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Case Report

Pathology Section

Therapeutic Venesection in Patient of Pulmonary Thromboembolism on Heparin: A Case Report

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ABSTRACT

Therapeutic venesection is a cornerstone in the management of polycythaemia to reduce hyperviscosity and thrombotic risk. However, its application in patients receiving anticoagulation, particularly heparin, is complex due to the potential for bleeding complications. The current case report describes a 40-year-old male who presented with progressive breathlessness, fatigue, and pedal oedema. Laboratory investigations revealed polycythaemia with elevated haemoglobin and haematocrit levels. The patient was diagnosed with acute pulmonary thromboembolism and severe pulmonary artery hypertension. He was initiated on unfractionated heparin therapy. Due to persistent symptoms and hyperviscosity, therapeutic venesection was performed after temporarily withholding heparin. A total volume of 450 mL of blood was removed. The procedure was well tolerated, with stable vital signs, and the patient showed symptomatic improvement following venesection. The present case highlights the therapeutic benefit and safety of venesection in a patient with polycythaemia and pulmonary thromboembolism receiving anticoagulation. Meticulous risk assessment, temporary cessation of anticoagulation at the time of venesection, and strict procedural monitoring were critical in preventing complications. A multidisciplinary approach ensured optimal patient outcomes. The novelty of the current case lies in the successful integration of therapeutic venesection in a high-risk anticoagulated patient, guided by an individualized risk-benefit assessment and coordinated multidisciplinary management to optimise safety and outcomes in complex thrombotic scenarios.

Keywords: Anticoagulation safety, Bleeding complications, Hyperviscosity, Polycythaemia, Pulmonary artery hypertension

CASE REPORT

A 40-year-old male presented with complaints of exertional dyspnoea, fatigue, pedal oedema, cough, fever, and facial puffiness for 10 days. He had a history of chronic Nonsteroidal Anti-Inflammatory Drug (NSAID) use for lower back pain for six weeks and alcohol consumption for the past 10 years. His father had a history of coronary artery disease. On examination, vital signs were stable. Cardiovascular examination revealed a loud pulmonary component of the second heart sound and the presence of a third heart sound. Respiratory examination showed bilateral crepitations. Laboratory investigations demonstrated haemoglobin of 18 g/dL, packed cell volume of 55.4%, and platelet count of 2.55 lakh cells/µL.

On admission, the patient was in a volume-overloaded state with reduced urine output and elevated serum creatinine (1.4 mg/dL). The chronic use of NSAIDs supported the diagnosis of acute kidney injury. The procoagulant state was attributed to polycythaemia. Peripheral blood smear revealed erythrocytosis with neutrophilic leukocytosis. Electrocardiography showed T-wave inversions, and the cardiac panel was positive for elevated brain natriuretic peptide (2,725 pg/mL). Echocardiography revealed severe pulmonary artery hypertension with a pressure of 75 mm Hg. Computed tomography pulmonary angiography demonstrated bilateral pulmonary artery thrombi with dilation of the main pulmonary trunk, consistent with acute pulmonary embolism.

Based on these findings, a final diagnosis of acute pulmonary embolism, severe pulmonary artery hypertension, acute kidney injury, and polycythaemia under evaluation was made.

The patient was started on intravenous unfractionated heparin with a 5,000 IU bolus followed by an infusion at 18 units/kg/hour. In view of elevated haemoglobin and haematocrit levels, therapeutic venesection was advised. Janus Kinase 2 (JAK2) mutation analysis was suggested but not performed. Therapeutic venesection was carried out on the seventh day of admission. The patient weighed 70 kg, and the calculated volume of blood removal based on body weight was approximately 560 mL (8 mL/kg). A standard volume of 450 mL was removed. Prior to the procedure, haemoglobin was 18 g/dL and packed cell volume was 55.2%. Heparin was temporarily discontinued before venesection. Pre and post procedure vital signs remained stable [Table/Fig-1].

