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CASE REPORT

Cardiac hypertrophy in polycythemia vera: A case report and review of literature

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Abstract

BACKGROUND

The combination of polycythemia vera (PV) with pathological cardiac hypertrophy is uncommon. In this study, we describe a case of PV accompanied by pathological cardiac hypertrophy. It is hypothesized that the pronounced cardiac hypertrophy in this patient has a strong connection with PV.

CASE SUMMARY

In 2021, a 34-year-old Chinese man experienced chest constriction, shortness of breath, and palpitations during vigorous activity. Each episode lasted several minutes and resolved spontaneously following cessation of vigorous activity. He occasionally experienced syncope and vertigo without a headache. He underwent cardiac magnetic resonance imaging and was diagnosed with "hypertrophic cardiomyopathy (HCM)". He was discharged after receiving symptomatic treatment, which resulted in an improvement. He presented to our department with chest constriction, shortness of breath, and respiratory distress for one month while climbing to the second floor in 2023. His blood pressure was 180/100 mmHg at the time of admittance, and he was receiving antihypertensive treatment. He had a history of PV for 2 years without treatment. Symptomatic treatment was implemented concurrently with the administration of hydroxyurea upon admission. Good blood pressure control was observed during the long-term follow-up, and echocardiography did not reveal any progression of myocardial hypertrophy.

CONCLUSION
Clinicians managing PV patients should remain highly vigilant regarding the risks of thrombosis and cardiovascular complications, particularly in those with refractory hypertension.

Key Words: Polycythemia vera; Cardiomyopathy hypertrophic; Hypertension; Thrombosis; Case report

Core Tip: Clinicians should be highly vigilant regarding the risk of thrombosis and cardiovascular complications when managing patients with polycythemia vera (PV). In young patients with hypertension, who have excluded common secondary causes and have difficult-to-control blood pressure, there should be an alert for PV. The coexistence of PV and myocardial hypertrophy is rarely reported. The myocardial hypertrophy observed in this case, which is difficult to explain for other reasons, proposes a new hypothesis that PV may be a potential trigger. Cytoreductive therapy may be an important factor in improving the patient's myocardial hypertrophy.

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INTRODUCTION

Polycythemia vera (PV) is a rare hematologic disorder that is classified as chronic myeloproliferative neoplasms (MPNs). The disease is distinguished by the acquired clonal proliferation of red blood cells, which leads to a significantly elevated peripheral blood hematocrit (HCT) and increased blood viscosity. It is also characterized by splenomegaly, elevated white blood cell and platelet counts, and other potential symptoms. Furthermore, complications such as thrombosis and hemorrhage may develop during the disease[1,2]. The relationship between the risk of cardiovascular disease and the underlying mechanisms of MPNs has been explained by a plethora of clinical and mechanistic studies in recent years[3]. Although numerous case reports have investigated the relationship between PV and thrombotic diseases, there are relatively few reports that have examined the association between PV and myocardial hypertrophy.

This study presents a case of PV complicated with cardiac hypertrophy. It offers a comprehensive description of the patient's experience with antihypertensive medication and gives a thorough overview of their long-term follow-up. An in-depth literature review was also conducted to investigate the possible link between PV and the risk of cardiovascular disease, focusing on clinical case studies. The objective is to provide new perspectives and insights for assessing cardiovascular risk and analyzing prognosis in patients with MPNs. These results not only enhance our comprehension of the correlation between PV and cardiovascular disease but also have significant implications for future clinical management guidelines.

CASE PRESENTATION

Chief complaints

A 34-year-old man presented with a two-year history of chest tightness and dyspnea, which worsened over the past month leading to hospital admission.

History of present illness

Two years ago, during vigorous activity, he experienced episodes of chest tightness, dyspnea, and palpitations lasting several minutes each time, resolving spontaneously upon cessation of activity. He occasionally felt dizzy but denied headaches, blurred vision, or syncope. He underwent echocardiography and cardiac magnetic resonance imaging (MRI) at our hospital and was diagnosed with HCM, for which symptomatic treatment was initiated. One month ago, he developed severe chest tightness and dyspnea with exertion, such as climbing to the second floor, accompanied by respiratory distress but without precordial pain.

