

Cerebral Infarction As the Presenting Feature of Janus Kinase 2 (JAK2) V617F-Positive Polycythemia Vera: A Case Report

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Yoshita Pendharkar¹, Sunanda Chaoji¹, Tanuja Manohar¹, Nishka Tiwari¹, Fahad Idrees Shaikh¹

¹. Internal Medicine, NKP Salve Institute of Medical Sciences and Research Centre and Lata Mangeshkar Hospital, Nagpur, IND

Corresponding author: Fahad Idrees Shaikh, fahad.shaikh2004@gmail.com

Abstract

Polycythemia vera (PV) is a clonal myeloproliferative neoplasm driven predominantly by the Janus kinase (JAK)2 V617F mutation and characterized by an absolute increase in red cell mass. Because its early features are frequently nonspecific, PV can remain undetected until a thrombotic event - commonly an arterial ischemic stroke - brings the patient to medical attention. We report a 56-year-old woman with a two-year history of systemic hypertension who presented with a six-day history of insidious, distally predominant weakness of the left lower limb, with no other neurological deficit or limb involvement recorded. Magnetic resonance imaging of the brain demonstrated an acute infarct in the right high frontal region together with chronic small-vessel ischemic changes. Laboratory evaluation revealed marked erythrocytosis (hemoglobin 19 g/dL, hematocrit 62%), a normal serum erythropoietin level, and a positive JAK2 V617F mutation; bone marrow biopsy showed erythroid hyperplasia, with no further morphological detail available. A working diagnosis of PV was made on the combined clinical, hematological, and molecular findings. In the absence of a documented alternative cause on the available work-up, PV was considered a plausible contributor to the infarction. The patient was started on dual antiplatelet therapy and a high-intensity statin for secondary stroke prevention, underwent serial therapeutic phlebotomy (hematocrit falling from 62% to 50.8% by day 22), and was discharged on hydroxyurea 500 mg twice daily. Left lower-limb power improved from 0/5 at admission to 3/5 at discharge on day 9. This case illustrates the importance of considering PV in the differential diagnosis of stroke, particularly when routine blood counts show unexplained erythrocytosis, since early recognition and cytoreductive therapy may reduce the thrombotic risk, though longer follow-up and a more complete stroke work-up are needed to confirm this in the individual patient.

Categories: Neurology, Hematology, Internal Medicine

Keywords: cerebral infarction, hydroxyurea, ischemic stroke, jak2 v617f mutation, myeloproliferative neoplasm, phlebotomy, polycythemia vera

Introduction

Polycythemia vera (PV) is a clonal myeloproliferative neoplasm associated with erythrocytosis. In most patients, a Janus kinase (JAK)2 mutation is involved [1,2]. Thrombosis is multifactorial and reflects increased blood viscosity, abnormal interactions among blood cells and the vascular endothelium, and inflammatory/prothrombotic mechanisms [2]. Because early symptoms may be mild or nonspecific, PV can remain undiagnosed until a thrombotic event prompts evaluation [2].

Cerebral infarction is a serious complication. A large prospective cohort identified thrombosis as a major complication of PV, with age over 65 years and a history of thrombosis as the strongest predictors of cardiovascular events [3], and an older series described vascular accidents in polycythemia [4]. These reports should be interpreted in the context of their cohort design and era. PV can also occur in younger adults, as described in a series of patients diagnosed before the age of 40 years [5]. An unexplained elevation in hemoglobin or hematocrit during stroke evaluation should therefore prompt targeted hematologic assessment. We report a case in which marked erythrocytosis and JAK2 V617F positivity were identified during evaluation of an acute ischemic stroke.

Case Presentation

A 56-year-old woman presented with a six-day history of insidious-onset weakness of the left lower limb, more pronounced distally than proximally. She had a two-year history of systemic hypertension on regular treatment.

