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GLIMPSE ON FACTS OF ORAL MENIFESTATION OF KAWASAKI DISEASE: AN EXPANSIVE OVERVIEW

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ABSTRACT
Kawasaki disease (KD) is also known as "mucocutaneous lymph node syndrome" which was first termed by Kawasaki. The origin of Kawasaki disease is unknown, and it is most commonly seen in children under the age of 5 years. It is characterized by disease of acute systemic vasculitis, which mainly involves medium sized arteries. Clinical features of Kawasaki disease includes fever, redness of palms and soles of the feet, oropharyngeal mucositis, rash, cervical lymphadenopathy, bilateral nonexudative conjunctivitis. Cardiac involvement is also seen in Kawasaki disease in the form of aneurysms, myocarditis and pericarditis. Clinical presentation of oral lesion involves erythema of lip and tongue, diffuse mucous membrane reddening and lingual papillae hypertrophy with subsequent development of strawberry tongue. Although it is a very rare disease and due to lack of adequate knowledge some cases might go unnoticed by dentist. Thus adequate knowledge and proper diagnosis of the disease helps in the treatment and reduce the mortality of the disease.

KEYWORDS: Kawasaki disease, Oro facial features, Oral Mucositis, Recurrence.

INTRODUCTION
Kawasaki disease was earlier described in the Japanese pediatrician Dr Tomisaku Kawasaki in 1967 which was previously termed as "acute infantile febrile mucocutaneous lymph node syndrome."[1] This disease mostly involves children who are less than 5 years of age. It is a disease affecting vasculitis of small and medium sized arteries.[2]

Etiology of Kawasaki disease includes micro-organisms such as Epstein-Barr virus, retrovirus, streptococcus, staphylococci and parvovirus. The recent etiological agent of the disease is known to be bacterial super antigen toxin.[3,4]

This immune activation may lead to subsequent development of signs and symptoms of the disease due to excessive release of endogenous immune mediators, interleukin-1, interleukin-6, interferon gamma and tumor necrosis factor leading to endothelial damage.[3,4]

The incidence and severity of formation of aneurysm significantly reduced by a single dose of immunoglobulin given intravenously.[3,4]

CLINICAL FEATURES
Symptoms which are vital for the diagnostic criteria of Kawasaki disease are:-duration of fever is more than five days that is refractory to antibiotics, involvement of oral cavity with strawberry tongue, cracking of the lips, and mucosal hyperemia, bilateral conjunctival injection, heterogeneous rash; peripheral involvement of extremities, including edema and cervical lymphadenopathy.[5]

Edematous changes are seen less and fibrous connective tissue begins to form within the vessel wall after 4 to 8 weeks followed by the symptoms.[6]

Myocardial infarction secondary to thrombosis causes cardiovascular deaths due to rupture of a large coronary

aneurysm. Proper history taking is essential in children in absence of clinical features mainly fever.[7]

Presence of clinical features decides the stage and severity of the disease.

Stage Classification of Kawasaki Disease[7]
Stage 1(0-9 days)
- Microvascular angiitis
- Acute endoarteritis and perivasculitis of main coronaries
- Myocarditis with and without specific myocardium involvement
Causes of death: heart failure and arrhythmia

Stage 2(12-25 days)
- Panvasculitis of main coronaries, with aneurysms and thrombi
- Intimal coronary proliferation
- Myocarditis, pericarditis and endocarditis
Causes of death: stage 1 plus myocardial infarction and aneurysm rupture

Stage 3(28-31 days)
- Bead-like coronaries
- Marked intimal coronary thickening
- Disappearance of microvascular angiitis

Causes of death: myocardial infarction
Stage 4 (40 days-4 years)
- Cicatrisation, stenosis, calcification and re-canalization of main coronaries
- Endocardial and myocardial fibrosis
Causes of death: myocardial infarction

FIGURES

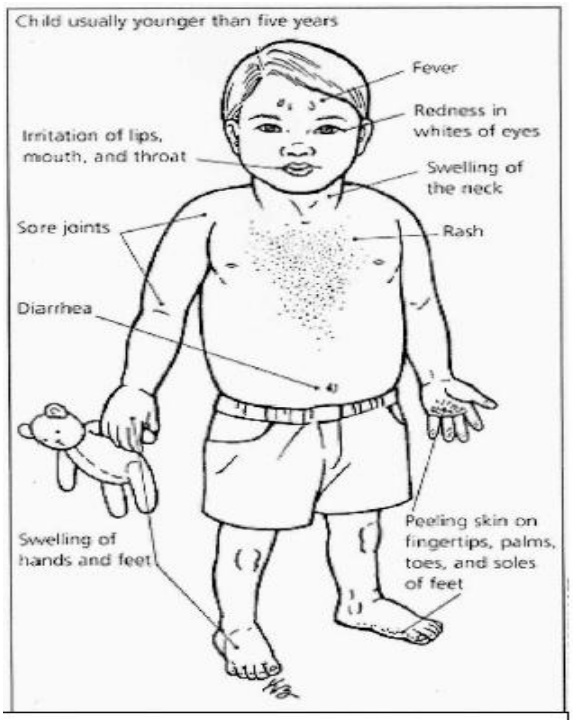

Figure 1: Clinical Guideline for families[8]

