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*Case Report*

# Sickle cell anemia, Aplastic crisis with Parvovirus B19 Infection

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**Abstract**

The term sickle cell disease (SCD) describes a group of inherited red blood cell disorders. People with SCD have abnormal hemoglobin, called hemoglobin S or sickle hemoglobin, in their red blood cells. The red cell sickling and poor oxygen delivery can also cause organ damage. Over a lifetime, SCD can harm a person's spleen, brain, eyes, lungs, liver, heart, kidneys, penis, joints, bones,or skin. Sickle cells cannot change shape easily, so they tend to burst apart or hemolyze. Normal red blood cells live about 90 to 120 days, but sickle cells last only 10 to 20 days**.** We present a case of 17-year-old male, a known case of sickle cell disease, admitted in the medical ward with vaso-occlusive crisis. During treatment, he developed high grade fever with sweating and worsening of his condition. Blood investigations, septic screen and serology for Parvovirus B19 revealed that he was parvovirus IgM positive. He was diagnosed as a case of sickle cell disease with aplastic crisis and low reticulocyte count. He recovered after ten day of treatment.

**Keywords:** Aplastic Crisis; Parvovirus B19; Pancytopenia; Sickle Cell Anaemia.

## Background

Parvovirus B19 is a single-stranded DNA virus belongs to genus Erythrovirus and family Parvoviridae. The first human pathogen of this family was discovered by Cossart and colleagues while screening normal blood bank donors' sera for the hepatitis antigen (one of the donors' serum samples was coded B19) [1].

Human parvovirus B19 was shown to be the etiologic agent of erythema infectiosum in hematologically normal persons [2,3]. Erythema infectiosum was originally named Fifth disease because it was the fifth of six classic exanthematous diseases of childhood to be described. Parvovirus B19 has also been associated with multiple other conditions [4,5].

## Case Report

We present a case of 17-year-old male, a known case of sickle cell disease, admitted in the medical ward with vaso-occlusive crisis. He presented with fever associated with body aches and pains. He was started on the usual supportive management with basic investigations done. He was put on analgesia for pain control and was maintained on good hydration status. However, after a few days, he developed high grade fever associated with sweating and worsening of his condition. Septic screen was also done. His laboratory parameters on day 1, day 3, day 6 and day before discharge are shown in Table 1.

The reticulocyte count took about one week to normalize as shown in the complete blood count report. The serology report showed Parvovirus IgM positive. The patient was managed conservatively. He received three units of packed red blood cell (PRBC) transfusion along with antibiotic coverage, antipyretics and ensuring good hydration. The patient improved and was discharged after 10 days of admission.

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**Table 1:** Laboratory reports of the patient on day 1, day 3 ,day 6 and day before discharge showing decrease in white blood cell count (Total count, low neutrophils and lymphocytes), hemoglobin level, platelets and reticulocyte count on day 3 and /or day 6 and subsequent increase of the values on the day before discharge

| Laboratory Parameters | Normal Values | Admission Day (Day 1) | Day 3 | Day 6 | Day before discharge |
|---|---|---|---|---|---|
| White blood cells | 4 -11 x$10^9$/L | 5.72 | 1.84 | 5.07 | 6.18 |
| Neutrophils | 40-70% of WBC | 4.72 | 0.48 | 2.65 | 3.71 |
| Lymphocytes | 20-45% of WBC | 0.82 | 0.94 | 1.72 | 1.93 |
| Hemoglobin | 130-180 g/L | 116.6 | 86.5 | 102.6 (Patient received transfusion) | 84.6 |
| Platelets | 150-400 x$10^9$/L | 189 | 110 | 128 | 148 |
| Reticulocyte count | 0.8-2.0% | 4.2 | 0.8 | 0.3 | 6.1 |

## Discussion

Aplastic crisis is rare complication in sickle patients. The aplastic crisis is temporary cessation of red cell production. Because of the markedly shortened red cell survival time in patients with sickle cell disease, a precipitous drop in hemoglobin occurs in the absence of adequate reticulocytosis.

Parvovirus B19 does not infect megakaryocytes; however, *in vitro*, parvovirus B19 proteins have a cytotoxic effect on megakaryocytes. Although, the infection may manifest with pancytopenia, it is not believed to contribute significantly as an etiology of true aplastic anemia [4,5]. The rapidly dividing erythrocyte precursors (particularly pronormoblasts and normoblasts, wherein they replicate to high titers), leads to the suppression of erythrogenesis seen during infection. No reticulocytes (immature erythrocytes) are available to replace aging or damaged erythrocytes as they are cleared by the reticuloendothelial system. Parvovirus B19 has a unique tropism for human erythroid progenitor cells. The virus requires the P blood antigen receptor (also known as globoside) to enter the cell. Rare individuals who lack the P antigen are immune to parvovirus B19 infection. The virus is cytotoxic to host cells [6,7]. Although decreases in hemoglobin levels of greater than 10g/L are rare in healthy children infected with parvovirus B19, decreases of 20-60 g/L may be observed in patients with hemoglobinopathies or hemolytic anemias. Occasionally, the virus infects leukocytes (especially neutrophils) [8].The infrequently reported fulminant thrombocytopenia associated with B19 infection may consist of two types - In one type, thrombocytopenia precedes the onset of rash due to bone marrow suppression, while the other type is probably mediated by immunologic mechanisms [9].

Among patients with sickle cell disease, severe aplastic crisis is frequently associated with parvovirus B 19 infections (childhood fifth disease). Other causes may be Epstein - Barr virus, streptococcal infection or use of hydroxyurea. The period of viremia lasts about one week. The complete blood count reveals a lower than relative normal hemoglobin and a decreased reticulocyte count. The aplasia usually persists for 5-10 days. Pancytopenia does not usually occur. The treatment is management of symptoms of anemia with simple transfusion using extended antigen-typed, leuko- depleted PRBCs. Recovery is heralded by increased peripheral red blood cells followed by reticulocytosis. In most cases erythema infectiosum requires no treatment, while some patients with B19-induced arthralgia may need symptomatic treatment (i.e., anti-inflammatory drugs). In cases of transient aplastic crisis caused by B19, prognosis is excellent once a satisfactory hemoglobin concentration is obtained by erythrocyte transfusion. Effective therapy of persistent B19 infection (pure red cell aplasia) consists of infusion of immunoglobulin (0.4 g/kg of body weight/day for 5 days or 1g/kg/day for 2 to 3 days), which is a good source of neutralizing antibodies as the majority of the adult population has been exposed to the virus. This treatment is usually ameliorative and very often curative, leading to a marked increase in reticulocyte count and corresponding rise in hemoglobin.

*Conflict of Interest* None

*Financial Disclosure* None

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