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SECONDARY POLYCYTHEMIA WITH CONCOMITANT PULMONARY AND AORTIC THROMBOEMBOLISM IN A CHRONIC SMOKER: A RARE CLINICAL PRESENTATION

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ABSTRACT

Secondary polycythemia is a frequent consequence of chronic hypoxemia, yet its association with simultaneous arterial and venous thromboembolism is exceptionally rare. This case report describes a 65-year-old male, a chronic smoker with a 30-pack-year history, who presented with progressive abdominal distension and worsening dyspnea. Physical examination revealed severe hypoxia (SpO2 69% on room air) and central cyanosis. Laboratory analysis confirmed significant erythrocytosis with a hemoglobin of 18.7 g/dL and a packed cell volume (PCV) of 69%. While D-dimer was markedly elevated at 3827 ng/mL, lower limb Doppler excluded deep vein thrombosis (DVT).Echo showed mild pulmonary hypertension, Computed Tomography Pulmonary Angiography (CTPA) subsequently identified thrombi in both the pulmonary artery and the aorta. Extensive workup, including negative JAK2 mutation screening (V617F and Exon 12) and a reactive bone marrow study, excluded primary polycythemia vera. The findings emphasize the hypercoagulable risk inherent in severe secondary polycythemia and the importance of systemic vascular imaging in the presence of unexplained thromboembolic markers.

KEYWORDS: Secondary Polycythemia, Pulmonary Embolism, Aortic Thrombus, Chronic Smoking, Hyperviscosity.

INTRODUCTION

Polycythemia represents an increase in red blood cell mass, which can be broadly classified into primary and secondary etiologies. Primary polycythemia, or polycythemia vera, is a myeloproliferative neoplasm characterized by clonal proliferation, whereas secondary polycythemia arises from elevated erythropoietin levels, typically driven by chronic hypoxemia. Chronic obstructive pulmonary disease (COPD) and long-term smoking are among the most common triggers for secondary erythrocytosis. While the physiological increase in red blood cells is initially a compensatory response to improve oxygen delivery, extreme elevations in hematocrit significantly increase blood viscosity.

Increased blood viscosity, combined with the pro-inflammatory and pro-thrombotic effects of chronic tobacco use, predisposes patients to both arterial and venous thrombosis. While venous thromboembolism (VTE) is a recognized complication of hyperviscosity, the simultaneous occurrence of pulmonary embolism and arterial thrombi in high-flow vessels like the aorta is highly unusual. This case highlights the diagnostic complexity involved in managing severe erythrocytosis and the necessity of distinguishing between primary and secondary forms to guide appropriate therapy.

Detailed Case Presentation and Investigation

A 65-year-old male farmer presented with a one-month history of insidious, progressive abdominal distension and acute-on-chronic breathlessness of one-week duration (MMRC Grade 3). The patient had a 30-pack-year smoking history and was a known hypertensive on treatment for six months. On admission, he was hypoxic with an SpO2 of 69% on room air, which corrected to 95% with 6 liters of supplemental oxygen. Physical

examination revealed central cyanosis of the tongue, elevated JVP, and bilateral pitting pedal edema. Respiratory examination showed scattered bilateral crepitations, while the abdominal examination noted uniform distension without shifting dullness or palpable organomegaly.

Initial investigations revealed a hemoglobin of 18.7 g/dL and a PCV of 69%, which remained consistently high throughout the admission. Peripheral smear confirmed a normocytic normochromic blood picture with increased hematocrit and no atypical cells. ABG analysis indicated respiratory acidosis (pH 7.2) with significant hypercapnia (PCO2 68 mmHg).

Biochemical markers showed a D-dimer of 3827 ng/mL and a normal CRP of 6. Despite the high D-dimer, lower limb venous Doppler was negative for DVT. However, CT chest and PFT confirmed emphysematous changes, and CTPA revealed the presence of both pulmonary and aortic thrombi. Bone marrow aspiration and biopsy showed a cellular marrow with a 2:1 M:E ratio and erythroid hyperplasia, but were otherwise reactive. Genetic testing for JAK2 mutations (V617F and Exon 12) was negative.

