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Case Report
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A Case Report of Parvoviral Infection of the Bone Marrow Masquerading as Lymphoma in a APLA Positive Young Male

Shouree K.R.[1], Mahabaleshwar M.[2], Vani Krishnamurthy[3]

[1]Senior Resident, Department of Pathology, JSS Hospital, JSS Academy of Higher Education and Research, Mysuru, Karnataka, India
[2]Assistant Professor, Department of Rheumatology, JSS Hospital, JSS Academy of Higher Education and Research, Mysuru, Karnataka, India
[3]Associate Professor, Department of Pathology, JSS Hospital, JSS Academy of Higher Education and Research, Mysuru, Karnataka, India

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ABSTRACT

Introduction: abnormal morphology can be seen frequently in the native cells of bone marrow in non-clonal disorders like viral, bacterial, parasitic infections, autoimmune diseases, after chemotherapy, megaloblastic anemia, and many more conditions. Blood counts may be normal or decreased in such scenarios. Such morphological atypicality can sometimes result in diagnostic fallacy of malignancies and unnecessary laboratory tests. When organomegaly and lymphadenopathy are seen apart from other clinical symptoms, it is possible for giant proerythroblasts found in marrow aspirate of a clinically undetected parvoviral infection to be misidentified as high-grade lymphoma cells. We hereby present a case of Parvoviral infection in an immunocompetent young male who was APLA positive. Diagnosis of Systemic Parvoviral infection was made after Immunohistochemical examination. Symptomatic therapy was administered along with anticoagulants for venous thrombosis of the lower limb veins. During the follow-up, the patient's blood counts were normal, and he had improved clinically. We conclude by highlighting, all sinister appearing cells may not be malignant.

Keywords: Parvovirus B19; Lymphoma; Giant Proerythroblasts; Bone Marrow; APLA.

Introduction

Parvovirus B19, the only known human pathogenic virus belonging to the family of *Parvoviridae* is a single stranded DNA virus[1]. Parvoviruses are known for their wide variety of clinical presentations, despite their propensity to damage erythroblasts in bone marrow[2]. Coexistence of peripheral blood cytopenia, organomegaly such as hepatomegaly, and lymphadenopathy, in conjunction with atypical cells in the bone marrow generally raises the suspicion of haematolymphoid malignancy especially Lymphoma. We present the case of a 32-year old male, APLA positive, having clinically undetected parvoviral infection that masqueraded as lymphoma.

Case History

In a tertiary care institution, a 32-year-old male having primary antiphospholipid antibody syndrome (APS) was receiving conservative treatment for venous thrombosis of lower limb veins.

During the hospital stay, he developed fever and pain abdomen with progressive anemia and thrombocytopenia. The hemoglobin and RBC count which were 11.3g% and 4.26 million/cumm respectively during admission had dropped to 8.5g% and 3.4 millions/cumm when he had developed fever spikes. Platelet was 0.37 L/cumm, from the earlier 0.89 L/cumm. Hepatomegaly and enlarged lymph nodes in the retroperitoneum detected on an abdominal CT scan. Bone marrow was aspirated along with marrow biopsy. Marrow was cellular displaying 50 to 60% cellularity. All the three blood cell precusors seen in varying stages of maturation (Fig 1). Along with the native marrow cells, good number of large cells were noted with high nucleocytoplasmic ratio (N:C), open nuclear chromatin and prominent nucleoli (Fig 2 and 3). Retroperitoneal lymph node biopsy and Immunohistochemical (IHC) evaluation along with IHC of marrow biopsy was suggested to rule out Lymphoma. IHC was done on bone marrow biopsy. The large atypical looking cells were highlighted with CD 71 (erythroid lineage marker) and C – kit (also called CD117, expressed predominantly in bone marrow stem/progenitor cells) and on review of the aspiration smears, the gigantic proerythroblasts, misinterpreted as atypical cells, had pink intranuclear inclusions, suggesting Parvoviral inclusion bodies (Fig 4). Patient had been discharged by the time of receiving IHC report and he could not be traced. Hence serological confirmation of the parvoviral infection was not possible.

Paraffin block of bone marrow biopsy was outsourced to get IHC done and hence PCR for Parvoviral antigen was also not possible.