History of past illness

The patient had a 10-year history of hypertension, with peak blood pressure reaching 200/150 mmHg. Current treatment includes sustained-release calcium channel blocker (nifedipine 30 mg QD) and beta-blocker (metoprolol 47.5 mg QD), maintaining blood pressure at 170-180/120-130 mmHg. Secondary hypertension screening conducted three years ago showed minimal suspicion of secondary hypertension. The patient had a stroke three years ago and has had erythrocytosis for the past three years. A bone marrow examination suggested PV, but no further investigation or treatment was

pursued. He had no history of smoking, alcohol abuse, or type 2 diabetes mellitus.

Personal and family history

His father and uncle died from a brain hemorrhage. His brother's routine blood examination showed erythrocytosis, and echocardiography indicated interventricular septum and left ventricular posterior wall thickness of 14-15 mm, suggesting myocardial hypertrophy. A bone marrow aspiration has not yet been performed.

Physical examination

The patient was alert and oriented, with no other abnormal findings observed.

Laboratory examinations

Table 1 shows the blood test results, including hemoglobin and erythrocyte count, *etc*. The results of blood tests at the time of successive hospitalizations are also shown.

Imaging examinations

Table 2 shows the findings of cardiac color Doppler ultrasound for all previous hospitalizations.

FINAL DIAGNOSIS

Based on his symptoms, medical history, blood tests, and imaging examination, the patient was diagnosed with HCM, PV, stage 3 hypertension (extremely high risk), and lacunar cerebral infarction.

TREATMENT

Upon admission, diltiazem hydrochloride tablets (30 mg BID) and sacubitril-valsartan (100 mg QD) were added to the original antihypertensive drugs nifedipine controlled-release tablets (30 mg QD) and metoprolol succinate (47.5 mg QD). During hospitalization, following a consultation with the hematology department, hydroxyurea (0.5 g BID) was added to reduce erythrocyte levels, and aspirin (100 mg QD) was prescribed for thromboprophylaxis. The patient was discharged when his symptoms of chest tightness and shortness of breath improved after pharmacological treatment. He continued the same medication regimen, and no modifications were made to the treatment plan during the follow-up period.

OUTCOME AND FOLLOW-UP

One month after discharge, the patient adhered to the prescribed treatment regimen. The patient's hemoglobin levels returned to normal, blood pressure was controlled at 140/90 mmHg, and no further progression of myocardial hypertrophy was observed.

DISCUSSION

PV is a MPN that is characterized by abnormal proliferation of the red cell lineage in the bone marrow, resulting in an abnormal expansion of red blood cells in the bloodstream. The blood is thickened as a result of this abnormal increase in red blood cells, which slows blood flow and increases the risk of thrombosis. In PV patients, thrombosis is one of the most prevalent complications and causes of mortality[1]. PV may also result in complications such as leukemia, bone marrow fibrosis, and hemorrhage events. Currently, it is believed that the cardiovascular mortality risk in PV patients can be significantly reduced by maintaining HCT levels below 45% through intensified therapy[4]. This further confirms the correlation between PV and increased cardiovascular disease risk. Table 3 is a compilation of case reports from PubMed documenting instances of PV occurring with concomitant cardiovascular diseases. According to the literature, cardiac complications—including myocardial infarction, heart failure, and structural changes in the heart—are particularly important in this comprehensive analysis of PV cases combined with cardiovascular diseases. Coronary artery thrombosis is frequently observed in individuals with PV and is characterized by the formation of thrombi with a honeycomb appearance[5,6]. This may be due to the process of thrombus recanalization following blockage. It is also possible for some PV patients to have recanalization without the need for coronary artery intervention. This can occur by conservative therapy after the occurrence of coronary artery thrombosis. This suggests that symptoms may not be severe in certain PV patients after experiencing coronary thrombosis. Hence, it is essential to be vigilant of potential cardiac problems in PV patients who experience nonspecific symptoms such as chest tightness, dyspnea, or fatigue. In such cases, it may be important to consider experimental dual antiplatelet or anticoagulant therapy[7]. In addition to cardiovascular embolism, individuals with PV are also at risk of cerebrovascular embolism, which can potentially result in a stroke[8]. In this instance, the patient initially manifested symptoms of stroke upon admission.

