# Polycythemia vera

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- Red Blood Cells Are Critical for Hemostasis and Thrombosis
- A molecular signature predicts hematologic evolution in polycythemia vera patients
- Thrombo-vera: a new thrombosis risk model for polycythemia vera using modern variable selection methods
- Digital Necrosis as Presenting Symptom of Polycythemia Vera
- Basophilia and eosinophilia in polycythemia vera and essential thrombocythemia: clinical, genotype, and prognostic correlates
- Current Advances in the Diagnosis and Treatment of Major Myeloproliferative Neoplasms
- Circulating Monocytes Contribute to Erythrocyte Clearance in Polycythemia Vera

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Myeloproliferative neoplasms (MPN) are a group of clonal haematological malignancies first described by Dameshek in 1957.

Philadelphia-negative myeloproliferative neoplasm are polycythemia vera (PV), essential thrombocythaemia (ET), pre-fibrotic myelofibrosis and primary myelofibrosis (PMF).

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Polycythemia vera (PV) is a hematopoietic stem cell neoplasm driven by somatic mutations in JAK2, leading to increased red blood cell (RBC) production uncoupled from mechanisms that regulate physiological erythropoiesis.

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Polycythemia vera (PV) is a clonal proliferative disorder of the bone marrow characterized by autonomous hematopoiesis, which results in a panmyelosis in the peripheral blood. It is typically characterized by an acquired mutation in JAK2 V617F. Progression to myelofibrosis (MF), characterized by worsening cytopenias and the development of constitutional symptoms, is seen in up to 10% of cases. Extramedullary hematopoiesis (EMH) in the spleen is a common finding in myelofibrotic transformation, but elsewhere in the body, it is extremely unusual. Randhawa et al. reported the case of a 69-year-old male whose PV progressed to secondary MF and who presented with compression of the thoracic spinal cord directly as a result of EMH. Cytogenetic and molecular findings in the bone marrow were in keeping with evolving myeloid disease. He was managed by surgical laminectomy with an excellent outcome. Extramedullary hematopoiesis may be seen in both PV and on transformation to MF. This very rare complication should be borne in mind when managing patients with myeloproliferative disorders. [1)].

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Sporadic reports of hemorrhage within the central nervous system in the setting of PV exist and are attributed to microvascular thrombotic events with hemorrhagic conversion. Though rare, spontaneous central nervous system hemorrhage in the absence of vascular malformation or an inciting event such as trauma can occur in the setting of myeloproliferative disorders like

Polycythemia Vera [2) 3)]

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Gautham et al. reported the anesthetic management of a patient with PV who underwent neurosurgical intervention for vestibular schwannoma excision [4)].

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Diffuse large B-cell lymphoma (DLBCL) represent the most frequently non-Hodgkin's lymphoma (NHL) (over 30%), especially in developing countries. Many associations of NHL with another neoplasia were described following chemotherapy or radiotherapy regimens. The coexistence of DLBCL with myeloproliferative neoplasms (MPNs) JAK2V617F positive at the onset was very rare reported in the literature. We describe a clinical case of a 52-year-old man who presented both diagnoses at the onset - DLBCL and MPN - polycythemia vera (PV) type. The patient was treated with two CHOP cycles (Cyclophosphamide, Hydroxydaunorubicin, Oncovin, Prednisolone) followed by six R-CHOP (Rituximab-CHOP) cycles, together with a platelet-reducing agent, achieving remission for 20 months, followed by a relapse which is under treatment. The clonally expansion of an abnormal pluripotent hematopoetic stem cell could be responsible for both, PV and DLBCL. However, recent reports suggested the possible involvement of two different clones. The clinical significance and the role of JAK2 mutation in the evolution of patients with NHLs, including DLBCL are still unknown. Further genetic and clinical studies have to point out common gene mutations for the two diseases and their connection with the diseases behavior under the treatment [5)].

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# Epidemiology

It is estimated to affect more than 100,000 people in the USA [6].

# Clinical features

Patients with PV have burdensome signs and symptoms including pruritus, fatigue, night sweats, concentration problems, and splenomegaly

# Diagnosis

Radiographic manifestations are varied and non-specific and can affect a number of systems, including:

splenomegaly

organ infarcts e.g. splenic infarcts

features of extramedullary hematopoiesis

Increased radiographic attenuation of cerebral arteries, veins and dural sinuses (may mimic cerebral vein thrombosis).