Final diagnosis of Systemic Parvoviral infection was made. Apart from anticoagulants for venous thrombosis, patient recovered completely with symptomatic treatment. He was in good health six weeks following the discharge and follow up blood

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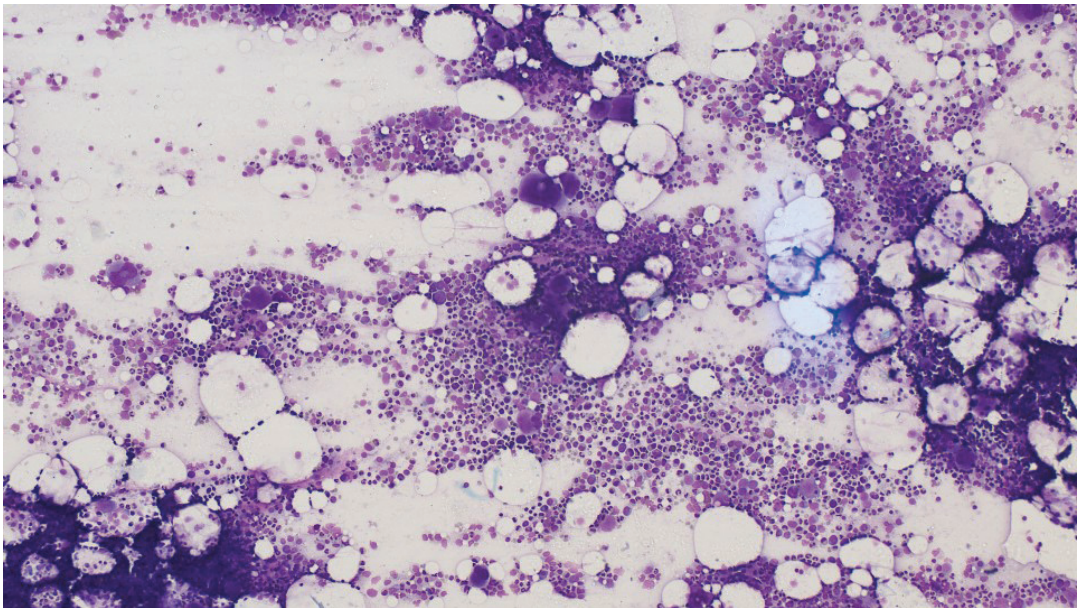

Figure 1. Cellular Bone Marrow Aspirate displaying trilineage hematopoiesis. (Leishman's stain.100X)

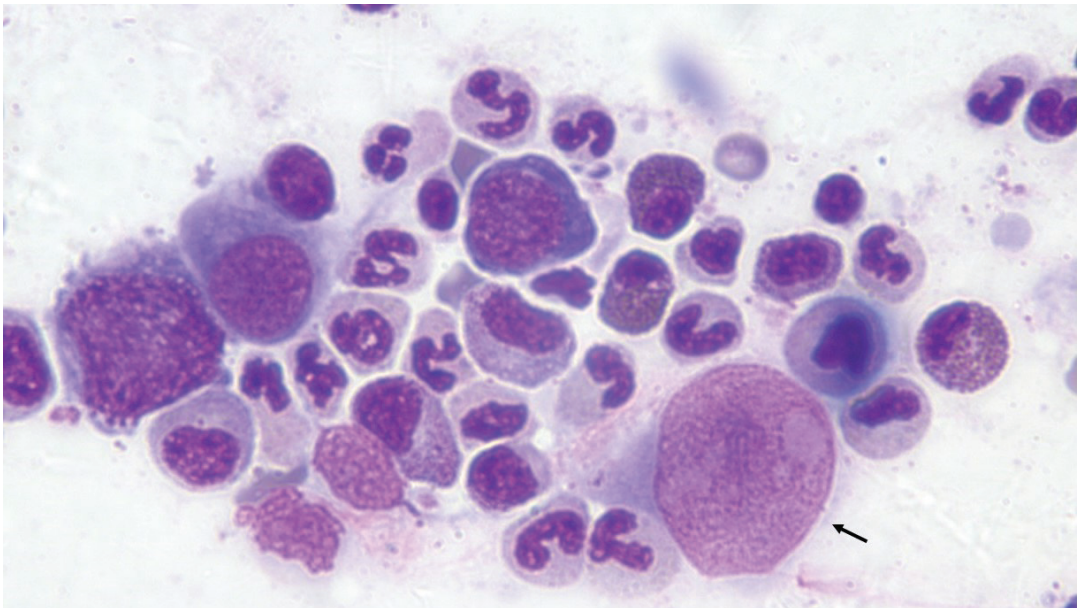

Figure 2. Bone Marrow Aspirate showing large, atypical cells with high N:C ratio (black arrow) and prominent nucleoli /? Viral inclusions (Leishman's stain, 1000X)

counts revealed 10g/dl of hemoglobin, 3.83 million/cumm of RBCs and 2.45 L/cumm of platelets.