Table 1 Blood test results of the patient at successive hospitalizations

Parameters	March 2021	January 2022	August 2022	October 2023	December 2023	Reference range
Hemoglobin	200 g/L	200 g/L	178 g/L	181 g/L	155 g/L	120-160 g/L
Hematocrit	0.595 L/L	0.569 L/L	0.516 L/L	0.524 L/L	0.449 L/L	0.4-0.5 L/L
Erythrocyte count	6.79×10^{12}/L	6.63×10^{12}/L	6.01×10^{12}/L	5.99×10^{12}/L	4.63×10^{12}/L	4×10^{12}-5.5×10^{12}/L
White blood cells	9.22×10^{9}/L	7.49×10^{9}/L	8.14×10^{9}/L	9.16×10^{9}/L	8.7×10^{9}/L	4×10^{9}-10×10^{9}/L
Platelet count	197×10^{9}/L	227×10^{9}/L	209×10^{9}/L	223×10^{9}/L	257×10^{9}/L	100×10^{9}-300×10^{9}/L

Table 2 Results of cardiac color Doppler ultrasound

Parameters	March 2021	January 2022	August 2022	October 2023
IVS (mm)	22	20.6	20.2	22
LVPW (mm)	21.9	28.9	18	16.4
EF%	75.4	75.8	67.2	68.4

IVS: Interventricular; LVPW: Left ventricular posterior wall; EF: Ejection fraction.

The underlying causes of thrombosis in individuals with PV are not fully understood; however, they may be related to higher HCT levels, increased blood viscosity, excessive platelet activation, and leukocytosis[9]. These factors collectively contribute to a hypercoagulable state in patients. Hence, considering the study findings described above, it is crucial to prioritize the prevention of thrombosis in the treatment guidelines for PV. This is particularly important for individuals with risk factors such as a history of thrombosis, age > 60 years, hypertension, hyperlipidemia, and leukocytosis. Regular phlebotomy and aspirin therapy (81 mg/day) are advised regardless of risk stratification[1]. Consideration may be given to twice-daily aspirin dosing for patients who are resistant to once-daily aspirin or at a higher risk of arterial thrombosis [2]. The potential of cytoreductive therapy to reduce thrombotic risk remains controversial. Pegylated interferon is an efficacious treatment for PV patients who are resistant to or intolerant of hydroxyurea, while hydroxyurea is the first-line cytoreductive therapy[10]. Nevertheless, there is currently no controlled trial that has confirmed the superiority of peg-IFN over hydroxyurea. A multicenter randomized controlled study has shown that cytoreductive therapy significantly decreases the risk of thrombotic recurrence. Antiplatelet agents and oral anticoagulants have also shown some efficacy in recurrence prevention[11]. Conversely, a large prospective multicenter study indicated a significant association between antiplatelet therapy and reduced risk of cardiovascular events, whereas cytoreductive therapy did not show such an association[12]. Despite the current lack of definitive conclusions regarding cytoreductive therapy and thrombotic risk, it may be considered an adjunctive therapy to antiplatelet treatment in high-risk thrombotic populations[13]. In patients with venous thrombosis, the addition of warfarin can further reduce recurrence rates[14]. However, maintaining a balance between thrombotic and bleeding risks is crucial during antithrombotic therapy. While the overall incidence rate of major hemorrhage in PV patients is 0.9%, it increases to 2.8% patient-years with combined antiplatelet and vitamin K antagonist therapy. Therefore, continuous monitoring of bleeding and thrombotic events in patients remains paramount.

The present case involves a patient with a history of previous cerebrovascular embolism who was admitted with symptoms of chest tightness and dyspnea, along with a positive Brain Natriuretic Peptide, indicating potential microcirculatory disorders alongside concerns about thrombotic recurrence. Therefore, the primary focus of treatment for this patient is aimed at preventing further thrombotic events. Conversely, the current therapy of aspirin and hydroxyurea has shown beneficial therapeutic effects in managing this patient's condition.

