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A Rare Complication: Cardiac Arrest and Pulmonary Embolism in Polyarteritis Nodosa

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Abstract:- Polyarteritis nodosa (PAN) is a rare systemic vasculitis affecting small and medium-sized arteries. While PAN primarily involves arterial vessels, its association with venous thromboembolic events such as pulmonary embolism (PE) and deep vein thrombosis (DVT) and their consequences such as cardiac arrest are infrequently reported. Understanding the clinical presentation and management of PAN complicated by PE, and cardiac arrest is crucial for optimizing patient care. We present a case of a patient with a history of polyarteritis nodosa (PAN) who presented with a cardiac arrest complicated by pulmonary embolism (PE). Through meticulous analysis and multidisciplinary intervention, the patient's condition was stabilized, shedding light on the complexities of managing such rare and life-threatening complications within the context of PAN. This case underscores the importance of prompt recognition, comprehensive evaluation, and targeted therapeutic interventions in navigating the intricate interplay of vasculitis, thromboembolic events, and cardiac complications in patients with Polyarteritis nodosa.

Keywords:- *Polyarteritis Nodosa, Cardiac Arrest, Pulmonary Embolism, Vasculitis*

I. INTRODUCTION

Polyarteritis nodosa (PAN) is a rare autoimmune disease characterized by systemic inflammation of medium-sized arteries. It is characterized by segmental inflammatory lesions, leading to stenosis, aneurysm formation, and thrombosis. In cases of vasculitis, the presence of impaired endothelial function may indicate direct activation and injury to endothelial cells, resulting in thromboembolic occurrences such as PE.[1][2] Based on a meta-analysis, patients diagnosed with systemic vasculitis like PAN, exhibit a significantly elevated risk of venous thromboembolism (PAN, pooled RR 3.00, 95 % CI 2.20–4.09).[3] Cardiovascular complications, including coronary arteritis, diffuse interstitial myocarditis, myocardial infarction and spontaneous coronary artery dissection are well-documented in PAN.[4][5] However, the association between PAN and cardiac arrest in the context of PE is less commonly documented due to scarcity of studies exploring this correlation. Here, we discuss the case of a 74-year-old female with PAN who suffered a cardiac arrest complicated by PE. The patient's survival was facilitated by the prompt diagnosis and treatment of cardiac arrest and pulmonary embolism, thereby contributing valuable insights to the existing literature.

II. CASE PRESENTATION

A 74-year-old female, previously diagnosed with PAN, presented to the local hospital with severe breathlessness, discomfort in the chest, and uneasiness. The patient had a known history of PAN for 8 years, for which she is receiving corticosteroid and Azathioprine therapy. On examination, she was tachypneic, tachycardic, and hypotensive. Her pulse oximetry indicated oxygen saturation of 78%. Upon cardiac system examination, tachycardia was noted without any other discernible abnormality. She remained oriented to time, person, and place throughout the examination. The remainder of the physical examination, including neurological and respiratory assessments, revealed no significant abnormalities. The ECG was conducted, revealing no abnormalities apart from sinus tachycardia, as depicted in *"Figure 1"*. A chest X-ray performed showed no remarkable findings. She denied having a personal or family history of chronic diseases such as hypertension (HTN), diabetes mellitus (DM) and coronary artery disease (CAD). Furthermore, there was no history of smoking, alcohol consumption, or use of any other substances. After admission, the patient was promptly put on continuous monitoring and oxygen therapy was administered. Approximately one hour later, she experienced an episode of ventricular fibrillation, which was successfully reversed by a single shock from a defibrillator (360J), resulting in the restoration of normal rhythm. A bedside echocardiogram was performed, which showed EF~60%, dilated RA/RV, severe TR, moderate PAH, RVSP=65 mmHg, concentric LVH, and sclerosed AV/MV. Based on suspicious findings suggestive of pulmonary embolism (PE), a 5000-unit dose of unfractionated heparin was administered. Upon stabilization, the patient was

