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RESEARCH ARTICLE

Concurrent Parvovirus B19 and Leptospira Infection Presenting as Pancytopenia In a 10-Year-Old Child: A Rare Pediatric Coinfection

Dr. Ashwini Ambalavanan[1], Dr. Lavanya Jeyakumar[2], Dr. Vasantha Bharathy C[3], Dr. Santhosh Kumar[4], Dr. Suenera P.V[5], Dr. Kousalya Kumar[6]
Saveetha Institute of Medical and Technical Sciences

*Corresponding Author
Dr. Vasantha Bharathy C

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Abstract: ***Background:*** Parvovirus B19 is known to cause transient aplastic crisis and pancytopenia, particularly in children with underlying haematological or immune compromise. Co-infection with Leptospira species is exceedingly rare and can complicate diagnosis and management. ***Case Report:*** We report a case of a 10-year-old girl previously healthy child presenting with seven days of intermittent fever, found to have pancytopenia(Hb- 9.3 g/dl, TLC – 930/mm3, Platelets – 60,000/mm3). Serology was positive for Parvovirus B19 infection IgM and IgM-positive leptospirosis. The child was managed with supportive care, broad-spectrum antibiotics, with full recovery. ***Conclusion:*** This case highlights the diagnostic challenge and importance of considering viral and bacterial co-infection in paediatric pancytopenia. Early identification and appropriate management can lead to a favourable outcome.

Keywords: Parvovirus B19, Leptospirosis, Pancytopenia, Co-infection, Child

INTRODUCTION

Human Parvovirus B19 (HPV B19) is a small, non-enveloped DNA virus from the *Parvoviridae* family, known to cause a spectrum of diseases ranging from erythema infectiosum to severe aplastic anaemia. The virus primarily affects erythroid progenitor cells, leading to transient suppression of erythropoiesis and, in some cases, pancytopenia.

Leptospirosis, a zoonotic bacterial infection caused by *Leptospira interrogans*, typically presents with fever, myalgia, and hepatic or renal involvement. Pancytopenia due to leptospirosis is rare, and co-infection with Parvovirus B19 has not been frequently reported in the literature. We present a case of Parvovirus B19-induced pancytopenia with concomitant leptospirosis in a previously healthy child, emphasizing the diagnostic complexity and therapeutic approach. No prior admissions, surgeries, or chronic illnesses. Normal antenatal, natal, and postnatal history. Immunization up-to-date as per NIS.

RESULTS:

A 10year old female child with no significant past medical history presented with history of **fever for seven days**, moderate grade, intermittent, without chills or rigors. There were **1–2 episodes of vomiting**, no respiratory, neurological, or abdominal complaints. Child was taken to local clinic, treated with iv antibiotics injection ceftriaxone for 3 days and investigations revealed reduced total counts with thrombocytopenia.

On examination, child was conscious, oriented and thin built, Vitals were stable. Cardiac examination revealed regular rhythm and no murmur. Respiratory system examination was normal. Per abdomen examination revealed hepatomegaly and splenomegaly. Multiple insignificant bilateral cervical and inguinal nodes present. Cranial nerve examination was normal and sensation was intact.

Repeat investigations showed pancytopenia (Hb-9.3, TLC-930, Platelet – 60,000), crp was positive with mild elevation of liver enzymes. Child was shifted to PICU for further management on day 1 of hospital admission. In view of persistent fever, elevated procalcitonin and elevated D-Dimer, pancytopenia, antibiotics were upgraded to inj. Piperacillin on day 2 of hospital admission along with iv fluids and other supportive medications. Other investigations showed elevated LDH, decreased fibrinogen, lipid profile – normal. leptospirosis IgM(IgM ELISA) and Parvovirus IgM was positive. serology done was negative.

Serial complete hemogram done showed increasing trend in platelet count and total count. Dengue, widal and other viral markers were negative. Peripheral smear showed normocytic normochromic to microcytic hypochromic anaemia, leucopenia and thrombocytopenia and no blast cells. Antibiotics were continued and child was haemodynamically stable. The child gradually improved with normalization of temperature, appetite, and activity. Blood counts stabilized, and no further fever spikes occurred.

