CASE REPORTS

KAWASAKI SYNDROME INDUCED FACIAL NERVE PALSY

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Abstract

Few cases have been reported about facial nerve palsy (FNP) as a manifestation or a consequence of Kawasaki disease (KD). KD when associated with FNP has more chance to develop coronary artery aneurysm. While the paralysis is usually self limited it may require 2 to 90 days to recover. Intravenous Immunoglobulin (IVIG) has been found to have favorable response in hours to few days. We report the case of an 8 months old boy with KD who developed left lower motor neuron facial nerve palsy in the second week of his illness and after appropriate treatment including IVIG. Additional dose of IVIG was necessary to treat his facial palsy. Although initial echocardiogram was normal the repeated one after the development of facial nerve palsy showed ipsilateral (left) coronary artery dilatation. This reports highlights that IVIG does not prevent the development of Bell's palsy with KD but may be necessary for the treatment. Also it emphasizes the fact that development of facial nerve palsy may be considered as an indicator for increased risk for the development of coronary artery dilatation.

Key words: Facial Nerve Paralysis, lower motor neuron, Kawasaki Disease, IVIG, Bell's palsy, Coronary artery dilatation

Introduction

In 1967, Kawasaki from Japan was the first to describe one of the febrile illnesses as a distinct disease known as acute infantile febrile mucocutaneous lymph node syndrome or Kawasaki disease (1). It took seven years more to be reported in the western literatures in 1974 (2). Neurological involvement in Kawasaki disease is not common apart from aseptic meningitis, irritability and lethargy. Less commonly encephalopathy, seizures, ataxia, cerebral infarction or myositis may occur (3). Few cases have been reported about facial nerve palsy (FNP) as a manifestation or a consequence of Kawasaki disease (1,4). Facial lower motor neuron paralysis might be due to vasculitis below the level of the facial nucleus (4). Some literature demonstrated that Kawasaki disease when associated with facial nerve palsy has more chance to develop coronary artery aneurysm (5). While the paralysis is usually self limited it may require 2 days to 3 months to recover but more favorable response in 36 hours only was reported by Bushra et al when Immunoglobulin was administered intravenously (6).

Case Report

An 8 months old boy who was ill for 8 days was admitted as Kawasaki disease based on the presence of all the clinical criteria. His Hemoglobin was 9.26 gm/dl white blood cell count was 16,100 cells/cumm and platelets count was 13,56,000 cells/cumm. His ESR was 120mm/hr and CRP was 56 mg/dl. ECG and echocardiography were reported as normal initially. Urine microscopy revealed 80 pus-cells/hpf. Urine, blood and CSF cultures were negative for any bacterial growth. He was managed with IVIG (2gm/kg) and aspirin 100mg/kg/day. In spite of ESR decreasing to 84mm/hr over 4 days he continued to have high fever and his platelets counts increased to 15,45,000 cells/cumm. On day 4 of admission he developed left lower motor neuron facial nerve paralysis with no other cranial nerve involvement or any other neurological deficit. ENT examination was normal. Brain and temporal bone Computed Tomography was normal. Repeated echocardiography revealed left coronary artery dilatation. He was given another dose of 2 gm/kg of IVIG. On the next day his fever subsided with significant clinical improvement including his facial nerve palsy. Patient was discharged from hospital in good general condition.

Discussion

Kawasaki disease is a panvasculiltis with multisystem involvement of unknown etiology that occurs throughout the world, almost exclusively in children (7). Neurological complications are well recognized in KD occurring in 1-30% of cases (8). Facial palsy was first described by Murayama in 1974 (4). Facial nerve palsy was first considered to be one of the neurological symptom and sign of Kawasaki disease in Japanese guidelines in1984 (9). The age of patients with KD and facial palsy reported are between 3 and 25 months with female predominance (10). Review of prior cases demonstrates that children with Kawasaki associated facial nerve palsy have more than twice the risk of coronary artery aneurysm (52% Vs 25%) as that of children who do not develop this neurological complication (6). Other studies also document that association of coronary artery with facial palsy could be a marker of more severe disease (10)]. In the reported case although the first echocardiography was reported normal, the repeated one after facial nerve involvement showed left coronary artery dilatation which also suggests he was suffering from severe disease. There was significant improvement regarding his FNP and his general condition when he received additional dose of immunoglobulin. Because the facial nerve palsy is likely to be caused by an inflammatory vasculitic process that affects the facial nerve, it has been postulated that it may be ameliorated by giving immunoglobulin (11). Bushra et al reported a case of facial nerve palsy in 12 weeks boy with KD who had complete resolution of facial nerve palsy within 36 hours of administration of IVIG. The precise mechanisms by which IVIG may work remain unknown. The modulation of the synthesis and release of pro-inflammatory cytokines may play a role (6). Thus we conclude that additional dose of IVIG may be necessary to treat the facial palsy in KD. Also development of facial nerve palsy may be considered as an indicator for increased risk for the development of coronary artery dilatation.

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