

Case Reports

Detection of Parvovirus B19 in a Case of Erythema Infectiosum with Myositis

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A well documented case of erythema infectiosum is being reported here for the first time from India which was associated with myositis that has not been reported globally. A 9-year-old child presented with moderate to high grade fever, mild anemia, and erythematous rash involving face, trunks and limbs associated with arthralgia, myalgia and myositis. Parvovirus B19 infection was confirmed by detection of IgM antibodies (in-house ELISA) and DNA (nested-PCR) in patient's serum.

Key words: *Erythema Infectiosum, Myositis, Parvovirus B19.*

A common characteristic childhood exanthem first described in 1889, erythema infectiosum was termed fifth disease (following measles, scarlet fever, rubella, and Dukes disease, which is no longer considered a distinct disease)(1).

Erythema infectiosum (E.I.) is caused by

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Manuscript received: August 25, 2005;

Initial review completed: December 5, 2005;

Revision accepted: February 20, 2006.

human parvovirus B19 and has been reported from various countries like England(2), Scotland(3), Japan(4), USA(5), etc. but, to our knowledge, not from India. However, globally, no case of E.I. associated with myositis has been reported, besides most of these reports were based on detection of B19 specific IgM antibodies only. We report here for the first time a case of E.I. with myositis, the causative agent of which was determined by detection of B19 specific IgM antibodies by ELISA and DNA by nested-PCR in patient's sera.

Case Report

A 9-year-old female presented on 11 September 2004 with moderate to high grade intermittent fever for 6 days, associated with erythematous maculopapular non-pruritic rash, myalgia and arthralgias. Erythematous maculopapular rashes were present over the entire body excluding palms and soles. The rash first appeared on face within 12 hrs of onset of fever later spreading to trunks and limbs. After 7-10 days there were central clearing in the rash giving a lacy or reticular appearance especially on cheeks which looked like "slapped-cheek". The rash persisted for about 3 weeks. She felt severe pain in calves which were so severe that the child was unable to walk. Pain worsened with movements and it persisted for 2 weeks. On examination there was slight swelling, induration and tenderness over the calf muscles. There was also a history of 3 episodes of vomiting and 1 episode of epistaxis during her illness. However, there was no history of sore throat, pharyngitis or any drug intake.

There was slight pallor; however, no icterus, edema or lymphadenopathy was

present. No abnormalities were detected in cardiovascular and central nervous system. No motor weakness was found however there was hepatomegaly of 3 cm and mild splenomegaly.

On 13th September, her hemogram showed hemoglobin of 9.3 g/dL, total leukocyte count of $8.2 \times 10^3/\mu\text{L}$ with 60% polymorphs, 38% lymphocytes, 1% monocytes, 1% eosinophils and no immature cells while ESR was 46 mm/h and platelet count was $155 \times 10^3/\mu\text{L}$. Her coagulation profile showed APTT of 26.5s (Control-31.8s) and PT of 12.2s (control-12.1s). Her serum LDH was 1342 IU/L, serum CPK 1790 IU/L, SGOT 39 IU/L and SGPT 49 IU/L. Her blood smear was negative for malarial parasite and blood culture was sterile. WIDAL and ASO titers were insignificant. CRP was within normal range (11.90 mg/dL). RF and antinuclear antibodies were negative.

On 21st September, her hemogram showed hemoglobin of 9.3g/dL, TLC was $9.9 \times 10^3/\mu\text{L}$ with 63% polymorphs, 31% lymphocytes, 1% monocytes, 5% eosinophils and no immature cells. Her LDH was 802 IU/L and CPK was 1080 IU/L.