Parameters	Day 1	Day 2	Day 3	Day 4
Hb	9.3	9.1	9.0	9.1
PCV	29.3	28.9	28.8	29.3
PLATELET	60,000	94,000	1.38	1.81
TLC	930	2120	2300	3200
ANC	550	1080	970	1260
CRP	75.3	53.2		
OTHERS :	**DAY 1**			**DAY 6**
D- DIMER	3879			304
PROCALCITONIN	3.02			0.22

DISCUSSION

The presentation of pancytopenia in a previously healthy 10-year-old child, subsequently found to have concurrent infections with Parvovirus B19 (B19) and Leptospira constitutes a rare and diagnostically challenging coinfection scenario. The differential diagnosis considered include dengue, HLH, Leukemia. The discussion below explores the possible pathophysiologic mechanisms, diagnostic considerations, and therapeutic implications, anchored in the available literature.

Parvovirus B19: marrow suppression beyond erythroid lineage

Parvovirus B19 is well known to infect erythroid progenitor cells via the P antigen receptor, causing their lysis and leading classically to pure red-cell aplasia or a transient aplastic crisis, especially in individuals with underlying haemolytic disorders (1). However, there is increasing recognition that B19 may also trigger more generalized haematopoietic suppression, extending to the myeloid and megakaryocytic lineages, thereby producing pancytopenia even in immunocompetent children.

In one report, a child with hereditary spherocytosis developed transient pancytopenia secondary to B19; in that case, B19-containing serum inhibited not only erythroid colony growth but also myeloid and megakaryocyte colonies (2). In a Korean paediatric cohort, B19 infection was associated with pancytopenia in ~4.2 % of cases (1/24) (3). More broadly, a retrospective PCR study found B19 DNA in 8.3 % of children evaluated for pancytopenia; though these patients had other haematologic disorders, the finding supports B19 as a contributor to pancytopenia (4).

Mechanistically, besides direct cytotoxicity to erythroid precursors, B19 may impair other lineages via indirect mechanisms: cytokine-mediated suppression of stromal support, immune-mediated destruction, or marrow stromal damage (5). In the present patient, the detection of B19 (by whatever method was used) supports the plausibility of B19 as causative or contributory to the pancytopenia, particularly in the absence of a known haematologic disorder. The fact that B19 alone—even in an otherwise healthy child—has been documented to cause transient pancytopenia supports our hypothesis (6).

Leptospira infection and haematologic involvement

Leptospirosis is a zoonotic spirochetal infection with a spectrum from mild febrile illness to severe multi-organ involvement (Weil's disease). While thrombocytopenia is relatively common in leptospirosis, pancytopenia is rarely documented.

A review and case-report series described two adult patients with leptospirosis who presented with pancytopenia and bone-marrow aplasia, which reversed after antibiotic therapy (7). Another paediatric case described secondary haemophagocytic lymphohistiocytosis (HLH) triggered by Leptospira in a 13-year-old girl, presenting with cytopenias and marrow haemophagocytosis (8). The spectrum of marrow involvement may thus include direct spirochetal effects, haemophagocytosis, endotoxin/cytokine-driven suppression, or bone-marrow infiltration. In our patient, the positive leptospiral serology or molecular confirmation suggests that this infection may have added to marrow suppression, either alone or synergistically with B19.

Pancytopenia in children typically prompts evaluation for aplastic anaemia, acute leukaemia, bone-marrow infiltration, HLH, and severe systemic infections. Failure to consider zoonotic infections (like Leptospira) or viral agents (like B19) may delay diagnosis. The presence of co-infection further complicates the picture given overlapping clinical features (fever, hepatosplenomegaly, cytopenias). Thus, in regions endemic for leptospirosis (including parts of India), and with children who may have B19 exposure, the threshold for serologic/molecular screening should perhaps be lowered.

Prompt recognition of both infections allowed targeted antimicrobial therapy (for Leptospira) and careful monitoring/support for B19 (with transfusion support as needed). Both agents are potentially reversible in terms of marrow suppression when managed appropriately.

CONCLUSION

This case of concurrent Parvovirus B19 and Leptospira infection presenting with pancytopenia in a previously healthy child highlights a rare but important diagnostic combination. It underscores the need for clinicians to maintain a broad differential in pancytopenia, particularly in endemic regions, and illustrates that dual marrow-suppressing infections may act additively or synergistically. Recognition and appropriate management of both infections can result in excellent outcomes, as was the case here.

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