# Phenotypic Characterization

A total of 111 patients with MPN suffering from polycythemia vera, essential thrombocythemia, or primary myelofibrosis (PMF) were examined. In almost all patients tested, CD34+ /CD38- stem cells expressed CD33, CD44, CD47, CD52, CD97, CD99, CD105, CD117, CD123, CD133, CD184, CD243, and CD274 (PD-L1). In patients with PMF, MPN stem cells often expressed CD25 and sometimes also CD26 in an aberrant manner. MPN stem cells did not exhibit substantial amounts of CD90, CD273 (PD-L2), CD279 (PD-1), CD366 (TIM-3), CD371 (CLL-1), or IL-1RAP. The phenotype of CD34+ /CD38- stem cells did not change profoundly during the progression from MPN to sAML. The disease-initiating capacity of putative MPN stem cells was confirmed in NSGS mice. Whereas CD34+ /CD38- MPN cells were engrafted in NSGS mice, no substantial engraftment was produced by CD34+ /CD38+ or CD34- cells. The JAK2-targeting drug fedratinib and the BRD4 degrader dBET6 induced apoptosis and suppressed proliferation in Myeloproliferative Neoplasm stem cells. Together, MPN stem cells display a unique phenotype, including cytokine receptors, immune checkpoint molecules, and another clinically relevant target antigens. Phenotypic characterization of neoplastic stem cells in MPN and sAML should facilitate their enrichment and the development of better, stem cell-eradicating (curative), therapies [7].

# Treatment

Polycythemia vera treatment.

# Outcome

Patient-reported outcomes

Patients with PV presenting with neurological diseases that require surgical intervention are at an increased risk due to various factors, such as immobility, prolonged surgical time, hypothermia and dehydration.

Gautham et al., report anesthetic management of a patient with PV who underwent neurosurgical intervention for vestibular schwannoma excision [8].

# Complications

Increased risks of thrombosis, mortality, and disease transformation to myelofibrosis or acute myeloid leukemia

Thromboembolism is the most common complication of PV, which has significant effects on the quality of life and survival of patients. To standardize the diagnosis, treatment, and prevention strategies of thromboembolism associated with PV, the Myelodysplastic Syndrome/Myeloproliferative Neoplasms (MDS/MPN) Association affiliated to Chinese Medicine Education Association has reached the following consensus based on available proofs from evidence-based medicine after repeated considerations. The consensus covers risk stratification, diagnosis and treatment of PV, multidisciplinary management of PV thromboembolism, and patient follow-up, thereby providing the reference for the standardized diagnosis, treatment, and management of thromboembolism for Chinese PV patients [9].

# Case series

The aim of this study was to determine the frequency and characteristics of peripheral neuropathy associated with polycythemia vera. A prospective clinical and electrophysiological study was performed on 28 patients with polycythemia vera. Other causes of neuropathy were excluded. Eleven patients experienced paresthesia, which was usually mild. In 13 (46%) patients, clinical examination revealed features suggesting polyneuropathy. Nerve conduction indexes were abnormal in 20 (71%) patients, suggesting the presence of predominantly sensory axonal polyneuropathy. In the somatosensory evoked potentials a delay of the P40 wave was seen in 17 patients, while 11 exhibited a delay of the N20 wave. Three of these patients also showed bilateral increases in the I-III, I-V, and III-V intervals of brainstem evoked potentials. In most cases, the delay was moderate and symmetrical. No differences in sex, age, duration of disease, hematocrit values, or platelet counts were found between patients with or without clinical or electrophysiological polyneuropathy. A high percentage of patients with polycythemia vera present clinical or electrophysiological signs of predominantly sensory axonal polyneuropathy which is probably secondary to ischemia, due to

increased blood viscosity and platelet dysfunction [10)]

# Case reports

A 74-year-old Japanese woman was referred for leukocytosis that occurred for the past one year. Oral iron supplementation was started as iron deficiency anemia (IDA), but three months later, physical examination revealed flushing of the skin on her hands. Finally polycythemia vera (PV) with IDA was diagnosed. There have been reports of PV combined with IDA, which can mask diagnosis and delay treatment because of the lack of symptoms and the anemic presentation. Several possibilities for the pathogenesis of IDA associated with PV have been proposed, including the presence of Helicobacter pylori [11)]