Moreover, there is scarce literature on the association of PV with cardiac structural changes. Hence, this case report on PV combined with myocardial hypertrophy holds particular clinical significance, offering a new perspective on the diversity and complexity of cardiac complications in PV.

Table 4 outlines the diagnostic criteria that distinguish common causes of myocardial hypertrophy, including hypertension and HCM. Hypertension-induced myocardial hypertrophy typically presents with symmetric characteristics, evidenced by echocardiography showing uniformly hypoechoic thickened myocardium[15], often with a wall thickness ≤ 15 mm. In contrast, HCM is characterized by ventricular wall thickening, particularly asymmetric hy-pertrophy of the interventricular septum. This condition often results in a reduced left ventricular cavity, and basal septal hypertrophy can lead to left ventricular outflow tract obstruction[16]. Fabry disease frequently manifests with concentric hypertrophy[17], and its diagnosis mostly depends on genetic testing. Amyloidosis leads to symmetrical myocardial hypertrophy[18], usually without evidence of ventricular high voltage on electrocardiography. It is identified with radionuclide imaging or cardiac biopsies. Athletes often experience physiological hypertrophy, which is marked by an increase in the thickness of the left ventricular wall.

In the present case, cardiac MRI of the patient revealed symmetric hypertrophy of the cardiac walls: Septum 17.3 mm, anterior wall 17.9 mm, lateral wall 21.0 mm, and inferior wall 17.7 mm. Cardiac ultrasound indicated extreme symmetric hypertrophy of the ventricular walls, which does not completely align with the etiological characteristics outlined in

Table 3 Case reports of polycythemia vera complicated with cardiac disease

Ref.	Biographical information	Diagnosis	Therapy	Prognosis	Pivot
Bahbahani *et al*[9]	Egyptian woman aged 37 years	Acute myocardial infarction, PV	Thrombolysis, hydroxyurea 15 mg/kg, aspirin 81 mg	After 4 weeks, myocardial perfusion imaging of the patient revealed no evidence of myocardial ischemia. Coronary CT angiography showed normal findings	Young individuals without atherosclerosis and its associated risk factors may experience cardiovascular thrombotic events due to PV
Zaman *et al*[7]	61-year-old female	Heart failure, microcirculatory disorder, PV	Normally treated with bloodletting, aspirin, and clopidogrel after diagnosis	During follow-up, the patient did not experience any new episodes of chest pain	PV can lead to microembolism in the cardiac microcirculation, resulting in impaired cardiac function
Duran Luciano and Sabella-Jiménez[31]	52-year-old Hispanic male	Acute myocardial infarction, *JAK2* negative PV	Antiplatelet, anticoagulation, and PCI therapy	Follow-up revealed improvement in cardiac function compared to previous assessments	*JAK2*-negative PV can also lead to cardiovascular thrombotic events
Inami *et al*[32]	64-year-old male	Acute myocardial infarction, recurrence of myocardial infarction after PCI, PV	PCI treatment, phlebotomy, and hydroxyurea for PV	No complications occurred	Patients with PV have a high risk of intrastent thrombosis following PCI
D'Onofrio *et al*[33]	86-year-old female	Severe stenotic aortic valve, pulmonary edema, post aortic valve replacement, respiratory circulatory failure	Aortic valve replacement, ECMO, CPR	The patient died	PV accompanied by severe thrombocytosis precluded antiplatelet and anticoagulant therapy, resulting in death from cerebral hemorrhage. Autopsy revealed extensive white thrombi formation in both the aortic valve and ventricles
Butt and Latif[34]	49-year-old male	Dilated cardiomyopathy, New York Classification III	Aspirin 100 mg, ramipril and bisoprolol in an increasing dose titration regimen. Furosemide 40 mg	During follow-up, the ejection fraction improved from 18% to 42%	Microvascular myocyte necrosis is considered the sole plausible pathophysiology of the cardiomyopathy
Haroun *et al*[35]	71-year-old Ethiopian man	PV, pericardial effusion, post-PV myelofibrosis	Discontinuation of hydroxyurea, pericardiocentesis	At 8 weeks following the initial consultation, during outpatient follow-up, complete blood cell counts revealed a leukocyte count of 13.6 × 10^9 cells/L, hemoglobin level of 9.9 g/dL, and platelet count of 556000/L	PV progressed to bone marrow fibrosis, resulting in extramedullary hematopoiesis and the formation of pericardial effusion

CT: Computed tomography; PV: Polycythemia vera; PCI: Percutaneous coronary intervention; *JAK2*: Janus kinase 2; ECMO: Extracorporeal membrane oxygenation; CPR: Cardiopulmonary resuscitation.

