

Coxsackie encephalitis in a child in Western India

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ABSTRACT

Enteroviral encephalitis in children has been rarely described from Western India. We describe a 5½-year-old child with Coxsackie encephalitis.

Keywords: Coxsackie, encephalitis, enteroviruses, myocarditis

Introduction

Enterovirus infections are common in both children and adults and range from benign short-lived febrile illnesses to life-threatening infections.^[1] Neurological manifestations of Group A and B Coxsackieviruses include aseptic meningitis, encephalitis, paralysis (Coxsackie A7), Guillain–Barre syndrome, transverse myelitis, cerebellar ataxia, peripheral neuritis but are rare.^[2] Enteroviral encephalitis has been reported recently from North and South of India but not from Western India.^[2–4] We report a child with encephalitis, in whom Coxsackievirus infection was confirmed by determining IgM levels.

Case Report

A 5½-year-old girl was hospitalized with fever for 2 days, nonprojectile vomiting for 1 day, and one episode of generalized clonic convulsion without postictal drowsiness. The child had a vesicular rash on both hands and feet 2 months back that subsided on its own in 2–3 days leaving hypopigmented macules over the affected area. There was no focal neurological deficit, altered sensorium, and milestones were normal. Child was immunized as per age.

On examination, she had tachycardia (heart rate = 110/min), hypotension (blood pressure of 94/70 mmHg, systolic blood pressure <5th centile) with normal perfusion, and normal respiratory rate. On general examination, she had tiny hypopigmented macules over the dorsum of both hands and on feet. There were no meningeal signs. She had a mild hepatomegaly. Other systemic examination was normal. Investigations showed hemoglobin of 12 g%, white cell count 13,700/cumm (85% neutrophils and 15% lymphocytes), platelet count 130,000/cumm, and erythrocyte sedimentation rate 10 mm at the end of 1 h. Cerebral spinal fluid (CSF) analysis showed protein of 83 mg/dl, sugar of 79 mg/dl with corresponding blood sugar of 129 mg/dl, and cells of 4 lymphocytes/hpf. CSF bacterial culture did not grow any organism and smear for acid-fast *Bacillus* was negative. Her liver transaminases were elevated (serum glutamic oxaloacetic transaminase = 102 IU/L, serum glutamic pyruvic transaminase = 78 IU/L). Her serum creatinine was elevated (1.5 mg%), and electrolytes (serum sodium = 138 mEq/L and serum potassium = 3.9 mEq/L) and blood urea nitrogen (15 mg%) were normal. Prothrombin time (19.8 s) and partial thromboplastin time (39.3 s) and serum ammonia (123 IU/L) were elevated, and blood sugar was low (38 mg%). In view of tachycardia with hypotension, she was suspected to have myocarditis and her creatine phosphokinase (CPK) was elevated (2066 mg/dL) and CPK-MB was also elevated (13 ng/ml, normal = 5.8 ng/ml). Echocardiography was normal. Her Leptospira IgM, dengue

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IgM, and OptiMAL test for malaria were negative. In view of encephalitis, hepatitis, myocarditis, myositis, and coagulopathy with thrombocytopenia and a previous rash, she was suspected to have Coxsackie infection and her Coxsackie IgM ELISA was positive. No neuroimaging was done. Child was treated with intravenous fluids and symptomatically. She improved in next 3 days. Her platelet count, creatinine, liver enzymes, coagulation profile normalized within next 3 days.

Discussion

The Coxsackieviruses are divided into two major subgroups, labeled A and B. Human beings are only natural hosts of Coxsackieviruses. Spread is from person to person by fecal-oral and possibly oral-oral (respiratory spread) routes. Poor sanitary facilities and lower socioeconomic status facilitate the spread. Coxsackie B3 has been found to be one of the main causes of certain debilitating or life-threatening diseases, such as viral myocarditis.^[5] The common organs to be affected as central nervous system (CNS), heart, and skin. It is also called as hand-foot-mouth disease due to classical rash involving the hands, feet, and oral regions. In our patient too, the child had a rash that involved the hands and feet 2 months before the neurological symptoms. Epidemics of Coxsackievirus have occurred in Taiwan (1998), in Bulgaria (1975), and in Malaysia (1997), in which acute neurologic disease followed by rapid clinical deterioration was observed.^[6,7] However, Coxsackie encephalitis in children in India has rarely been reported. CNS involvement occurs at the same time as of other secondary organ involvement, but sometimes may be delayed as seen in our patient whereby CNS involvement occurred 2 months after the skin manifestation.

Although cardiac involvement is more common with Coxsackie infections, our patient had elevated CPK-MB, tachycardia, and hypotension. However, echocardiography was normal and child

did not have any other clinical manifestations of myocarditis such as fatigue, heart failure, or arrhythmias.

Thus, we conclude that CNS involvement is one of the clinical presentations of Coxsackie infection and should be suspected in children with encephalitis.

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Conflicts of interest

There are no conflicts of interest.

References

1. Sawyer MH. Enterovirus infections: Diagnosis and treatment. *Semin Pediatr Infect Dis* 2002;13:40-7.
2. Sapkal GN, Bondre VP, Fulmali PV, Patil P, Gopalkrishna V, Dadhania V, *et al.* Enteroviruses in patients with acute encephalitis, Uttar Pradesh, India. *Emerg Infect Dis* 2009;15:295-8.
3. Kumar A, Shukla D, Kumar R, Idris MZ, Misra UK, Dhole TN. An epidemic of encephalitis associated with human enterovirus B in Uttar Pradesh, India, 2008. *J Clin Virol* 2011;51:142-5.
4. Lewthwaite P, Perera D, Ooi MH, Last A, Kumar R, Desai A, *et al.* Enterovirus 75 encephalitis in children, southern India. *Emerg Infect Dis* 2010;16:1780-2.
5. Liu Z, Yuan J, Yanagawa B, Qiu D, McManus BM, Yang D. Coxsackievirus-induced myocarditis: New trends in treatment. *Expert Rev Anti Infect Ther* 2005;3:641-50.
6. Centers for Disease Control and Prevention (CDC). Deaths among children during an outbreak of hand, foot, and mouth disease - Taiwan, Republic of China, April-July 1998. *MMWR Morb Mortal Wkly Rep* 1998;47:629-32.
7. Lum LC, Wong KT, Lam SK, Chua KB, Goh AY, Lim WL, *et al.* Fatal enterovirus 71 encephalomyelitis. *J Pediatr* 1998;133:795-8.