

# Facial Nerve Paralysis Associated with Kawasaki Disease

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*Kawasaki disease is a multisystem disorder with varying clinical expression. This is a report on one case of Kawasaki disease which during its clinical course developed facial nerve palsy and spontaneous recovery without specific treatment.*

*It is hoped that this report will serve to remind physicians of the association of facial nerve paralysis with Kawasaki disease.*

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**Key Words:** Facial palsy, Kawasaki Disease

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Kawasaki disease was first reported by Kawasaki (Kawasaki 1967) in Japan in 1967 and has been recently well described as a discrete clinical entity.

The illness is characterized by multiple organ system inflammation and diffuse arteritis (Amano et al. 1980). The mucous membrane, skin, lymph nodes, and cardiac systems are the main sites of involvement. In addition, neurological complication have also been reported in this disease (Murayama 1974; Hattori et al. 1987; Amano and Hazama 1980; Aso and Watanabe 1984; Tamai et al. 1984; Kleiman and Passo 1988; Nigro and Midulla 1988). In 1974, Murayama first reported a case of Kawasaki disease with facial palsy (Murayama 1974) and thereafter, other additional cases have been documented (Hattori et al. 1987; Aso and Watanabe 1984; Tamai et al. 1984; Kleiman and Passo 1988; Nigro and Midulla 1988).

In our report, we describe an infant who initially presented with clinical features of Kawasaki disease and then developed a peripheral facial nerve palsy.

## CASE REPORT

A 7-month-old female infant was admitted to our hospital with the following history. There was a 10 day history of fever and a 4 day history of generalized rash, conjunctivitis with swelling of the hands and feet.

Physical examination revealed an ill appearing infant with a diffuse, focally confluent, erythematous maculopapular rash covering her entire body, except her face, palms and soles. Her conjunctivae were inflamed, and she had reddish lips and a strawberry tongue. She had bilateral cervical lymphadenopathy measuring 2×2 cm on each side. The abdomen was soft but slightly distended. There was indurative edema of the hands and feet. The remainder of the physical examination was within normal limits.

The white blood cell count was initially 10000/cu mm and later on the eighth hospital day rose to 19200/cu mm with 59% polymorphonuclear leukocytes, 5% band forms, 30% lymphocytes, 3% monocytes, 1% basophils and 2% atypical lymphocytes. On the eleventh hospital day the white blood cell count fell to 12200/cu mm with 36% polymorphonuclear leukocytes, 1% band forms, 50% lymphocytes, 10% monocytes, 1% eosinophils and 2% basophils. The hemoglobin level was initially 7.8 g/dl

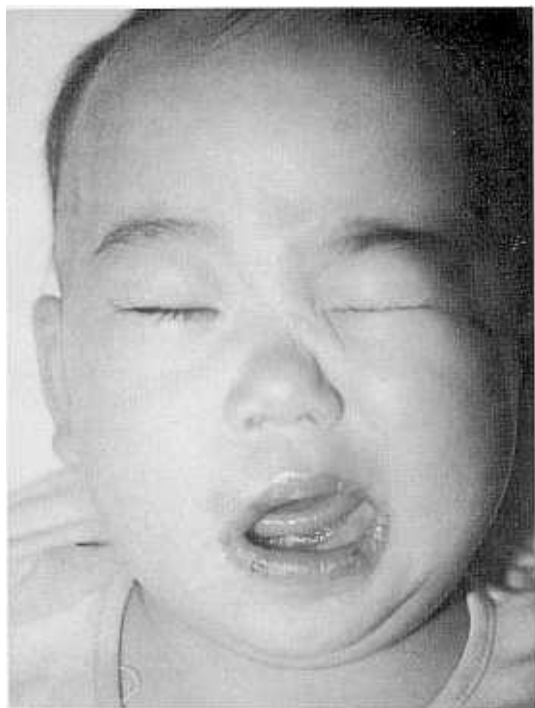
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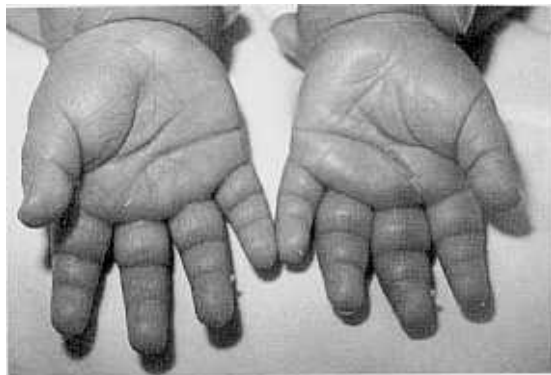
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*Fig. 1. Right peripheral facial palsy occurred on the 17th day of illness.*



*Fig. 3. Complete recovery of the facial palsy was observed approximately on the 40th day of illness.*