Table 4 Characteristics of different cardiac hypertrophy diseases

Name of disease	Typical features	Means of identification
HCM	Asymmetric septal hypertrophy, often accompanied by left ventricular outflow tract obstruction	Genetic testing and cardiac MRI
Hypertensive heart disease	Symmetrical myocardial hypertrophy, generally, ventricular wall thickness is ≤ 15 mm	History of hypertension for many years
Fabry disease	Concentric hypertrophy	α-Galactosidase A activity assay, *GLA* gene test
Myocardial amyloidosis	Symmetrical myocardial hypertrophy, characterized on electrocardiography by low voltage or normal voltage	Radionuclide imaging, cardiac biopsy line histology and amyloid staining
Physiological hypertrophy	In athletes, the unique condition of mild, uniform left ventricular wall thickening may be accompanied by an increase in left ventricular cavity diameter	Cardiopulmonary exercise test

HCM: Hypertrophic cardiomyopathy; MRI: Magnetic resonance imaging.

Table 4. Clinical studies have identified various cardiac issues in PV patients, including chamber enlargement, interventricular septal hypertrophy, pulmonary hypertension, left ventricular systolic and diastolic dysfunction, and impaired relaxation of the valve fibrous ring[4]. Therefore, it is speculated that PV may play a role in the process of myocardial hypertrophy. The precise mechanisms leading to such profound myocardial hypertrophy in patients with PV remain unclear and necessitate further research to elucidate potential pathways[19,20]. In this case, the duration of hypertension in the patient was longer than the time at which myocardial hypertrophy was detected, and the patient's sibling also exhibited signs of myocardial hypertrophy. Consequently, it is challenging to attribute the myocardial hypertrophy solely to factors such as hypertension, genetic predisposition, HCM, or PV. However, we believe that PV plays a significant role within the context of these multifactorial influences. Cardiac color Doppler imaging is necessary in patients with PV, as cardiac complications are extremely severe. The prognosis of patients is significantly improved by the timely intervention that is made possible by the early detection of indications of left ventricular systolic dysfunction through cardiac color Doppler. The utilization of tissue Doppler imaging (TDI) technology provides a new approach for the earlier diagnosis of myocardial dysfunction in patients with PV. Research suggests that the longitudinal strain of the left ventricular myocardium and the decreased TDI parameters of the fibrous ring around the atrioventricular valve can be used as early diagnostic criteria for myocardial dysfunction in PV patients[21].

Several mechanisms for myocardial hypertrophy in patients with PV are currently proposed. Firstly, the increased red blood cell mass in PV patients increases blood viscosity, which in turn increases vascular resistance[22], particularly in the microcirculation and arterioles, increasing left ventricular afterload. This viscosity increase can occasionally lead to microvascular flow obstruction[23], which can impact the delivery of oxygen and nutrients, as well as the removal of waste metabolites, resulting in relative myocardial hypoxia. To accommodate the increased afterload and oxygen demand, myocardial cells undergo hypertrophy to improve contractility and preserve the equilibrium between oxygen supply and demand. Secondly, erythropoietin (EPO) may possess cardioprotective properties[24]. Thus, cardiac complications may result from decreased EPO levels in PV patients. Thirdly, Janus kinase 2 (*JAK2*) gene mutations, as one of the primary pathogenic mechanisms of PV, have been associated with myocardial hypertrophy in animal models[25]. The *JAK2 V617F* mutation can lead to cardiac disease through inflammatory mechanisms, and can cause myocardial hypertrophy[26]. Mechanistically, the mutation increases JAK-STAT pathway expression, which subsequently enhances the expression and activation of the Absent in melanoma 2 (AIM2) inflammasome. The activated AIM2 inflammasome then stimulates the production of the inflammatory cytokine interleukin-1β, promoting apoptosis and contributing to the pathophysiological processes of cardiovascular diseases[27].