Differential Diagnosis

The primary differential diagnosis focused on the etiology of the patient's polycythemia. Polycythemia vera (PV) was a significant consideration given the high hematocrit and the presence of multi-system thrombosis. However, the lack of splenomegaly, a normal platelet count, and negative JAK2 mutations argued against a primary myeloproliferative disorder.

The patient’s smoking history and emphysematous lung changes pointed strongly toward secondary polycythemia due to chronic hypoxemia.

Regarding the thromboembolic presentation, the differential included DVT with secondary pulmonary embolism (PE). However, the negative lower limb Doppler led to the consideration of *in situ* pulmonary artery thrombosis triggered by hyperviscosity. The discovery of an aortic thrombus necessitated ruling out other systemic causes of arterial thrombosis, such as occult malignancy, paraneoplastic syndromes, or heparin-induced thrombocytopenia. USG abdomen and CECT of the chest and abdomen were normal, ruling out solid organ malignancy.

Criteria, Treatment Given, and Prognosis

The diagnosis of secondary polycythemia was established using the WHO criteria for erythrocytosis, specifically by confirming the absence of PV through negative JAK2 testing and the presence of a clear secondary trigger (chronic smoking and emphysema).

Treatment was initiated with therapeutic anticoagulation for the pulmonary and aortic thrombi. Simultaneously, the patient underwent therapeutic phlebotomy to acutely reduce the PCV from 69% to a target range below 50%, aiming to decrease blood viscosity and prevent further clotting. Oxygen therapy was maintained to manage the underlying hypoxemia. The patient's prognosis is fair, provided there is strict adherence to smoking cessation and long-term anticoagulation management. The presence of an aortic thrombus remains a high-risk factor for systemic embolization, requiring long-term monitoring.

DISCUSSION

This case illustrates the profound hematological alterations that can occur in response to chronic smoking. The patient's PCV of 69% represents a critical level where blood viscosity increases exponentially, creating a state of rheological failure. In secondary polycythemia, the increase in red cell mass is often considered a compensatory mechanism to improve.

oxygen-carrying capacity in the face of lung disease. However, when the hematocrit exceeds 55–60%, the disadvantage of increased viscosity begins to outweigh the benefit of increased oxygen content, leading to impaired microcirculatory flow and stasis.

The occurrence of an aortic thrombus is particularly noteworthy. While arterial thrombi are typically associated with atherosclerotic plaque rupture, the hyperviscous state in this patient likely permitted the formation of a thrombus even in a high-flow vessel like the aorta. This suggests that the hemodynamic stress of severe polycythemia is sufficient to overcome the natural antithrombotic properties of the arterial endothelium. Furthermore, the *in situ* formation of pulmonary thrombi, suggested by the absence of peripheral DVT, underscores that hyperviscosity is a systemic pro-thrombotic state affecting both the venous and arterial circuits.

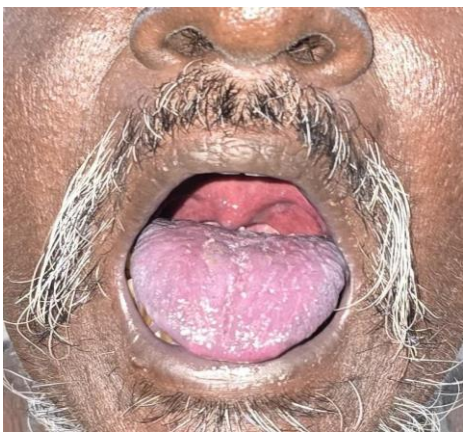
Treatment must balance the reduction of hematocrit with the maintenance of adequate oxygen delivery. While phlebotomy is the cornerstone of managing hyperviscosity, overly aggressive reduction in a patient with underlying emphysema could theoretically worsen tissue hypoxia.