*Fig. 2. Desquamation on hands occurred on the 19th day of illness.*

and later on the seventh hospital day rose to 12.2 g/dl. The platelet count was initially 348000/cu mm and later on the eleventh hospital day rose to 919000/cu mm. The elevated erythrocyte sedimentation rate initially was 58 mm/hr and later on the

fourth hospital day fell to 30 mm/hr. The elevated C-reactive protein level initially was 26 mg/dl and later on the fourth hospital day fell to 8.4 mg/dl. The serum albumin level was initially 2.4 g/dl and later on the fourth hospital day rose to 3.4 g/dl. The other laboratory findings were within normal limits. Sinus tachycardia of 180 to 200 beats per minute was another finding noted on the electrocardiogram. Echocardiogram was done on hospital day eight and fifteen. There was no evidence of coronary aneurysm on either of these days.

The diagnosis of Kawasaki disease was established and treatment was initiated with aspirin (100 mg/kg/day) and high dose gammaglobulin (400 mg/kg/day) for 5 successive days. Fever disappeared on hospital day five. Thereafter, aspirin was continued at 20 mg/kg/day.

On hospital day seven, the patient presented with a strange facial expression (Fig. 1). Physical examination revealed the presence of a right peripheral facial palsy. Other neurological findings were normal. Further investigation of cerebrospinal fluid showed 37 white blood cells/cu mm with a differ-

entail of 4% polymorphonuclear leukocytes and 96% mononuclear cells. The cerebrospinal fluid protein was 58 mg/dl and the cerebrospinal fluid glucose was 49 mg/dl. Cerebrospinal fluid cultures were negative for growth. The electroencephalogram (EEG) and brain computerized tomographic (CT) scanning (both with and without contrast) were normal. Electrodiagnostic examinations (electromyography, electroneuronography) were done. The findings were suggestive of acute phase of diffuse mild incomplete peripheral type right facial neuropathy. Although the aspirin therapy was continued, no specific treatment was given. Desquamation on hands and feet occurred on hospital day nine (Fig. 2). Complete recovery of the facial palsy was noted approximately 40 days after the illness began (Fig. 3).

## DISCUSSION

Kawasaki disease is primarily an illness of infants and young children involving extensive inflammation in multiple organ systems (Amano et al. 1980; Kawasaki et al. 1974; Melish et al. 1976; Meade and Brandt 1982; Bell et al. 1983). The etiology and pathogenesis remains unknown. Criteria for the diagnosis of Kawasaki disease were those developed by the Research Committee on Mucocutaneous Lymph Node Syndrome (Kawasaki et al. 1974) and independently by Melish et al. (1976). They include: (1) fever; (2) conjunctival injection; (3) mouth changes consisting of erythematous lips, strawberry tongue, or erythema of the oropharynx; (4) acute edema and erythema of hands and feet, subacute desquamation; (5) erythematous rash; and (6) cervical lymph node enlargement of at least 1.5 cm diameter. The prognosis varies depending on the severity of cardiovascular complications (Kato et al., 1975; Onouchi et al. 1975). Approximately 15% of patients with Kawasaki disease will develop coronary arteritis severe enough to cause aneurysms, stenosis, and tortuosity of the coronary arteries (Kato et al. 1975). Aneurysm formation and thrombotic occlusions may lead to myocardial ischemia and sudden death (Fujiwama and Hamashima 1978).

It is known that Kawasaki disease causes neurological complications such as irritability, meningeal signs with pleocytosis and mild elevation of the protein content of cerebrospinal fluid (Murayama 1974; Melish et al. 1976; Meade and Brandt 1982; Mitsudome et al. 1981), aseptic meningitis (Amano and Hazama 1980), and febrile convulsion (Tamai et

al. 1984). Also seizure and encephalopathy have been described (Bernhardt 1976). However, serious neurologic manifestation of Kawasaki disease are infrequent.

Our patient initially presented with the clinical features of Kawasaki disease and then developed a right peripheral facial palsy on the 17th day of illness. Hattori et al. (1987) reported two patients with Kawasaki disease showing facial palsy and reviewed 10 case previously reported cases of facial palsy complicating Kawasaki disease. They reported the onset of facial palsy was between the 7th and 22nd clinical day of the disease (mean of 15 days). Our patient had cerebrospinal fluid pleocytosis. However, our patient had no clinical evidence of meningitis. Cerebrospinal fluid pleocytosis has been reported as frequently associated with Kawasaki disease. Terasawa et al. (1983) speculated that the etiology of facial nerve palsy in Kawasaki disease may have been ischemic effects arising from vasculitis of arteries supplying the peripheral nerves. Our case is a classic example of facial palsy in Kawasaki disease. Complete recovery without any treatment and no sequelae of the facial palsy was observed.

It is our hope that this report will serve to remind physicians of association of facial nerve paralysis with Kawasaki disease.

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