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A 70-year-old male with a known case of beta-thalassemia trait and was on yearly follow-up was found to have a hemoglobin of 14.8 g/dL, hematocrit of 47.7%, and RBC count of 6.0 × 1012/L. Total leukocyte count (TLC) was 5 × 109/L and platelet count was 4 × 109/L. Secondary causes of polycythemia were ruled out (e.g., renal or cardiac disease and smoking). He did not have symptoms of hyperviscosity syndrome. The abdominal ultrasound showed no abnormalities. On further investigation, a JAK-2 (Exon 14) mutation was detected in this patient confirming the diagnosis of polycythemia vera (PV) [12)].

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A case of a fertile-age woman with menometrorrhagia, whose blood loss and consequent iron depletion worked as a limiting factor for Hb and Htc increase, delaying the proper diagnosis. Splenomegaly, iron deficiency markers, and low erythropoietin supported PV investigation. The correction of iron depletion led to the unveiling of covert erythrocytosis. Concomitant hemoglobinopathies and secondary causes for erythrocytosis were excluded. The diagnosis was confirmed with polymerase chain reaction (PCR) for V617F-JAK2 mutation and bone marrow biopsy. As this case highlights, despite not meeting diagnostic criteria at presentation, masked PV exhibited clinical, laboratory, and imaging features of active symptomatic disease. For that, a higher level of suspicion must be held for fertile-age women who present with normal Hb and Htc levels and significant iron depletion, in the presence of low serum erythropoietin or splenomegaly [13)].

## 2022

A 46-year-old man who had an isolated cerebellar infarct with high hematocrit and hemoglobin levels and low serum erythropoietin levels is described. Further investigations eventually led to the unmasking of a JAK2 mutation-negative polycythemia vera [14)].

## 2019

Spontaneous hemorrhage/bleeding in PV patients is seldom reported in Neurosurgical literature.

Entezami et al., report the case of a 76 year-old male with PV who developed a spontaneous subdural

hematoma requiring surgical evacuation. He improved significantly after the resolution of brain compression and mass effect caused by the hematoma. Sporadic reports of hemorrhage within the central nervous system (CNS) in the setting of PV exist, and are attributed to microvascular thrombotic events with hemorrhagic conversion. Though rare, spontaneous CNS hemorrhage in the absence of vascular malformation or an inciting event such as trauma can occur in the setting of myeloproliferative disorders such as PV [15].

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A clinical case of a 52-year-old man who presented both diagnoses at the onset - DLBCL and MPN - polycythemia vera (PV) type. The patient was treated with two CHOP cycles (Cyclophosphamide, Hydroxydaunorubicin, Oncovin, Prednisolone) followed by six R-CHOP (Rituximab-CHOP) cycles, together with a platelet-reducing agent, achieving remission for 20 months, followed by a relapse which is under treatment. The clonally expansion of an abnormal pluripotent hematopoetic stem cell could be responsible for both, PV and DLBCL. However, recent reports suggested the possible involvement of two different clones. The clinical significance and the role of JAK2 mutation in the evolution of patients with NHLs, including DLBCL are still unknown. Further genetic and clinical studies have to point out common gene mutations for the two diseases and their connection with the diseases behavior under the treatment.

The coexistence of NHLs and especially DLBCLs and MPNs JAK2 positive is very rare. Although DLBCL alone has good prognosis, other prognostic factors should be checked when it is associated with PV. The presence of JAK2V617F seems to be a candidate but whose role in DLBCL evolution, natural or under treatment has to be cleared up [16].

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The most common neurologic manifestations of polycythemia vera (PV) are cerebral infarction and transient ischemic attacks, while cerebral hemorrhage or intracranial dissection has been rarely associated with PV. Here we report the first case of a 59-year-old patient with intracranial supraclinoid internal carotid artery (ICA) dissection causing cerebral infarction and concomitant subarachnoid hemorrhage due to pseudoaneurysm rupture as clinical onset of PV. This case report discusses the possible mechanism and treatment of this extremely rare condition [17].

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A 68-year-old woman presented with an acute SDH requiring craniotomy and a thrombosed cortical vein was noted intraoperatively. Computed tomography venography showed thrombosis of the superior sagittal sinus. She had polycythemia vera with the V617 Jak2 gene mutation and was managed with aspirin and hydroxyurea [18].

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