Discussion

Parvovirus B19 is generally asymptomatic in healthy adults. In a small proportion of patients, it presents with a broad array of systemic and haematological manifestations. Symptomatic disease is common in immunocompromised individuals[3]. The B19V virus primarily affects the erythroblasts in the bone marrow because its receptor is only present on erythroid precursor cells[4]. Therefore, pure red cell aplasia, the common haematological symptom, is believed to stem from erythropoiesis being compromised along the course of the disease[5].

Maturation arrest and atypia of the erythroblasts are the characteristic bone marrow picture in parvoviral infection. Occasionally, morphological atypia of the normoblasts can be so extensive, which may lead to suspicion of bone marrow infiltration with a malignancy. Clinical presentation of progressive pancytopenia with organomegaly and lymphadenopathy in an immunocompetent individual further bolsters the suspicion. The present case of the young man admitted for management of deep vein thrombosis with prior hypercoagulable state

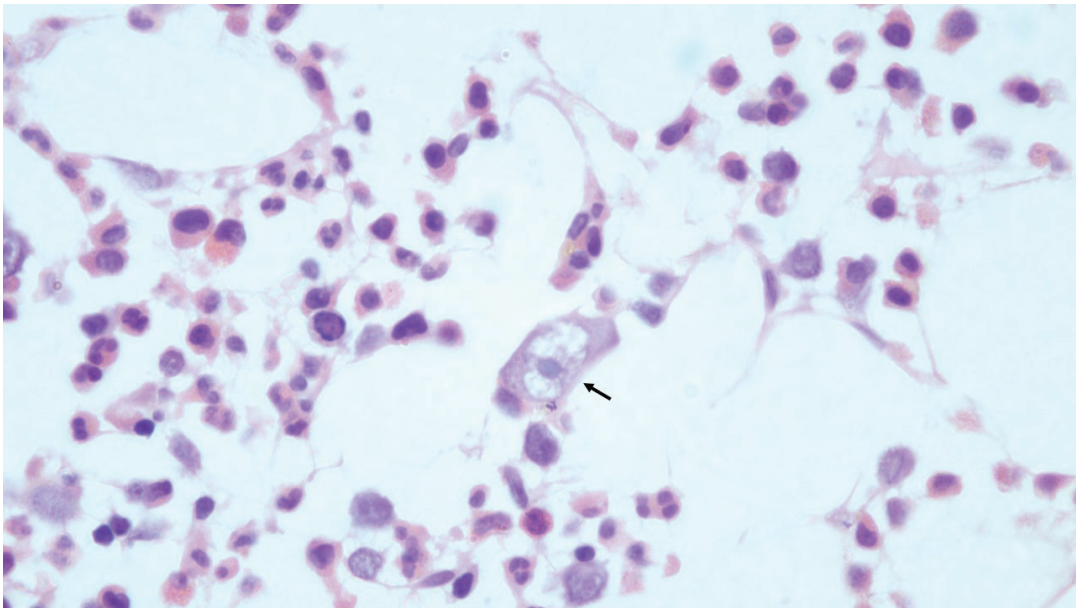

Figure 3. Bone Marrow Trephine Biopsy showing large, atypical cell shown by black arrow (H&E, 200X)