On examination, her pulse was 68 beats/min and regular, and her blood pressure was 150/90 mmHg. No hepatosplenomegaly was detected. At admission, she was fully conscious and oriented to time, place, and person; speech and memory were intact. All cranial nerves and bilateral fundi were reported as normal. Motor examination showed normal tone and power (Medical Research Council (MRC) score [6] was 5/5) in the right upper limb, left upper limb, and right lower limb. The left lower limb was hypotonic, with power

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graded 0/5. Biceps and triceps jerks were 2+ bilaterally; the right knee jerk was 2+, the left knee jerk was absent, and ankle jerks were absent bilaterally. The right plantar response was flexor, and the left was extensor. Pain, touch, temperature, vibration, and joint-position sensation were normal bilaterally. Upper-limb coordination was normal; left lower-limb coordination could not be assessed because of weakness, and gait could not be assessed. The National Institutes of Health Stroke Scale (NIHSS) score [7] was 4, and the modified Rankin Scale score [8] was 5 at admission. At discharge on day 9, left lower-limb power was documented as 3/5, with an NIHSS score of 2 and a modified Rankin Scale score of 4. Other discharge neurological findings, including repeat reflex, sensory, cranial nerve, and tone assessments, were not available in the records provided, as shown in Table 1.

Neurological domain	At admission	At discharge (day 9)
Mental status	Fully conscious; oriented to time, place, and person; speech and memory intact	Not recorded
Cranial nerves	All cranial nerves normal; fundi normal bilaterally	Not recorded
Power, left upper limb (MRC 0-5)	Power 5/5; tone normal	Not recorded
Power, left lower limb (MRC 0-5)	Power 0/5; hypotonia	Power 3/5 (documented at discharge)
Power, right upper limb (MRC 0-5)	Power 5/5; tone normal	Not recorded
Power, right lower limb (MRC 0-5)	Power 5/5; tone normal	Not recorded
Muscle tone	Normal in all limbs except hypotonia in left lower limb	Not recorded
Deep tendon reflexes	Biceps/triceps 2+ bilaterally; right knee 2+, left knee absent; ankle jerks absent bilaterally	Not recorded
Plantar responses	Right flexor; left extensor	Not recorded
Sensory examination	Pain, touch, temperature, vibration, and joint position normal bilaterally	Not recorded
Coordination	Upper limbs normal; left lower limb not assessable due to weakness	Not recorded
Gait	Could not be assessed	Not recorded
NIHSS score	4	2
Functional status (e.g., modified Rankin Scale)	5	4

TABLE 1: Neurological examination at admission and at discharge on day 9, based on the available clinical records.

MRC muscle-strength scale (0 = no contraction; 5 = normal power). The only documented discharge motor-power measurement was left lower-limb power (3/5); other discharge neurological findings were not available in the records provided.

MRC: Medical Research Council

At presentation, the complete blood count showed hemoglobin 19 g/dL (reported laboratory reference range 12-16 g/dL), red-cell count 7.90 million/μL (3.5-6.0 million/μL), hematocrit 62% (37-52%), mean corpuscular volume (MCV) 78.4 fL (67-96 fL), mean corpuscular hemoglobin (MCH) 24.1 pg (27-32 pg), and white cell count 8,730/μL (4,000-10,000/μL) [9]. The peripheral smear was described as showing predominantly normochromic red cells with mild hypochromia and anisopoikilocytosis, occasional microcytes and elliptocytes, and adequate platelets. Serum bilirubin was 0.3 mg/dL (reference range: 0.2-1.3 mg/dL), and renal function was reported as normal. Serial hematologic parameters before and after therapeutic phlebotomy are listed in Table 2.

Parameter	Reference range	Initial presentation	Day 4 (post-phlebotomy 1)	Day 9 (post-phlebotomy 2)	Day 22 (13-day post-phlebotomy 2)
Hemoglobin (g/dL)	12-16	19.0	17.9	16.7	15.50
RBC count (million/ μ L)	3.5-6.0	7.90	7.58	6.90	6.49
Hematocrit (%)	37-52	62.0	59.0	53.7	50.80
MCV (fL)	67-96	78.4	77.9	77.9	78.20
MCH (pg)	27-32	24.1	23.6	24.2	23.90
WBC count (cells/ μ L)	4,000-10,000	8,730	6,780	8,600	5,070

TABLE 2: Serial hematologic parameters before and after therapeutic phlebotomy.