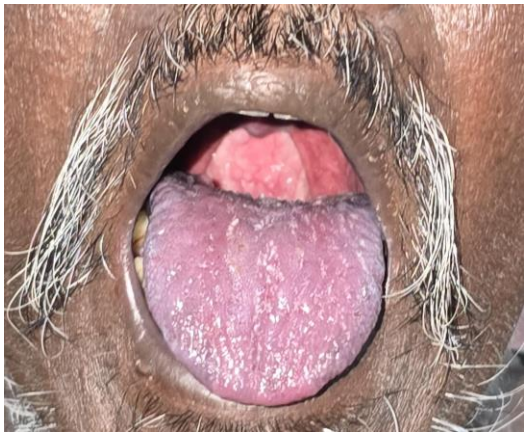
Therefore, the target PCV is often slightly higher in secondary cases (e.g., 50–52%) compared to the <45% target in PV. Anticoagulation remains essential, though its efficacy may be hampered by the physical characteristics of hyperviscous blood, necessitating careful monitoring of coagulation profiles.

CONCLUSION

In conclusion, severe secondary polycythemia induced by chronic smoking can manifest as a catastrophic hypercoagulable state involving both the pulmonary and systemic arterial circulation. This case demonstrates that the absence of traditional markers like the JAK2

mutation does not preclude a patient from developing life-threatening thrombosis. Clinicians should maintain a high index of suspicion for multi-site thromboembolism in patients with extreme erythrocytosis, especially when a high D-dimer is present despite negative peripheral venous studies. Effective management requires a combination of phlebotomy, anticoagulation, and aggressive treatment of the underlying hypoxic trigger.





Investigations

complete blood count

CBC	15/5	20/5	21/5	24/5	26/5	31/5	3/6	6/6
TC	11.8	14.3	13.2	8.9	8.3	6.9	7.2	7.4
RBC	7.4	6.5	6.8	6.9	6.3	6.7	6.12	6.36
HB	18.7	16.5	17.4	18.2	16.7	16.5	16	16.3
PCV	69	61	63	64	59	62	57	59
MCV	93	91	94	91	92	90.6	94	92.5
PLATELET	144	127	149	166	184	221	233	263

RFT

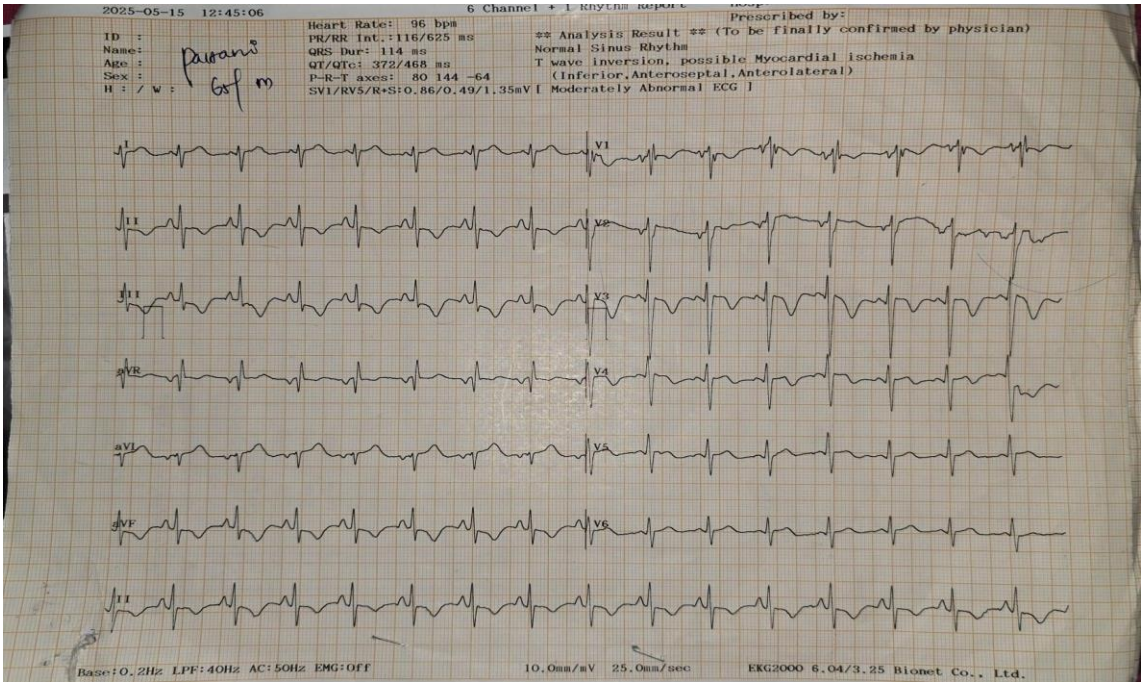
Date	16/5	17/5	20/5	25/5	31/5	2/6
UREA	27	39	25	31	44	23
CREATININE	0.7	0.9	1	0.8	1.0	1.12
SODIUM	139	142	139	130	128	143
POTTASIUM	4.9	3.5	3.8	3.3	4.6	3.8
RBS	187	198	311	108	146	162