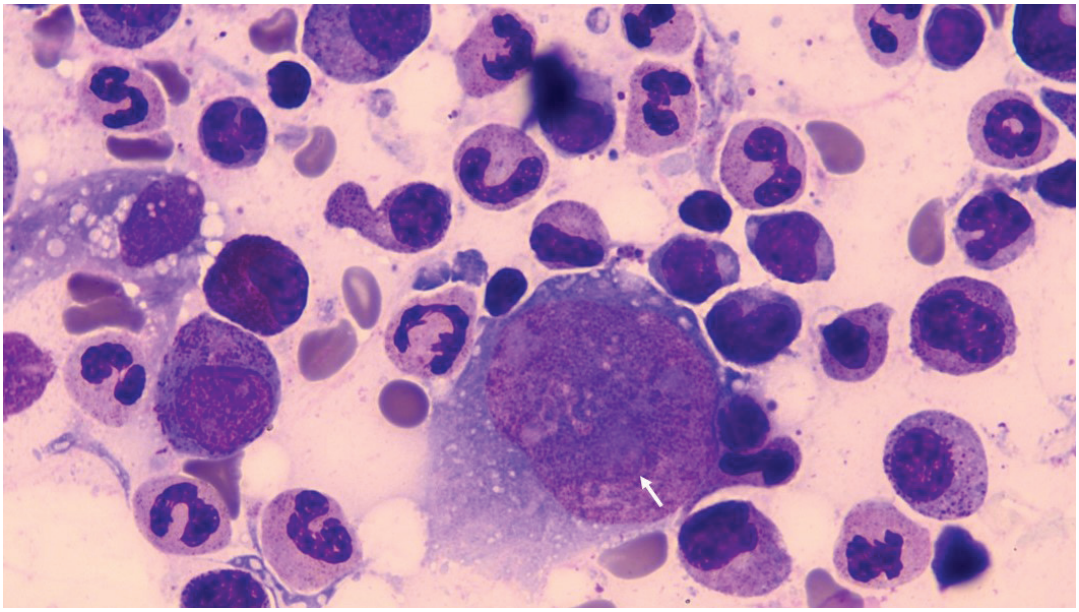

Figure 4. Bone Marrow Aspirate showing giant Proerythroblasts with nuclear inclusions shown by white arrow (Leishman'stain, 1000X)

is an example for misinterpretation as lymphoma infiltration into bone marrow on morphological grounds. Clinical and ultrasonographic findings of hepatomegaly and retroperitoneal lymphadenopathy along with morphological atypia of the erythroblasts in the marrow of this otherwise immunocompetent individual were the deceptive findings.

The link between acute parvoviral infection and lymphadenopathy was originally observed in three of the SLE patients who had enlargement of the epitrochlear, cervical, and supraclavicular lymph nodes[6]. Since then, numerous reports of parvoviral infections presenting at different sites with lymphadenopathy have been made[4,7,8,9,10,11]. Later diverse nodal response patterns have been documented such as necrotizing lymphadenitis, reactive hyperplasia with varied patterns of B cell responses[4], and apoptotic sinus histiocytosis.[7] In many of the case reports, the nodal enlargement has clinically raised the suspicion of lymphoma[7,8].

Rarely, instances of hepatitis and hepatomegaly have been documented in conjunction with systemic parvoviral infection; often manifesting as jaundice, anemia, and increased enzyme levels[12,13]. Chronic hepatitis, fulminant hepatic failure, and increased hepatic enzyme levels are all examples of parvovirus-

induced liver illness[14]. In a study by Mihaly *et al*[15], the author has stated that, 4.1% of patients infected with parvovirus may have virus related hepatitis. The patient in this instance exhibited moderate liver function abnormalities, including a slight rise of bilirubin and hepatic enzymes, but after six weeks, follow-up revealed that everything had reverted to normal.

A varied spectrum of clinical manifestations is the feature of viral infections such as Parvovirus. They can manifest in the host in unusual and unexpected clinical circumstances. Atypia and cytopathic effects induced by them can sometime mislead pathologists towards a more sinister diagnosis and result in unnecessary anxiety and laborious investigations.

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Mini Curriculum and Author's Contribution

1. Shouree K.R. - MBBS;MD;DNB.Contribution : Manuscript writing. ORCID:0009-0002-4734-2123

2. Mahabaleshwar M. - MBBS;MD;DM. Contribution: Manuscript editing. ORCID:0000-0001-7421-2418

3. Vani Krishnamurthy - MBBS;MD;PhD. Contribution: Conceptualising and writing the manuscript, manuscript submission. ORCID:0000-0002-6938-3228

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Corresponding author
Vani Krishnamurthy
E-mail: vanidrsri@gmail.com