Parameters	17/1/25	18/1/25	19/1/25	20/1/25	21/1/25	25/1/25
Hb (g/dL)	17.4	18.5	17.8	17.8	18	17.8
PCV (%)	52.7	56.3	55.1	54.7	55.2	56.8

[Table/Fig-1]: Haemoglobin and haematocrit levels pre and post venesection.
Hb: Haemoglobin; PCV: Packed cell volume; Reference Ranges: Haemoglobin: 13.5-17.5 g/dL for males, 12.0-16.0 g/dL for females, PCV: 41%-53% for males, 36%-46% for females.

There was a decreasing trend in haemoglobin and haematocrit levels following venesection. The patient experienced significant symptomatic relief related to acute pulmonary embolism and severe pulmonary artery hypertension. At discharge, haemoglobin was 17.8 g/dL.

The patient was discharged on tablet apixaban 10 mg, tablet sildenafil 10 mg, and tablet torsemide (Dytor) 10 mg. Follow-up details were unavailable as the patient did not return for subsequent visits.

DISCUSSION

Polycythaemia, also known as erythrocytosis, refers to an elevated red blood cell mass in the body. The condition can be suspected in individuals with increased haematocrit levels, typically exceeding 49% in men and 48% in women, or with elevated haemoglobin concentrations, defined as more than 16.5 g/dL in men and 16.0 g/

dL in women. An increased red blood cell count may also support the diagnosis [1].

Secondary polycythaemia develops over time and is not inherited. It arises from malignancies involving haematopoietic stem cells or from other systemic disorders. One of the most common causes of secondary polycythaemia is tissue hypoxia, which stimulates erythropoiesis and leads to increased blood viscosity, thereby raising the risk of complications such as thrombosis and ischaemia [2].

Acute pulmonary embolism is a critical and potentially fatal condition caused by a blood clot obstructing one or more pulmonary arteries. It remains a major global contributor to morbidity and mortality. Timely initiation of anticoagulation therapy is crucial for effective management. Traditionally, treatment involves low-molecular-weight heparin or unfractionated heparin, followed by oral vitamin K antagonists for long-term anticoagulation [3].

Therapeutic phlebotomy is commonly used as a first-line intervention, with one of its primary goals being the reduction of thrombotic risk. In patients with polycythaemia, venesection has been associated with a lower incidence of thrombotic events and reduced cardiovascular-related mortality [2].

Tefferi A and Barbui T, described the risk factors for thrombosis in polycythaemia and outlined current therapeutic goals for thrombosis prevention, which include periodic phlebotomy to maintain a haematocrit target of <45%, combined with aspirin therapy. Additionally, systemic anticoagulation is recommended in patients with a history of venous thrombosis [1].

Therapeutic venesection in patients at increased risk of thrombosis requires careful risk assessment and planning. In general, the procedure should be performed with caution in patients receiving heparin therapy to minimise the risk of bleeding and haemodynamic instability. Performing venesection in patients with pulmonary thromboembolism on heparin requires particular attention, as bleeding is the most common adverse effect of heparin and occurs in approximately 5-10% of patients [4].

Ensuring patient safety through adequate monitoring protocols before, during, and after the procedure is essential to prevent adverse outcomes. Patient-specific factors such as age, sex, weight, current medications, and medical co-morbidities should be evaluated prior to venesection [5]. Laboratory parameters and vital signs should be carefully reviewed. Procedural safety measures include maintaining a sterile environment to minimise complications and ensure patient comfort. Emergency equipment and medications should be readily available. The procedure should be performed by trained personnel skilled in phlebotomy. Proper arm inspection, selection of an appropriately sized needle, use of sterile atraumatic technique, and avoidance of excessive venous manipulation are recommended.

Key considerations when performing therapeutic venesection in a patient receiving heparin include monitoring coagulation parameters, particularly Activated Partial Thromboplastin Time (aPTT), to ensure it remains within the therapeutic range of approximately 1.5-2.5 times the baseline value [6]. If prolonged, venesection should be approached with caution and appropriately timed. Preprocedure clinical assessment should include evaluation for signs of active bleeding or a history of easy bruising [6]. Monitoring platelet count is essential to exclude heparin-induced thrombocytopaenia, which increases the risk of thrombotic complications [7].