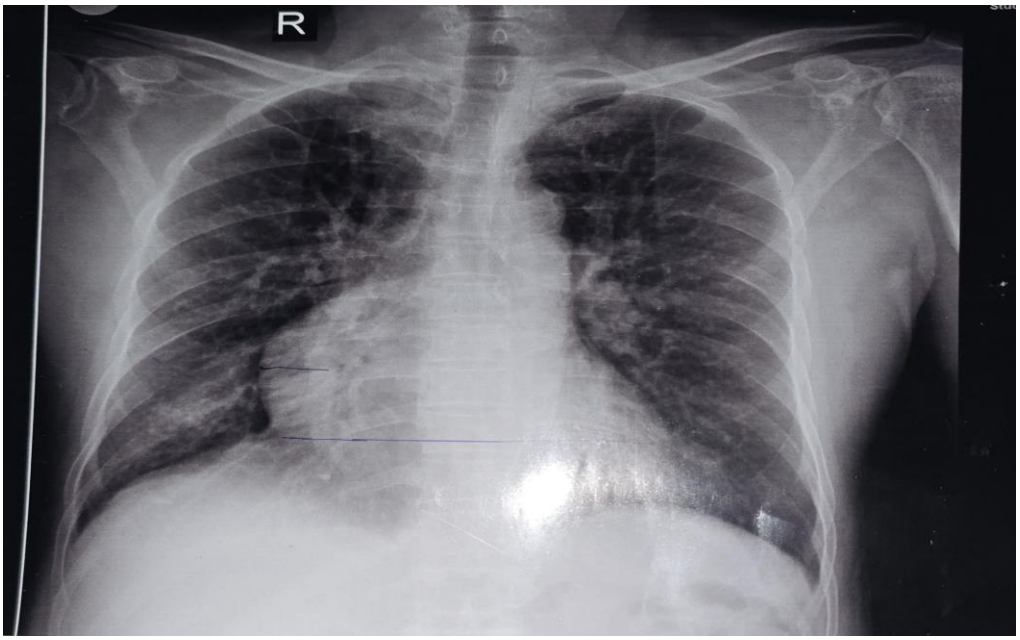
LFT

Date	17/5	20/5	25/5	26/5	31/5	2/6
TOTAL BILIRUBIN	2.8	2	1.7	0.7	2.1	1.8
DIR. BILIRUBIN	1.6	1.09	0.8	0.2	1	0.9
IND. BILIRUBIN	1.2	1	1.1	0.5	1.1	0.8
SGOT	44	30	65	31	39	92
SGPT	28	21	26	45	17	47
ALP	45	50	66	43	42	66
TOTAL PROTEIN	5.1	5.35	5.8	7.8	5.1	6.1
ALBUMIN	3.0	2.55	2.7	5.2	2.5	3.5
GLOBULIN	2.1	2.8	3.2	2.6	2.7	2.7