WBC: white blood cell; MCV: mean corpuscular volume; MCH: mean corpuscular hemoglobin; RBC: red blood cell

Serial blood counts after therapeutic phlebotomy are summarized in Table 2. According to the last recorded measurement, hemoglobin had decreased to 15.5 g/dL and hematocrit to 50.8%; the hematocrit had fallen from baseline but remained above the commonly recommended target of less than 45% [2]. Her serum erythropoietin was 7.07 mIU/mL (reference range: 4.3-29 mIU/mL). A value within the laboratory reference range is not independently diagnostic and does not, by itself, exclude secondary erythrocytosis. JAK2 V617F testing was positive, and bone marrow biopsy was reported as showing erythroid hyperplasia. The available pathology description did not specify marrow cellularity, panmyelosis, or megakaryocyte morphology.

Brain MRI demonstrated a few altered-signal foci in the right high-frontal region, appearing hypointense on T1-weighted imaging and hyperintense on T2-weighted imaging and fluid-attenuated inversion recovery (FLAIR), with diffusion restriction reported on diffusion-weighted imaging (DWI). Also, no susceptibility blooming was reported on gradient-recalled echo (GRE) imaging (Figure 1). The report also described mild generalized cerebral atrophy and bilateral frontoparietal subcortical and periventricular white-matter hyperintensities consistent with nonspecific chronic small-vessel changes (Fazekas grade I). Motion artefact was noted, and the corresponding apparent diffusion coefficient (ADC) images were unavailable for review.

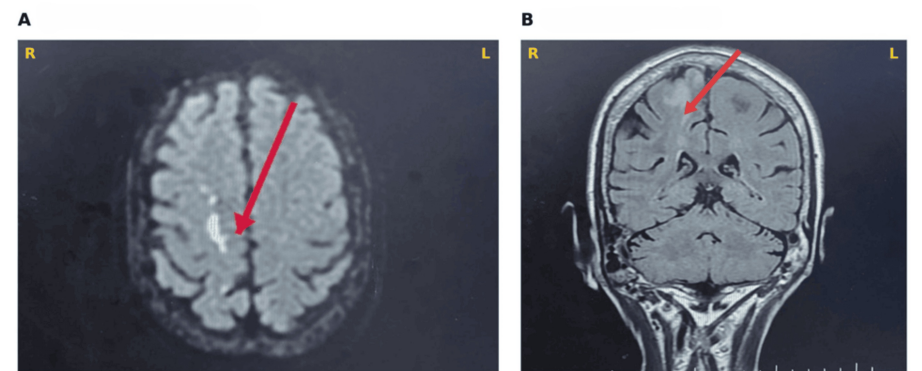


FIGURE 1: Brain magnetic resonance imaging (MRI) findings. (A) Axial diffusion-weighted imaging (DWI; b = 1000 s/mm²) demonstrating a focal area of increased signal in the right high-frontal region, as described in the radiology report. (B) Coronal T2-weighted fluid-attenuated inversion recovery (T2-FLAIR) image demonstrating the corresponding region. The arrows indicate the reported right high-frontal abnormality.

The intracranial MR angiography report described the arteries of the anterior and posterior circulations and the circle of Willis as normal, without reported focal stenosis or occlusion. Figure 2 shows a representative

coronal projection image from the multiple overlapping thin-slab acquisition (MOTSA) neck angiography series. Although the neck angiography series was acquired, the available report did not separately characterize the carotid and vertebral arteries. Therefore, the absence of extracranial arterial stenosis or occlusion cannot be independently established.