Procedural modifications, such as employing atraumatic techniques and applying prolonged pressure at the venesection site until haemostasis is achieved, are important to minimise bleeding [8]. Optimal fluid management with adequate hydration and haemodynamic support should be ensured [8]. Post venesection monitoring should include vital signs, inspection of the venesection site for bleeding or haematoma formation, and assessment for signs of hypovolaemia or vasovagal reactions.

Therapeutic venesection is considered a safe and effective symptomatic treatment in patients with secondary polycythaemia. Most patients report either complete resolution of clinical symptoms or significant laboratory improvement [9]. In the present case report, clinical symptoms improved more markedly than laboratory parameters. Symptoms related to acute pulmonary embolism and severe pulmonary artery hypertension-including shortness of breath, fatigue, and manifestations of hyperviscosity-showed significant improvement, thereby reducing thrombotic risk and cardiovascular complications.

Recent studies suggest that cytoreductive agents, including newer interferons, demonstrate improved tolerability and efficacy in symptom control [10]. Ropeginterferon alfa-2b has been shown to reduce moderate-to-severe symptoms in low-risk polycythaemia vera compared to standard therapy, indicating better tolerability and clinical benefit [11].

A multidisciplinary team approach involving a transfusion medicine specialist, physician, and haematologist is essential for effective management. Patient education should address the risks of venesection while on anticoagulation, post-procedure care, avoidance of pressure or trauma at the venesection site, and prompt reporting of symptoms such as dizziness, excessive swelling, or bruising to minimise complications.

CONCLUSION(S)

The present case demonstrates that therapeutic venesection in a patient with pulmonary thromboembolism receiving heparin requires careful risk assessment, particularly regarding bleeding and haemodynamic instability. The procedure was safely performed following temporary cessation of heparin with close monitoring of vital signs. Preprocedural evaluation of coagulation status, adequate hydration, and stepwise reintroduction of anticoagulation were key factors in minimising complications. When necessary, alternative anticoagulant strategies and reversal agents may be considered. A multidisciplinary approach remains essential to optimise patient outcomes and ensure procedural safety.

REFERENCES

[1] Tefferi A, Barbui T. Polycythemia vera: 2024 update on diagnosis, risk stratification, and management. Am J Hematol. 2023;98(9):1465-87.
[2] Haider MZ, Anwer F. Secondary polycythemia. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan. Available from: https://www.ncbi.nlm.nih.gov/books/NBK562233/
[3] Vyas V, Sankari A, Goyal A. Acute pulmonary embolism. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Dec 11. Available from: https://www.ncbi.nlm.nih.gov/books/NBK560551/
[4] Raskob GE, Carter CJ, Hull RD. Heparin therapy for venous thrombosis and pulmonary embolism. Blood Rev. 1988;2(4):251-58.
[5] Lim HY, Ho WK. Performing therapeutic venesection in a doctor's surgery. Aust Fam Physician. 2017;46(3):132-138.
[6] Patel P, Huang D. Heparin. [Updated 2025 Aug 9]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK538247/
[7] Dager WE, Dougherty JA, Nguyen PH, Militello MA, Smythe MA. Heparin induced thrombocytopenia: treatment options and special considerations. Pharmacotherapy. 2007;27(4):564-87.
[8] Piran S, Schulman S. Treatment of bleeding complications in patients on anticoagulant therapy. Blood. 2019;133(5):425-35.
[9] Ahmed M, Khalid MF, Abbas A, Shaikh HG, Ammad ud din M, Mushtaq MU, et al. Phlebotomy in secondary polycythemia: does it improve clinical symptoms? A systematic review. Blood. 2022;140(Suppl 1):11050-51.

[10] Ahmed T, Hobbs GS, Sirignano DS, Foster J, Sosa A, Chien HL, et al. Burden of illness in polycythemia vera: a retrospective medical chart audit study. J Clin Oncol. 2024;42(16_suppl):e18530.

[11] Barbui T, Vannucchi AM, De Stefano V, Carobbio A, Ghirardi A, Carioli G, et al. Ropeginterferon versus standard therapy for low-risk patients with polycythemia vera. NEJM Evidence. 2023;2(6):EVIDoa2200335.

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