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transferred to the tertiary care facility for further evaluation and management. On the subsequent day, laboratory investigations revealed elevated D-dimer levels and Troponin I. Other laboratory parameters were within normal limits, as shown in "*Table 1.*" CT Pulmonary Angiography revealed acute pulmonary emboli in bilateral central-segmental pulmonary arterial branches with no evident infarction, as illustrated in *"Figure 2"*. Consequently, thrombolysis was performed using Injection Tenecteplase (30 mg). During hospitalization, the patient was medically managed with Inj Clexane, Tab. Apixaban, anti-ischemic medications, statins, beta blockers, antiplatelets, antacids, and other supportive care. She improved clinically and symptomatically with the above given treatment. The subsequent laboratory results demonstrated improvement without any additional abnormalities. Her further hospital stay was uneventful. She is being discharged with prescribed medications and stable hemodynamics. Additionally, she was counseled on lifestyle modifications, including the importance of medication adherence, regular physical activity, and a heart-healthy diet. Close outpatient follow-up was arranged to monitor her response to therapy and to assess for any potential complications or recurrence of symptoms.

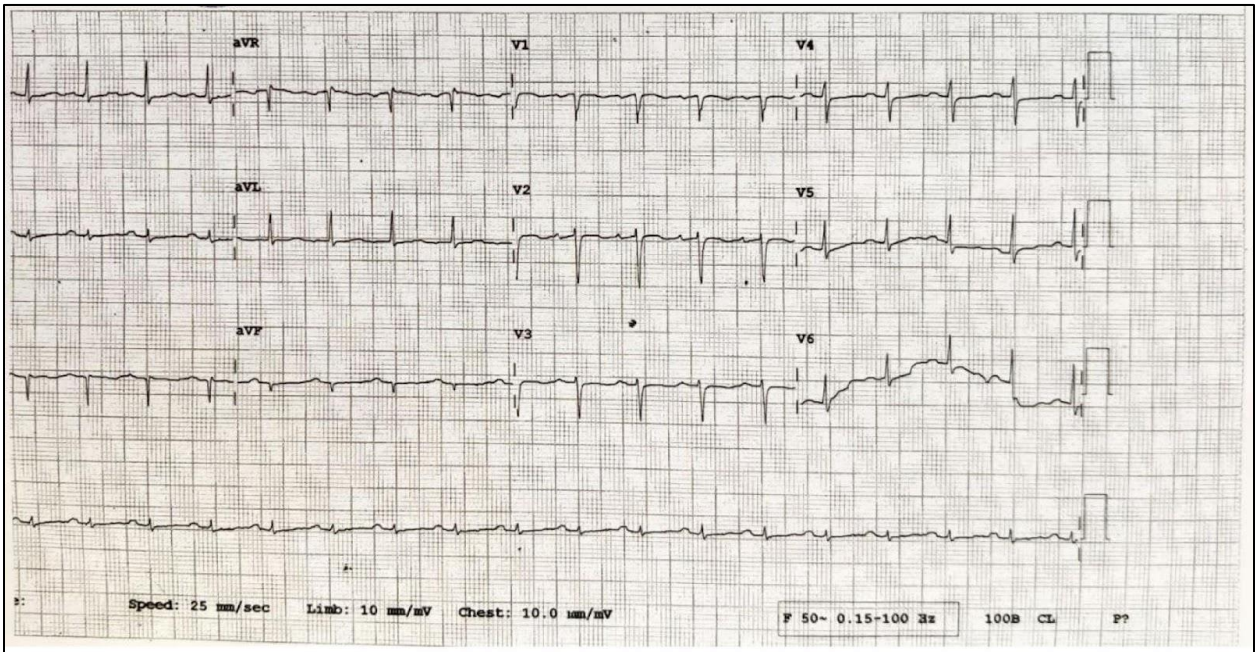

Figure 1. ECG upon admission showing sinus tachycardia

Table 1. Laboratory Findings:

Lab Tests	Patient's Value on day 1	Patient's Value on day 9	Normal Range
D- Dimer	5953 ng/mL	980 ng/mL	0-500 ng/mL
Troponin I	163.7 pg/ml	—	0-14 pg/ml
NT- Pro BNP	2850 pg/ml	<18 pg/ml	≤ 75 years: 0-125 pg/ml, >75 years: 0-450 pg/ml
Sodium	136.3 MMOL/L	141.9 MMOL/L	135-148 MMOL/L
Potassium	3.7 MMOL/L	4.32 MMOL/L	3.5- 5.5 MMOL/L
Creatinine	0.6 mg/dl	0.75 mg/dl	0.6- 1.2 mg/dl