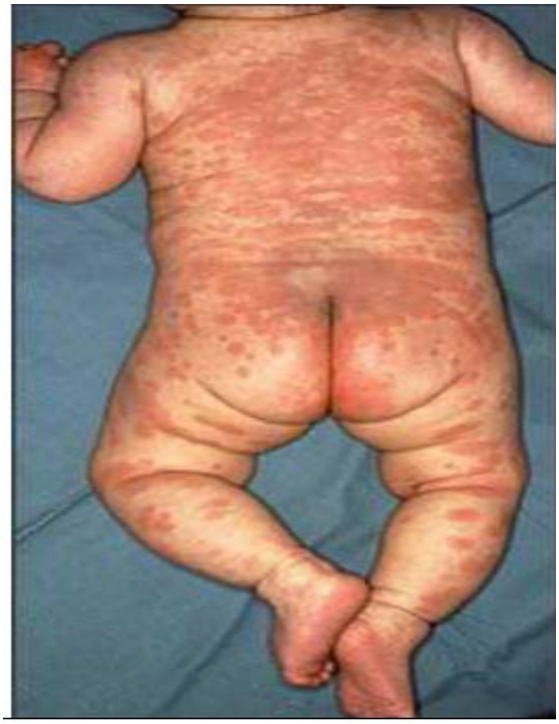

Figure 2: Infant with Kawasaki disease with an erythematous, predominantly truncal rash.[8]

Figure 3: Strawberry tongue[8]

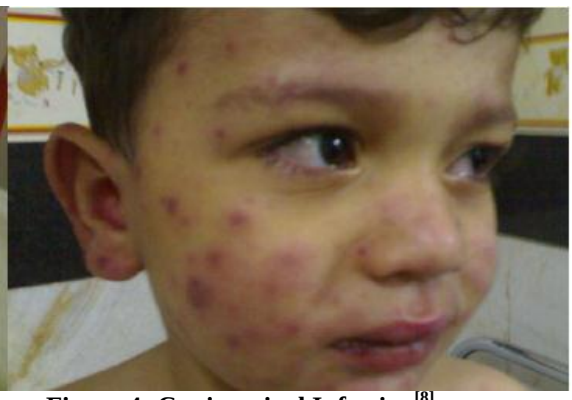

Figure 4: Conjunctival Infection[8]

Laboratory Investigations

With addition to the clinical signs and symptoms, there are non-specific laboratory findings, which include increased leucocyte count, thrombocytosis and an increased ESR. The recent studies shown that these elevated laboratory values may not be present, initially but all patients with Kawasaki Disease are likely to develop laboratory abnormalities during the course of their illness.[8]

Management

The treatment of Kawasaki disease is very standard. In Acute phase 2g/kg IV dose of immunoglobins as single effusion and 100 mg/kg dose of aspirin at in divided doses is given.[9,10] Once fever and inflammation have reduced, aspirin dose should be reduced to 3-5 mg/kg/day. Within 24 hrs most patients experience fever and by 48hrs 90% of patients are febrile. Low-dose aspirin in the sub-acute phase issued to reduction in platelet adhesiveness which further prevent cardiac sequelae.[9,10]

Pulse steroid therapy has also been shown to be effective. Only supportive management is needed in the oral manifestations of the disease as in most instances they are self-limited. Symptoms such as facial palsy may recover quite early.[11] Permanent facial palsy is seen very rarely. During the therapy to rule out cardiac involvement, patients should undergo electrocardiograms and echocardiograms.[12]

Long-term Management

Children who have Kawasaki disease without evidence of abnormalities on echocardiography appear to return to their usual state of health without any cardiac sequelae; however, some follow-up studies have demonstrated prolonged endothelial dysfunction and abnormal lipid profiles, even in children without demonstrable coronary abnormalities. Therefore, long-term surveillance studies are ongoing in this population.

About one half of the coronary artery aneurysms associated with Kawasaki disease resolve by echocardiography and angiography within one to two years, particularly those that are smaller and fusiform. Unfortunately, myointimal proliferation may lead to stenosis of the diseased coronary artery over time. Stenosis is most common in coronary arteries with giant aneurysms and occurs at the entrance to or exit from an aneurysmal area. Thrombosis leading to myocardial infarction in a stenotic or aneurysmal coronary artery is the leading cause of death in these children and occurs most often in the first year after illness onset. Therefore, serial imaging and stress tests are necessary in patients with significant coronary artery abnormalities, and cardiac catheterization with angiography is often performed to better delineate the morphology once the inflammation has resolved.

Decisions about interventions for individual patients usually should be made in consultation with a cardiac surgeon and an experienced adult interventional cardiologist. Excision of aneurysms has been unsuccessful, and deaths have resulted. Coronary artery bypass grafts, angioplasty, stents, rotational ablation, and cardiac transplantation have been performed with some success on small numbers of severely affected patients. In the current AHA guidelines, a stratification system has been developed to categorize patients by their risk of myocardial infarction and to provide guidelines for management.[14]

CONCLUSION

Kawasaki disease is an acute vasculitis of childhood that predominantly affects the coronary arteries. The etiology of Kawasaki disease remains unknown, although an infectious agent is strongly suspected based on clinical and epidemiologic features. A genetic predisposition is also likely, based on varying incidences among ethnic groups, with higher rates in Asians. Symptoms include fever, conjunctival injection, erythema of the lips and oral mucosa, rash, and cervical lymphadenopathy. Some children with Kawasaki disease develop coronary artery aneurysms or ectasia, ischemic heart disease, or sudden death. Kawasaki disease is the leading cause of acquired heart disease among children in developed countries. This article provides a summary of the history, etiology, clinical diagnosis, treatment guidelines and lifelong follow up of Kawasaki Disease. Kawasaki disease has two phases: an acute phase lasting 1 to 2 weeks, followed by a chronic ("convalescent") phase. Untreated disease usually resolves spontaneously after several weeks.

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