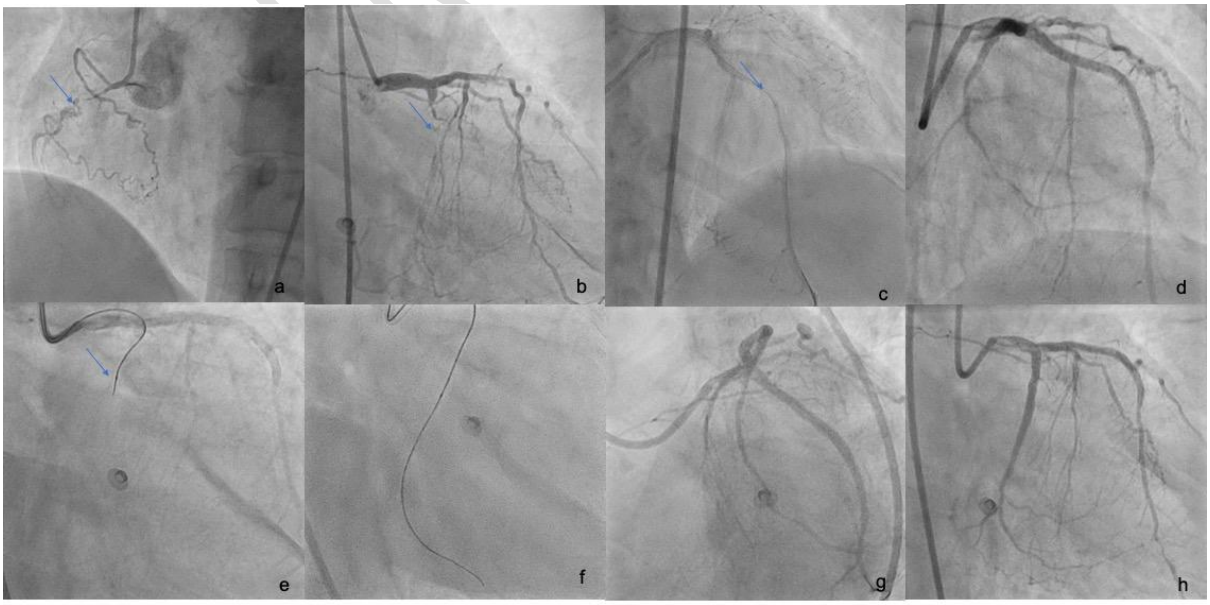

Figure 3a: Right coronary artery showing proximal total occlusion, 3b: Left Circumflex artery showing proximal chronic total occlusion, 3c: Diffusely diseased Left anterior descending artery with a stenosis of maximum 90%, 3d: Post Drug Eluting Stent (DES) Implantation in Left anterior Descending artery with TIMI III flow, 3e: Coronary Guide Wire with Microcatheter in Left circumflex artery, 3f: Coronary guide wire crossed the CTO and parked in major obtuse marginal, 3g: Final result of left circumflex artery post DES implantation, 3h: Final result post stent implantation in LAD and LCx arteries

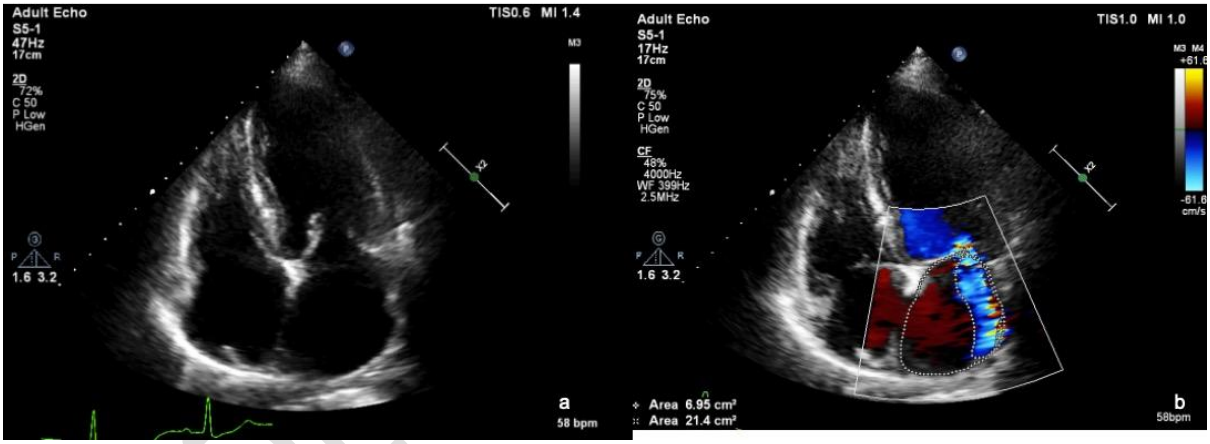

Figure 4: Echocardiogram images showing mild mitral regurgitation (post revascularization).

Video 1: Coronary Angiogram Left system (Anterior Posterior Caudal)

Video 2: Coronary Angiogram Left system (Anterior Posterior Cranial)

Video 3: Coronary Angiogram Right system