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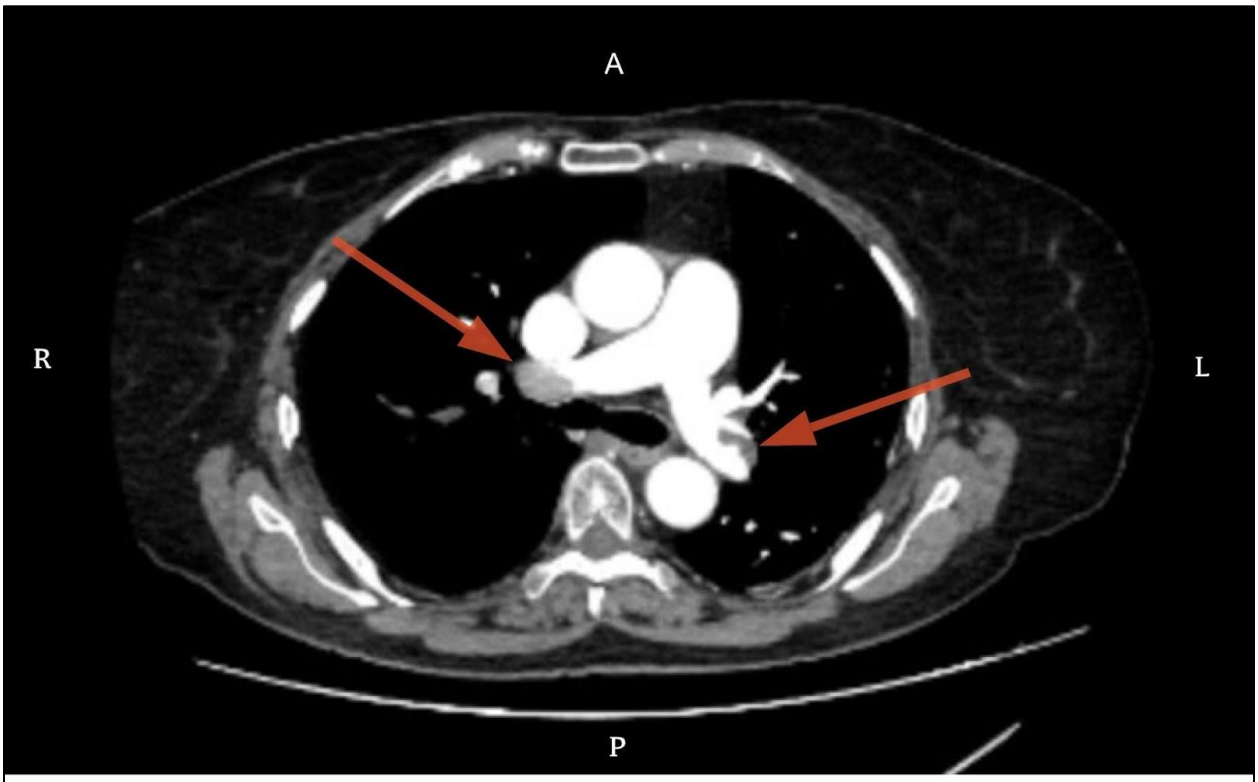

Figure 2. MDCT Pulmonary angiography showing bilateral acute pulmonary emboli in central-segmental pulmonary arterial branches (red arrows) with no evident infarction.