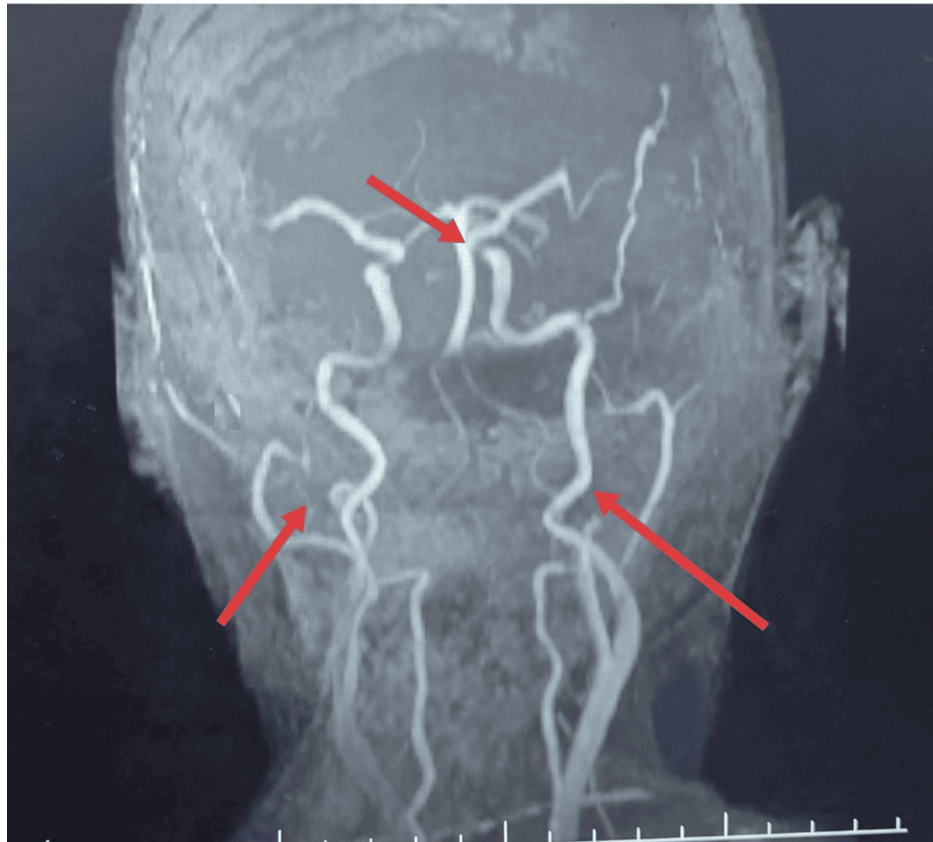


FIGURE 2: Coronal projection image from the neck multiple overlapping thin-slab acquisition (MOTSA) magnetic resonance angiography (MRA) series. Arrows indicate representative visible arterial pathways.

A working diagnosis of PV was made on the combined findings of marked erythrocytosis (hemoglobin 19 g/dL, hematocrit 62%), a positive JAK2 V617F result, and bone marrow reported as showing erythroid hyperplasia. Against the World Health Organization (WHO)-based criteria discussed in Tefferi et al. [2], the hemoglobin/hematocrit and JAK2 V617F major criteria were met. The bone marrow major criterion could not be verified because the available description did not provide the morphological details required (marrow cellularity, panmyelosis, and megakaryocyte morphology), and the minor criterion of subnormal serum erythropoietin was not met because the level was within the reference range. The diagnosis is therefore presented as a clinical diagnosis supported by the available hematological and molecular findings, rather than as one in which every formal criterion was independently verified.

A comprehensive evaluation for cardioembolic and extracranial large-vessel sources, including the documented electrocardiography or telemetry, echocardiography, and detailed carotid and vertebral artery imaging findings, was not available in the submitted records. Although the radiology report described the intracranial arteries and circle of Willis as normal, this does not exclude a cardiac embolic source, extracranial vascular disease, or a small-vessel mechanism. PV represents a plausible contributor to the patient's thrombotic risk; however, the available evidence does not establish that PV was the exclusive cause of the cerebral infarction.

For secondary stroke prevention and PV management, the patient received the following (Table 3).

Phase	Drug (as recorded; generic)	Dose	Route/frequency	Duration
In hospital	Clopitab-A (clopidogrel + aspirin)	75 mg + 75 mg	Oral, at bedtime	During admission
In hospital	Atorvas (atorvastatin)	40 mg	Oral, at bedtime	During admission
In hospital	Telma (telmisartan)	20 mg	Oral, once daily	During admission
In hospital	Therapeutic phlebotomy	About 350 mL per sitting	Three sittings, three to four days apart	-
At discharge	Hydroxyurea	500 mg	Oral, twice daily	2 months
At discharge	Clopitab-A (clopidogrel + aspirin)	75 mg + 75 mg	Oral, at bedtime	2 months
At discharge	Atorvas (atorvastatin)	40 mg	Oral, at bedtime	2 months
At discharge	Telma (telmisartan)	20 mg	Oral, once daily	2 months
At discharge	Folic acid	5 mg	Oral, once daily	15 days
Discharge advice	Outpatient phlebotomy	-	Twice weekly	Until hematocrit <45%

TABLE 3: Treatment during hospitalization and at discharge, from the discharge record. Drug names are shown as recorded (brand) with the corresponding generic names.