Coagulation profile

Date	15/5	17/5	26/5	31/5	2/6	3/6
PT	19.6	15.5	13.2	19.2	19.7	16.6
APTT	38.4	32.8	24.6	29.6	52	58
INR	1.05	1.14	0.82	1.5	1.5	1.2





- ESR- 10 FBS- 65 D dimer- 3827
  PPBS -280
  HBA1C-8.1 SERUM ELECTROPHORESIS:
- CRP -6 NORMAL PATTERN
- CKN -48
- CKMB -23 ABG ANALYSIS

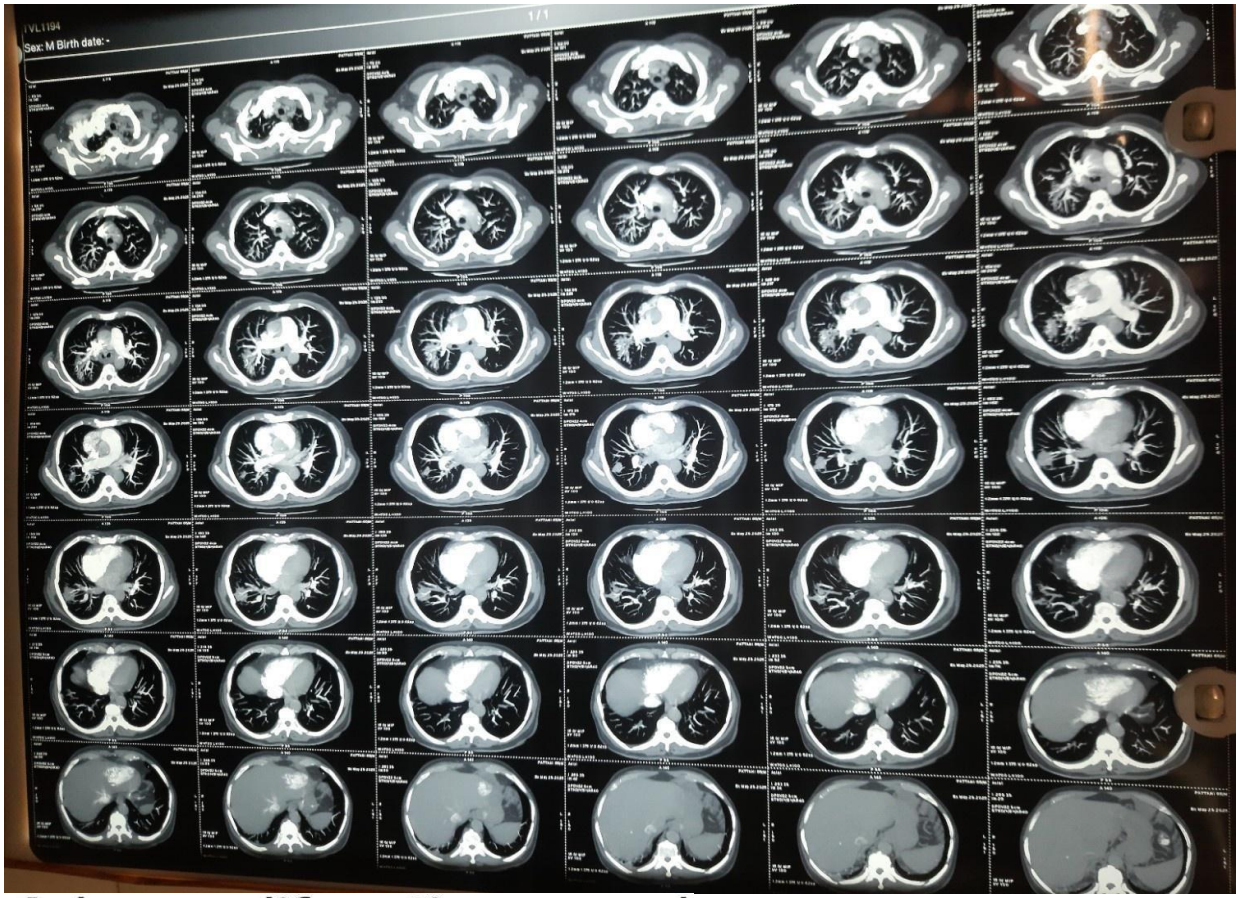
- HHH- NEGATIVE PH- 7.2
  PO2- 110
- TOTAL CHOLESTROL-132 PCO2- 68
- TGL-148 HCO3 - 28.5
- HDL-42 TCO2- 30.6
- LDL-60 GLU- 129
- VLDL-30 LAC- 1.5
- Cardiac: normal Echo
- Respiratory :ct chest and pft shows Emphysematous changes
- Polycythemia :primary or secondary as Epo was normal
- D dimer elevated
- Dvt was suspected but Doppler was normal
- Malignency was suspected CECT chest and abdomen done and it was normal
- CTPA revealed both pulmonary and aorta thrombus
- Detailed echo done it came to be normal
- For Primary polyccythemia bonemarrow and jak2 mutation done it was also normal