After conducting investigations for secondary hypertension in our patient and ruling out other potential causes, refractory hypertension in this young patient appears closely linked to PV. The patient had a long-standing history of hypertension, with inadequate blood pressure control on nifedipine and metoprolol. Consequently, upon admission, diltiazem and sacubitril/valsartan were added to the treatment regimen. Following the addition of hydroxyurea and aspirin to the treatment plan, blood pressure control significantly improved. During follow-up, there was no observed progression of myocardial hypertrophy or further issues with blood pressure control. The initial antihypertensive regimen was insufficient, but after adjusting the medication and incorporating hydroxyurea, blood pressure improved, suggesting that the combination of these medications may have enhanced the patient's prognosis. Additionally, there was a decrease in the patient's erythrocyte levels. Although hydroxyurea appears to play a crucial role, we cannot disregard the potential contributions of sacubitril/valsartan and diltiazem in suppressing erythrocyte production. This challenging case of resistant hypertension underscores the difficulties in managing hypertension in patients with PV, indicating that antihypertensive strategies in this population can be particularly complex. For cardiologists, it is essential to monitor hemoglobin levels in young patients with hypertension. Adhering to the World Health Organization (WHO) criteria for the diagnosis of PV[28], early intervention is recommended for patients meeting diagnostic criteria to prevent adverse outcomes. The WHO diagnostic criteria for PV are summarized in Table 5. Comprehensive diagnostic evaluations are advised to confirm the diagnosis and ensure appropriate management of patients. With regard to hematologists, it is crucial that they assess cardiovascular risk in PV patients during treatment and to implement appropriate interventions to prevent adverse events.

In PV patients with concurrent hypertension, antihypertensive medications that are currently available are effective in reducing blood pressure. Among them, ACE inhibitors may be the best option since they reduce blood pressure while also limiting abnormal red blood cell production, thereby minimizing the requirement for cytoreductive treatment[29].

Management of PV extends beyond the responsibility of hematologists and requires a multidisciplinary approach. PV is associated with a high risk of cardiovascular events, necessitating the involvement of cardiologists. The primary risk in PV is thrombosis; thus, vascular surgeons are crucial in managing patients with deep vein thrombosis. Additionally, given the potential for PV to progress to acute leukemia, oncologists must provide ongoing monitoring. A multidisciplinary management strategy that integrates the expertise of hematologists, cardiologists, vascular surgeons, oncologists, and other specialists can facilitate the development of a comprehensive treatment plan, addressing all aspects of the disease to improve patient outcomes and quality of life.

Following analysis of the patient's historical data on left ventricular posterior wall thickness and hemoglobin levels (Figure 1), a correlation between these variables was identified. Decreasing hemoglobin levels corresponded to a reduction in the degree of myocardial hypertrophy. Furthermore, effective hypertension control correlated with decreased myocardial hypertrophy[30]. The reversibility of myocardial hypertrophy in this patient further supports the diagnosis of secondary myocardial hypertrophy.

Future research should focus on further experimental studies and data analysis to elucidate the intrinsic mechanisms linking cardiovascular disease risk with PV. Investigating the role of tumor-associated genes in ventricular remodeling could provide a theoretical foundation for cardiovascular risk assessment and prognosis in PV patients, as well as uncover new mechanisms underlying cardiovascular disease.

Table 5 World Health Organization criteria for polycythemia vera

Major criteria	Minor criterion
Hemoglobin 16.5 g/dL in men Hemoglobin 16.0 g/dL in women, or Hematocrit 49% in men Hematocrit 48% in women, or increased RCM[1]	Subnormal serum erythropoietin level
BM biopsy showing hypercellularity for age with trilineage growth (panmyelosis) including prominent erythroid, granulocytic, and megakaryocytic proliferation with pleomorphic, mature megakaryocytes (differences in size)	
Presence of *JAK2 V617F* or *JAK2* exon 12 mutation	

[1]Diagnosis of polycythemia vera requires meeting either all 3 major criteria, or the first 2 major criteria and the minor criterion.
RCM: Red cell mass; *JAK2*: Janus kinase 2.