III. DISCUSSION

Polyarteritis nodosa (PAN) presents a complex clinical scenario characterized by systemic inflammation and necrosis of medium-sized arteries, potentially affecting multiple organ systems. While PAN predominantly involves the skin, joints, and peripheral nerves, it can also affect the cardiovascular system, leading to coronary arteritis, myocarditis, and valvular abnormalities.[6] The systemic inflammation and endothelial dysfunction associated with PAN predispose patients to a hypercoagulable state, increasing the risk of thromboembolic events such as PE. PE results from the migration of thrombi, typically originating from deep venous thrombosis, to the pulmonary vasculature, causing obstruction of pulmonary arteries and subsequent hemodynamic compromise. In the context of PAN, the risk of thromboembolic events may be further augmented due to the underlying vasculitic process and associated endothelial injury.[7] Diagnosing pulmonary embolism in patients with PAN can be challenging, as clinical manifestations may overlap with other pulmonary and cardiovascular conditions. Patients with PAN often present with nonspecific symptoms such as dyspnea, chest discomfort, and tachycardia, which may mimic acute coronary syndrome or exacerbation of vasculitis. Within existing literature, cardiac arrest triggers in PAN patients include coronary vasculitis, acute myocardial infarction, coronary artery spasms, and conduction system disruption.[8] However, there is no research exploring cases, where cardiac arrest is simply due to pulmonary embolism without underlying coronary abnormalities in patients with PAN.

In our case, the patient's presentation of severe breathlessness, chest discomfort, and tachycardia warranted a comprehensive evaluation, including bedside echocardiography and CT pulmonary angiography, to confirm the diagnosis of pulmonary embolism. Bedside echocardiography revealed features of acute cor pulmonale, while CT pulmonary angiography confirmed the presence of bilateral pulmonary emboli. The management of cardiac arrest complicated by pulmonary embolism requires a multidisciplinary approach aimed at stabilizing the patient's hemodynamic status, restoring pulmonary perfusion, and preventing recurrent thromboembolic events. In patients with PAN, concurrent management of vasculitis exacerbation is essential to mitigate ongoing systemic inflammation and endothelial injury. In our case, aggressive resuscitative measures, including cardiopulmonary resuscitation (CPR) and administration of thrombolytic therapy, were initiated

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promptly to achieve return of spontaneous circulation (ROSC). The role of immunosuppressive therapy, such as corticosteroids and immunomodulatory agents, in the management of PAN exacerbation cannot be overlooked. However, the timing and selection of immunosuppressive agents must be balanced with the risk of exacerbating thrombotic events and hemorrhagic complications, especially in the acute setting of pulmonary embolism. By detailing the comprehensive evaluation and multidisciplinary management approach utilized in our patient, our report offers practical guidance for clinicians encountering similar cases.

IV. CONCLUSION

The presented case underscores the diagnostic and therapeutic complexities inherent in managing cardiac arrest complicated by pulmonary embolism in patients with a history of PAN. The limited available literature underscores the need for additional studies to deepen our understanding of thromboembolic complications in systemic vasculitis and refine management approaches tailored to this population. Our case report contributes to bridging this gap by providing valuable insights into the diagnostic and therapeutic strategies employed in a real-world clinical setting and provides a basis for further investigation and refinement of management protocols. Further research efforts should focus on elucidating the underlying mechanisms, optimizing diagnostic strategies, and evaluating the effectiveness of therapeutic interventions in patients with PAN-associated pulmonary embolism and cardiac arrest.

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➢ *Competing Interest:*
The authors have no relevant financial or non-financial interests to disclose.

➢ *Informed Consent:*
Complete written informed consent was obtained from the patient for the publication of this study and accompanying images.

➢ *Conflicts of Interest:*
There are no conflicts of interest between authors.

➢ *Ethics Approval:*
Not required because study involves routine clinical care without any experimental interventions. The study was performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

➢ *Author's Contribution:*
The authors confirm contribution to the paper as follows: Conceptualization (Dr. Urja Mehta); Original draft preparation and writing (Dr. Urja Mehta, Dr. Jeet Gajjar); Critical revision for important intellectual content (Dr. Urja Mehta); Final approval of the version to be published (Dr. Urja Mehta, Dr. Jeet Gajjar).

LIST OF ABBREVIATIONS

1. PAN Polyarteritis Nodosa
2. PE Pulmonary Embolism
3. AV/MV Atrial Valve/Mitral Valve
4. DVT Deep Vein Thrombosis
5. EF Ejection Fraction
6. LVH Left Ventricular Hypertrophy
7. PAH Pulmonary Artery Hypertension
8. RA/RV Right Atrium/Right Ventricle
9. RVSP Right Ventricular Systolic Pressure
10. TR Tricuspid Regurgitation

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