During admission, she received a fixed-dose combination of clopidogrel 75 mg and aspirin 75 mg (Clopitab-A) at bedtime as dual antiplatelet therapy, atorvastatin 40 mg at bedtime, and telmisartan 20 mg once daily. Therapeutic phlebotomy was performed in three sittings three to four days apart, with about 350 mL of blood removed at each sitting, producing a stepwise reduction in hemoglobin and hematocrit (Table 2). At discharge, she was prescribed hydroxyurea 500 mg twice daily, dual antiplatelet therapy (clopidogrel 75 mg + aspirin 75 mg at bedtime), atorvastatin 40 mg at bedtime, and telmisartan 20 mg once daily, each for two months, together with folic acid 5 mg once daily for 15 days. Discharge advice was to continue phlebotomy twice weekly until the hematocrit was below 45%, to have a complete blood count after one week, and to follow up with a hematologist. Therefore, the planned dual antiplatelet course was two months; the antithrombotic therapy beyond that period, outpatient phlebotomy outcome, and any later hydroxyurea titration were not documented. A previous thrombotic event places a patient with PV in a high-risk category for which cytoreductive therapy is generally recommended [2].

Discussion

Cerebral infarction has a broad differential diagnosis, including cardioembolism, small-vessel disease, large-artery atherosclerosis, and prothrombotic disorders. PV should be considered when stroke is accompanied by otherwise unexplained erythrocytosis. Diagnostic assessment integrates blood counts, JAK2 mutation testing, serum erythropoietin, and bone marrow morphology where indicated [2]. Relative erythrocytosis due to reduced plasma volume must be distinguished from absolute erythrocytosis, and primary from secondary causes [2].

In this patient, hemoglobin (19 g/dL) and hematocrit (62%) exceeded the female thresholds cited by Tefferi et al. [2]. Because serum erythropoietin was within the laboratory reference range, the available diagnostic evidence included JAK2 V617F positivity and a marrow report describing erythroid hyperplasia. A normal-range erythropoietin level is not independently diagnostic and does not, by itself, exclude secondary erythrocytosis. The limited marrow description does not permit independent assessment of all morphologic diagnostic criteria, and the diagnosis of PV in this patient is accordingly a working diagnosis based on the combined hematological, molecular, and limited marrow findings.

PV-associated thrombosis is attributed to increased blood viscosity and abnormal interactions among erythrocytes, leukocytes, platelets, and the vascular endothelium, with additional inflammatory and prothrombotic effects [2,4]. These are mechanisms proposed in the literature; none was directly demonstrated in this patient, and population-level associations cannot establish the mechanism of an individual stroke.

The documented findings are distal-predominant left lower-limb weakness, right high-frontal foci reported to show diffusion restriction, a report of no focal intracranial stenosis or occlusion, and marked JAK2 V617F-

positive erythrocytosis. A right high-frontal lesion may be anatomically compatible with left-sided, leg-predominant weakness if the medial frontal motor region is involved.

The accompanying neurological signs are compatible with an acute central lesion. Flaccid weakness with hypotonia, a depressed or absent tendon reflex, and an extensor plantar response on the same side can occur in the early phase after an acute upper motor neuron lesion, with spasticity and hyperreflexia typically emerging later. Tone and reflexes were not recorded at discharge, so this evolution could not be documented. Two findings are not explained by a right frontal infarct alone. The bilaterally absent ankle jerks may reflect age-related change or an unrecognized peripheral cause, and a peripheral (for example, lumbosacral or neuropathic) contribution to the absent left knee jerk was not evaluated, as no spinal imaging or nerve conduction studies were available.

PV may have contributed to the infarction, but this remains an inference. Chronic bilateral frontoparietal and periventricular white-matter changes (Fazekas grade I), together with hypertension, provide possible alternative or additive mechanisms. The reported normal intracranial angiography does not exclude cardioembolism or extracranial large-artery disease; a complete etiologic work-up was not available in the submitted records. PV is therefore a plausible and potentially important contributor, not a proven exclusive cause.

