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# Dyspnoea with ticagrelor : case report

Dr. Ajinkya Bhosle, Dr. Anil Potdar

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**ABSTRACT:** Dyspnoea in acute coronary syndrome (ACS) patients has always been considered a challenging clinical scenario for diagnosis and treatment. P2Y12 platelet receptor inhibitors (i.e. clopidogrel, prasugrel and ticagrelor) are currently forms the mainstream treatment of ACS patients. Although dyspnoea is a common ticagrelor side effect with the increasing use of ticagrelor in these patients due to its beneficial effects on ischaemic event prevention and mortality, diagnostic and management policies can get tricky. The present case report focused on management of one such suspected ticagrelor induced dyspnoea patient.

**Keywords** : acute coronary syndrome , dyspnoea, ticagrelor

## I. CASE PRESENTATION

75-year male known case of hypertension got admitted with complaints of chest pain with sweating since 2 hours and no other significant history. On examination, his heart rate was 80-85/min, sinus in rhythm with ST-T changes in V1-V4 of ECG and blood pressure of 130/70 mm hg with clear lungs and no murmur on auscultation. 2D Echo showed Ejection fraction of 25% with apico-septal hypokinesia. Patient was then loaded with dualantiplatelets(tabletecosprin 300mg and tablet clopidogrel 300 mg) and statin (tablet atorvastatin 80mg) along with supportive therapy in the form of oxygen and pain killers.

Patient was immediately taken for emergency angiography and subsequently angioplasty was done with 2 drug eluding stents in proximal left anterior descending branch of coronary artery in same sitting. Patient was shifted to ICU for further management with IV infusion of nor-adrenaline to maintain MAP more 65mm hg and IV tirofiban infusion 0.15mcg/kg/min for 18 hours. Post procedure, patient was started on tab brillinta (ticagrelor) 90mg twice a day, tablet ecosprin 75 mg after lunch daily, tablet atorvastatin 40 mg once at night and tablet Ramipril 1.25 mg after lunch daily.

On day 3, patient developed non-productivecough with an episode of mild hemoptysis and signs of bilateral crepitations with reduced right sided basal air entry, moist mucous membrane, dry skin.No pallor / pedal edema. Patient progressive developed dyspnea at rest with tachypnea of more than >25/min and saturation of less than 92% on Oxygen supplementation via venturi mask, Arterial blood gas (Abg) showed signs of hypoxemic failure,hence NIV (noninvasive ventilation) was started.Patient did not have notable fever, wheeze, chest pain or abdominal distension. Patient was holding his hemodyanamics with sinus rhythm throughout.

HRCT chest was done which showed right sided mild to moderate effusion with passive atelectasis and consolidation of the basal segment of right lower lobe. Mild left pleural effusion and eventration of right hemidiaphragm was noted.USG chest showed lung comet score of 56, IVC congested and non tappable pleural fluid. Repeat 2D ECHO showed EF 25% with no dilated chambers, no valve abnormality, grade 2 diastolic dysfunction.This along with high NT PRO-BNP raised suspicion of left ventricular failure. Thus, fluid restriction of less than 1200ml and Iv frusemide 20mg twice daily was advised.

On blood investigations, total WBC counts were slightly raised with neutrophilic predominance but normal procalcitonin andnegative forblood, sputum and urine culture.SARS-COV-2 RNA also was not detected. Remaining blood investigations were unremarkable and non-contributory to establish a diagnosis which included normal thyroid, hepatic functions.

On day 4 and 5 with worsening hypoxia and more episodes of hemoptysis, lead to worsening signs of failure. Patient was conscious but required dubutamine and nor-adrenaline support to maintain MAP more 65mmhg and noninvasiveventilator support to maintain saturation of more than 92% with FiO2 40-60%. X ray chest showing signs of pneumonitis.

Ticagrelorsensitiy testing was suggested and brillinta was stopped and tab prasugrel 10mg daily once was started in place. On day 6-7 patient patient's condition improved, was slowly weaned off from inotropes and NIV. With no more episodes of hemoptysis. Patient was continued on

dual antiplatelets, statin, ACE inhibitors and supportive therapy.

As patient's lung functions improved, he was mobilized out of bed and Shifted to ward on day 9 of admission.