PERIPHERAL SMEAR STUDY:
RBC-Shows normocytic normochromic cells.
No hemoparasites are seen.
WBC-Normal in count, morphology and distribution.No atypical cells seen .
Platelet-Adequate.
IMPRESSION: NORMOCYTIC NORMOCHROMIC BLOOD PICTURE WITH INCREASED HEMATOCRIT

Bone marrow studies

- Bone marrow aspiration:
- Cellular marrow M:E RATIO - 2:1
- ERYTHROID SERIES-hyperplastic and Shows normoblastic pattern of maturation
- MYELOID SERIES - Shows normal pattern of maturation MEGAKARYOCYTES - Adequate
- IMPRESSION - REACTIVE MARROW STUDY WITH ERYTHROID HYPERPLASIA .

- **BONE MARROW BIOPSY :Reactive marrow**

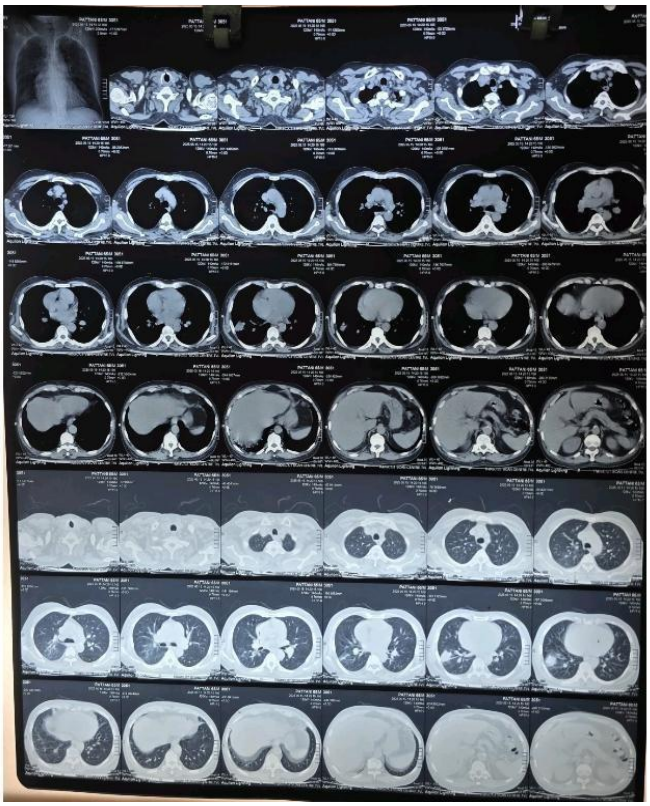


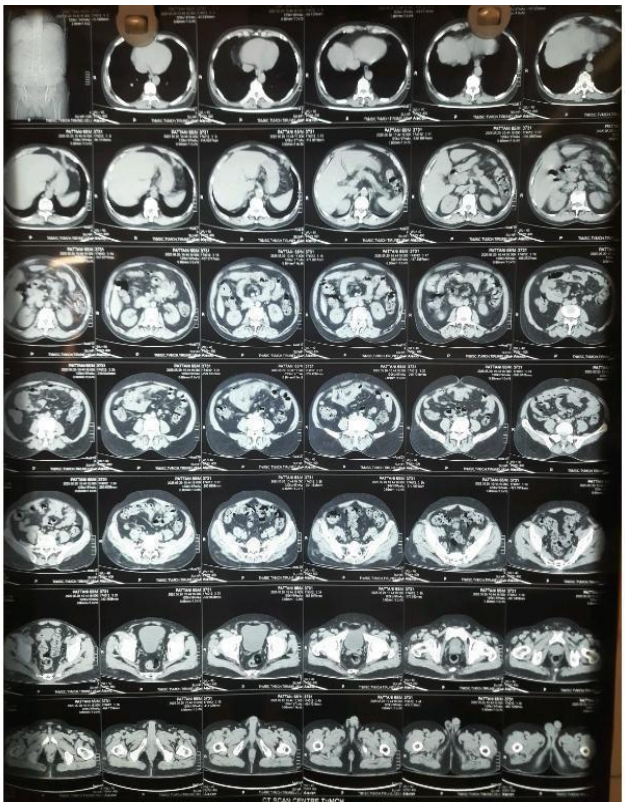
Myleoproliferative panel

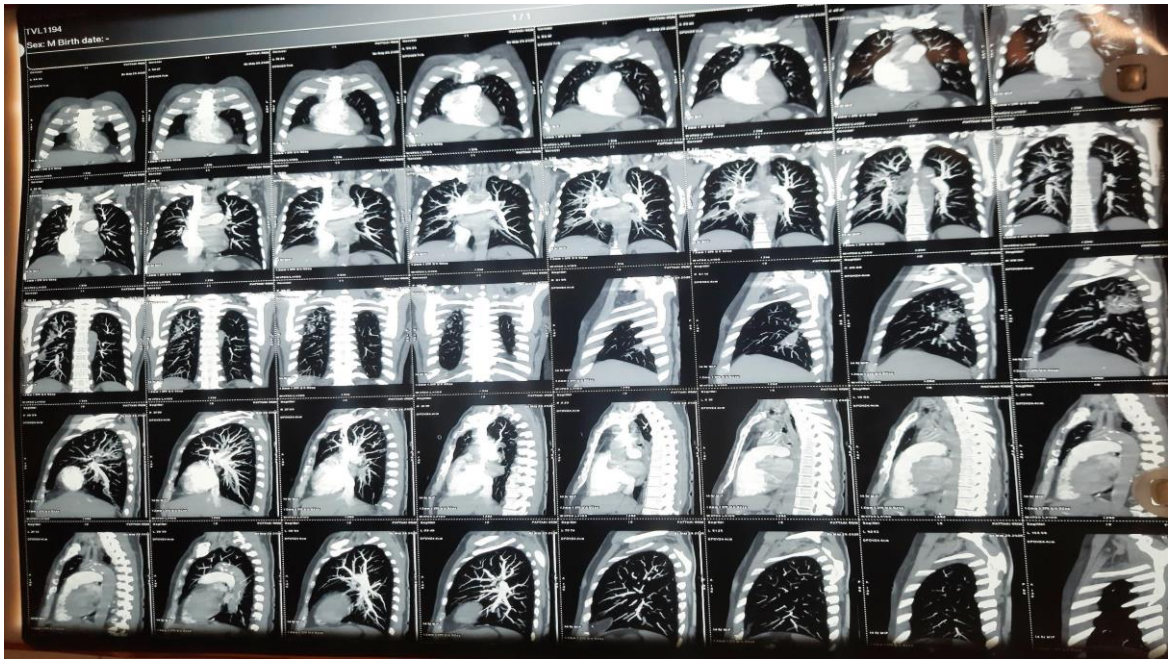
JAK 2

1.V617F/G1849T- NOT DETECTED

2.EXON 12 -NOT DETECTED







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