A large prospective cohort of 1,638 patients with PV reported thrombosis and cardiovascular death among its main outcomes, with a history of thrombosis and age over 65 years the most important predictors of cardiovascular events [3]. Such cohort data support vigilance for vascular complications but do not establish the frequency or mechanism of stroke in an individual patient. Leukocytosis has been associated with thrombotic risk in PV and other Philadelphia chromosome-negative myeloproliferative neoplasms [10,11]. This patient's admission white cell count was 8,730/ μ L (8.73×10^9 /L), below the 9.5×10^9 /L threshold reported in one study [10]; population-level associations should not be used to infer an individual stroke mechanism.

JAK2 V617F is a common molecular driver in PV and produces constitutive JAK-STAT signaling [2]. The limited marrow description available here (erythroid hyperplasia without details of panmyelosis or megakaryocyte morphology) is an important diagnostic-reporting constraint [2].

Historical treatment studies identified long-term safety concerns with chlorambucil, which was associated with an increased incidence of acute leukemia [12], and showed poorer remission and survival with radiophosphorus than with busulfan in a randomized comparison [13]. Current management aims to reduce thrombotic risk through hematocrit control, antithrombotic therapy when appropriate, and cytoreduction for higher-risk patients. A hematocrit target below 45% is commonly recommended, with hydroxyurea among the cytoreductive options used in high-risk PV [2,14]. In this case, hematocrit decreased after phlebotomy but was 50.8% at the last recorded measurement. The discharge plan specified continued phlebotomy until the hematocrit was below 45%, but the available data do not establish whether the target was reached later.

This case illustrates that unexplained erythrocytosis encountered during stroke evaluation merits prompt hematologic assessment, because PV is a treatable source of thrombotic risk. The main limitations of the report are the incomplete neurological and imaging documentation described further.

The admission record documented mental status, cranial nerves, motor power and tone, reflexes, plantar responses, sensation, and coordination. Left lower-limb power improved from 0/5 at admission to 3/5 at discharge on day 9, with NIHSS and modified Rankin Scale scores improving from 4 to 2 and from 5 to 4, respectively. However, other discharge neurological findings and later serial examinations were unavailable.

No ADC images were available. Only a single magnetic resonance angiography (MRA) projection image was available for independent review. The intracranial vessels were reported as normal, but extracranial carotid/vertebral findings were not documented, and a complete stroke etiologic work-up was not available.

Bone marrow findings were summarized only as erythroid hyperplasia. The last available hematocrit remained above 45%, and later laboratory values and the total follow-up duration were unknown. The antithrombotic plan beyond the prescribed two-month course of dual antiplatelet therapy, the outcome of outpatient phlebotomy, and any hydroxyurea titration were not documented. These gaps restrict quantitative assessment of neurological recovery and definitive attribution of the infarct mechanism.

Conclusions

PV is an important, treatable prothrombotic disorder that should be considered when ischemic stroke is accompanied by unexplained erythrocytosis. In this patient, marked erythrocytosis, JAK2 V617F positivity, and reported marrow erythroid hyperplasia supported a working diagnosis of PV, which is a plausible contributor to a right high-frontal infarct. Hypertension, chronic small-vessel changes, and an incomplete cardioembolic and extracranial work-up mean that exclusive causation by PV cannot be established.

Hematocrit decreased after phlebotomy but remained above 45% at the last available measurement, and the outcome of the planned outpatient phlebotomy was not documented. Although left lower-limb power was documented as improving from 0/5 at admission to 3/5 at discharge on day 9 (NIHSS 4 to 2; modified Rankin scale 5 to 4), no later serial neurological examination or longer-term outcome was available. Systematic documentation of longer-term neurological outcomes and submission of original diagnostic-quality imaging would strengthen future reports of this kind.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Yoshita Pendharkar, Sunanda Chaoji, Tanuja Manohar, Nishka Tiwari, Fahad Idrees Shaikh

Acquisition, analysis, or interpretation of data: Yoshita Pendharkar, Sunanda Chaoji, Tanuja Manohar, Nishka Tiwari, Fahad Idrees Shaikh

Drafting of the manuscript: Yoshita Pendharkar, Sunanda Chaoji, Tanuja Manohar, Nishka Tiwari, Fahad Idrees Shaikh

Critical review of the manuscript for important intellectual content: Yoshita Pendharkar, Sunanda Chaoji, Tanuja Manohar, Nishka Tiwari, Fahad Idrees Shaikh

Supervision: Sunanda Chaoji, Tanuja Manohar

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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