## II. DISCUSSION

In the RESPOND study,[1] ticagrelor therapy was associated with greater platelet inhibition as compared with clopidogrel treatment in both 'responders' and 'non-responders' to clopidogrel, as defined by the pharmacodynamic response to a clopidogrel loading dose in ACS patients.

Dyspnoea is one of the distressing symptoms experienced by patients. It can result from a variety of conditions, including cardiac, pulmonary, renal and liver diseases, anaemia and metabolic abnormalities. A substantial proportion (at least 25%) of patients with ACS may present with dyspnoea as the predominant symptom.[2]

Dyspnoea is a common side effect of ticagrelor, and Studies have suggested that ticagrelor inhibits the sodium-independent equilibrative nucleoside transporter–1, which may increase adenosine plasma levels and explain drug-related dyspnea.[3]

In phase 2 studies, ticagrelor was associated with a dose-dependent incidence of dyspnoea of 10 to 20%, compared with 0–6.4% in patients treated with clopidogrel.[4,5] Ticagrelor-related dyspnoea is frequently presented as sudden and unexpected air hunger or unsatisfied inspiration with or with haemoptysis. With most episodes being reported as mild, its pattern varies widely; from very brief episodes lasting minutes, generally starting in the first week of treatment, to sustained or intermittent episodes occurring over several weeks.[6]

The evaluation of a patient with dyspnoea continues to be dependent on a thorough history and physical examination. Frequently, the diagnosis of ticagrelor-related dyspnoea is based on exclusion. In addition to the patient's interview regarding similar symptoms, the exclusion of alternative dyspnoea causes must be performed clinically and through various diagnostic modalities.

We should keep in mind that, if a patient was initially selected for ticagrelor therapy due to a perceived high-risk profile, there are no meaningful reasons to downgrade the antiplatelet strategy to clopidogrel, except in the case of either a dangerous or an intolerable side effect or development of a contraindication to ticagrelor as proposed ticagrelor-associated dyspnoea management recommendations based on current knowledge.[7]

## III. ACKNOWLEDGMENTS

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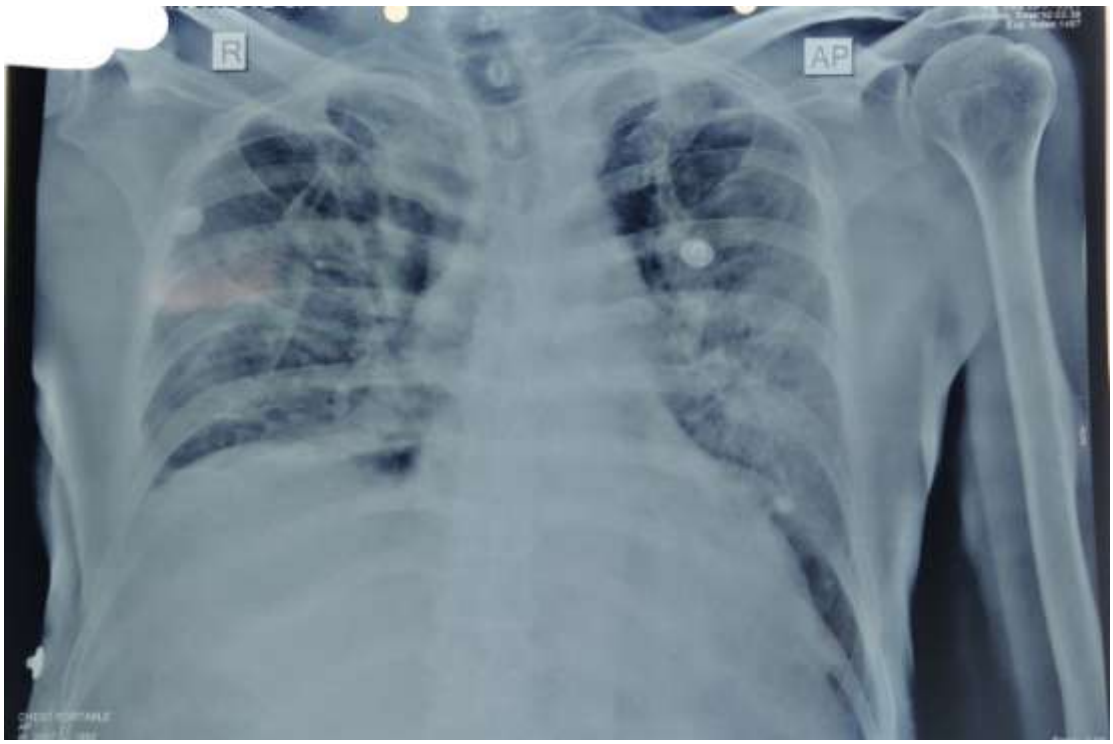