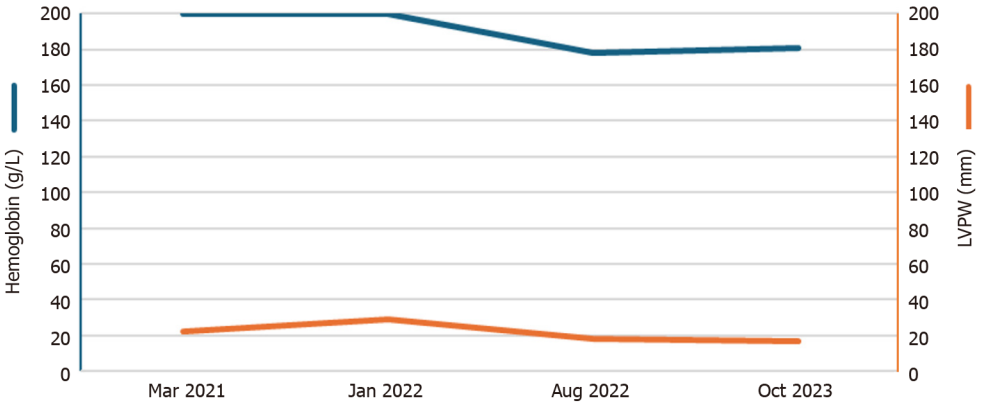

Figure 1 Line graphs of the posterior wall of the left ventricle and hemoglobin data for all previous hospitalizations of this patient. At higher levels of hemoglobin, the thickness of the posterior wall of the left ventricle increases. Subsequently, the thickness of the posterior wall of the left ventricle decreases as the hemoglobin level decreases. LVPW: Left ventricular posterior wall.

Future studies could be conducted in the following specific areas to validate the hypotheses proposed in this paper and to explore the relationship between PV and cardiac complications: (1) Prospective cohort studies: Regularly measure blood viscosity in PV patients and correlate these measurements with cardiac imaging results (*e.g.*, echocardiography, MRI); (2) Experimental research: Utilize tissue oxygenation monitoring techniques (*e.g.*, near-infrared spectroscopy) to assess myocardial oxygen supply in PV patients. Combine these assessments with measurements of cardiac biomarkers (*e.g.*, lactate dehydrogenase, natriuretic peptides) to analyze the specific effects of erythrocytosis on myocardial metabolism and hypertrophy; (3) Comparative prospective studies: Evaluate cardiac function and myocardial hypertrophy in PV patients with varying levels of EPO. Consider interventional studies to assess whether EPO replacement therapy offers protective effects against cardiac complications; and (4) Clinical observational studies: Given that animal models have demonstrated myocardial hypertrophy in *JAK2*-mutant mice, perform clinical observational studies to analyze the relationship between *JAK2* mutations and cardiac inflammatory markers, and assess their impact on cardiac structure and function. Exploring these research directions in depth will enhance our understanding of the mechanisms underlying myocardial hypertrophy in PV patients and provide more effective prevention and treatment strategies.

CONCLUSION

This report details a case of PV combined with myocardial hypertrophy. The observed myocardial hypertrophy and symptoms of hypertension may be associated with PV. Clinicians managing PV patients should remain highly vigilant regarding the risks of thrombosis and cardiovascular complications, particularly in those displaying refractory hypertension. Compared to historical cases, the occurrence of myocardial hypertrophy in this case, which is difficult to attribute to other causes, proposes a new hypothesis that PV could be a potential underlying trigger. Therefore, early diagnosis and management of cardiovascular diseases in PV patients are crucial for improving long-term prognosis. It is hoped that this case, along with others in the future, can present similar observational results, thereby contributing to a better understanding of the pathophysiology and management of myocardial hypertrophy in patients with PV.

FOOTNOTES

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Zhao QN revised and contributed equally to the manuscript and figures as co-corresponding authors; all authors have read and approved the final manuscript.

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