Additionally, on 13th September, serum was collected for IgM ELISA for EBV and found to be negative and the diagnosis of viral exanthem with myositis was made. As the symptoms and signs were suggestive of erythema infectiosum, we took the consent of the parents of the patient for further evaluation and detection of the causative agent. IgM antibodies by ELISA was detected against B19 (in-house) using VP1 and VP2 as antigen as described previously (6). Then the detection of B19 DNA was done by nested-PCR briefly, DNA was extracted from serum by QIA amp ultrasense kit (Qiagen, Germany). Nested-PCR was performed as described previously (6) except for using different set of primers

from VP1 unique region(7). One microliter product of the first step PCR was taken for the second step PCR and the sample having amplicons with 853 bp were regarded to have B19 DNA. Commercial IgM ELISA kits were used for Epstein-Barr virus (Human, Germany), dengue (Panbio, Australia), rubella (Pathozyme, Scotland) and measles (Nova Tec) and were found to be negative.

During her stay in the hospital she was on ceftriaxone injections for 8 days and was also given a course of empirical antimalarials but symptoms did not resolve. Child was discharged on 27th September in stable condition on paracetamol and vitamin B complex. Till the report was written, the child was well and healthy, no recurrence had occurred. However, her CPK and LDH levels returned to normal a month after discharge on follow up.

Discussion

B19 is associated with a large spectrum of clinical manifestations(8-10). B19, the causative agent of E.I., is highly endemic in India with seroprevalence of 39.9% but these infections are usually ignored due to lack of awareness. Though the disease is self limiting but various complications with B19 infection like arthritis(11), pure red cell aplasia (PRCA)(6), pure amegakaryocytic thrombocytopenia(10), transfusion transmitted infection(12) make the diagnosis important. B19 infection should be considered in the differential diagnosis of patients with any kind of rash fever illness in children, E.I. is one of them. E.I. has a worldwide distribution, with school outbreaks in late winter and early spring. It affects primarily the 4-10-year age group. E.I. is characterized by confluent erythematous, edematous patches or plaques on the cheeks, with sparing of the nasal bridge and periorbital regions. The rash spreads to the trunk and extensor extremities, which undergo

patchy clearing resulting in a lacy reticular pattern. Occasionally, mild prodromal symptoms precede the rash; these include low-grade fever, headache, pharyngitis, malaise, myalgias, nausea, diarrhea, and joint pain.

At the time of hospital admission, bacterial infection was suspected in the patient and she received early treatment with antibiotics, but the symptoms did not resolve, moreover the blood culture was sterile, WIDAL and ASO titers were insignificant and also there was no evidence of any autoimmune disorder (negative ANA and RF) so the diagnosis of viral exanthem was considered. Further investigations revealed the causative agent of the disease. IgM ELISA for measles, rubella, dengue, EBV and B19 was done and the serum was found to be positive for B19 specific IgM antibodies, moreover B19 DNA was also detected by nested-PCR. Severe myalgia, edema and tenderness over calf muscles were suggestive of myositis. Serum CPK and LDH level were tested twice at the interval of 8 days and found to be highly elevated on both occasions. EMG and muscle biopsy, the gold standard tests for the diagnosis of myositis, could not be done due to refusal by patient. On the basis of these clinical features and investigations finally the diagnosis of erythema infectiosum with myositis was made.

Various complications due to B19(8) such as arthralgia, limb weakness(9) encephalitis, brachial plexus neuropathy, ocular neuropathy, and recurrent paresthesias have been reported. but there was no report on myositis following E.I. In just one report only weakness of arms muscles without any neurological deficit has been reported(9). Thus we document a case of erythema infectiosum due to B19 associated with myositis of lower limb. Further studies needed to look for its association with myositis.

Contributors: JK: conceptualisation, design of research, data interpretation, supervision of laboratory, final manuscript; JS: Clinical data collection, review of literature, draft of the manuscript.

Funding: Partially funded by a research grant of Sanjay Gandhi Post Graduate Institute of Medical Sciences.

Competing interests: Nil.