**Figure 1** day 3 xray chest

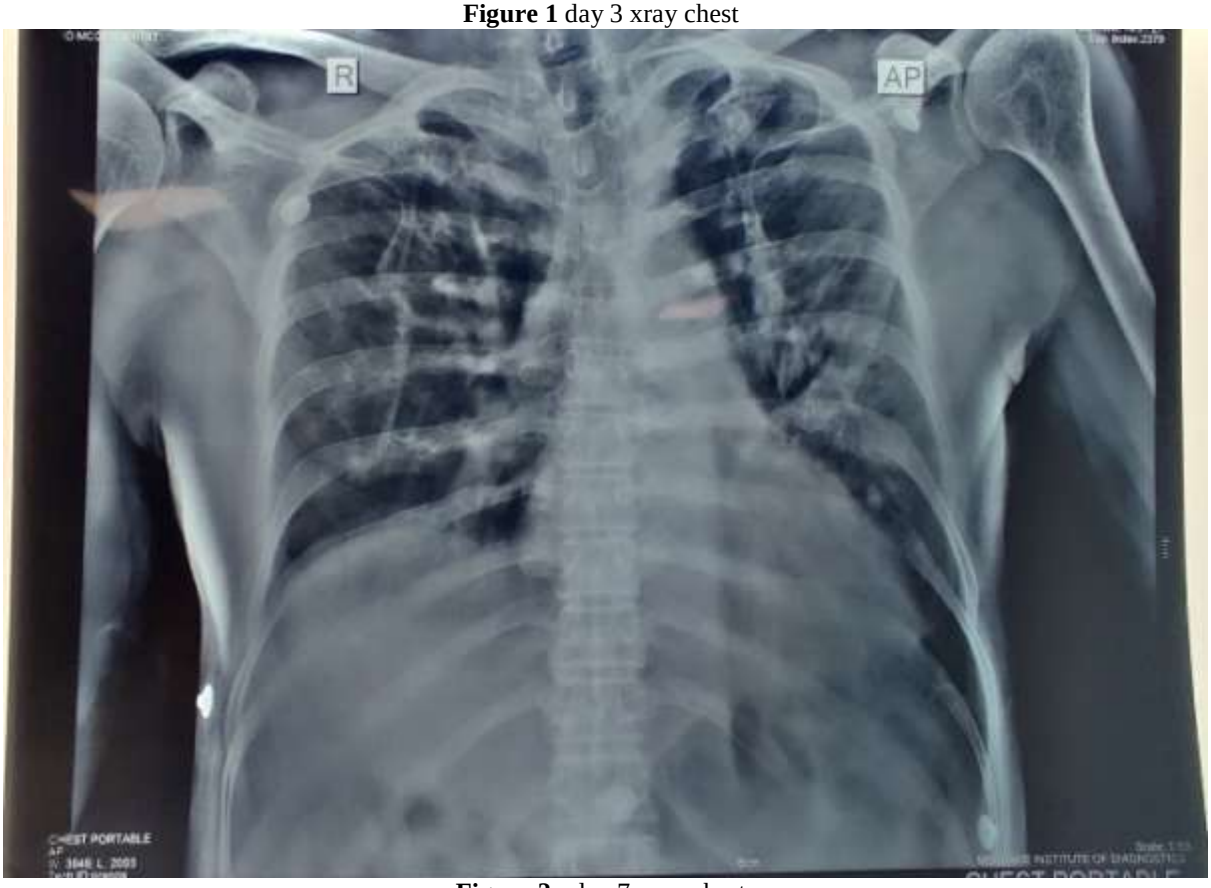


**Figure 2** : day 7 xray chest