REFERENCES

1. Mancinci, A. Exanthems in childhood: An update. *Pediatr Ann* 1998; 27: 163-170.
2. Anderson MJ, Lewis E, Kidd IM, Hall SM, Cohen BJ. An outbreak of erythema infectiosum associated with human parvovirus infection. *J Hyg* 1984; 93: 85-93.
3. Boyce BF, Forgie I. An outbreak of 'slapped cheek' parvovirus infection (erythema infectiosum) in a Scottish street. *Scott Med J* 1987; 32: 70-71.
4. Okabe N, Koboyashi S, Tatsuzawa O, Mortimer PP. Detection of antibodies to human parvovirus in erythema infectiosum (fifth disease). *Arch Dis Child* 1984; 59:1016-1019.
5. Dym H. Erythema infectiosum—the fifth disease: case report. *J Oral Maxillofac Surg* 1990; 48: 418-419.
6. Kishore J, Mukhopadhyay C. Persistence of Parvovirus B19 IgM antibodies and DNA in pure red cell aplasia resulting in myelodysplasia: A case report. *Indian J Pathol Microbiol* 2004; 47: 78-81
7. Hemauer A, von Poblitzki A, Gigler A, Cassinotti P, Siegl G, Wolf H, *et al.* Sequence variability among different parvo-virus B19 isolates. *J Gen Virol* 1996; 77: 1781-1785
8. Kishore J, Kapoor A. Erythrovirus B19 infection in humans: *Indian J Med Res* 2000; 112:149-164.
9. Faden H, Gary GW, Korman M. Numbness and tingling of fingers associated with parvovirus B19 infection. *J Infec Dis* 1990, 161: 354-355.
10. Kishore J, Misra R, Gupta D, Ayyagari A.

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- Raised IgM antibodies to parvovirus B19 in juvenile rheumatoid arthritis. *Indian J Med Res* 1998; 107:15-18.
11. Bhattacharyya J, Kumar R, Tyagi S, Kishore J, Mahapatra M, Choudhry VP. Human parvovirus B19-induced acquired pure amegakaryocytic thrombocytopenia. *Br J Hematol* 2005; 128: 128-129.
12. Srivastava M, Kishore J, Choudhary N. Transfusion transmitted parvovirus B19 infection in multitransfused thalassemia major patients. *Clin Lab* (In press).

Isolated Left Lung Aplasia with Bronchial Asthma

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Congenital lung anomalies are categorised as pulmonary agenesis, aplasia and hypoplasia with distinct clinical implications. An 8-year-old boy was referred for an "opaque left hemithorax" for which he had received antituberculous therapy. A detailed evaluation including flowing contrast computed tomography of the thorax and fiberoptic bronchoscopy led to a diagnosis of left lung aplasia. He also had wheezing dyspnea, which was confirmed as bronchial asthma. Congenital lung defects with associated asthma was reported only twice till date. A high index of suspicion is required to recognise such a patient.

Key words: *Bronchial asthma, Congenital lung anomalies, Lung aplasia.*

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*Manuscript received: October 23, 2005;
Initial review completed: December 27, 2005;
Revision accepted: February 20, 2006.*

Congenital malformations of the lung are rare disorders occurring with variable degree of severity. These are the result of insult to the developing embryo during the fourth and fifth weeks of intrauterine life(1). Boyden clearly categorised these congenital anomalies as pulmonary agenesis, aplasia and hypoplasia(2). This categorisation is widely accepted as each condition has distinct and important clinical implications. The clinical presentation being variable, diagnostic errors often occur.

Although congenital lung anomalies were sporadically documented from the sub-continent(3-7), the occurrence of asthma in such patients is extremely rare. This association was reported only twice before, both of whom were adults when documented with pulmonary agenesis and associated asthma(7,8). The paucity of such a report in children in the literature prompted this description of an 8-year-old boy with pulmonary aplasia who also had asthma.

Case Report

An 8-year-old boy was referred to our institute for evaluation of a left-sided "opaque hemithorax". Since early childhood, he had experienced paroxysmal wheezing dyspnea along with dry cough, which had aggravated during change of season. However, there were no associated nasal symptoms. He was the