*IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*
*e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 20, Issue 12 Ser.8 (December. 2021), PP 48-50*
*www.iosrjournals.org*

# Diabetes Mellitus following Kawasaki Disease: Is there a link to common etiology?

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***Abstract.*** *A 4 year old child with insulin-dependent diabetes mellitus (IDDM) presented with diabetic ketoacidosis within a month of having a disease being diagnosed as Kawasaki disease (KD). Blood sugar was 511 mg/dl and HbA1C was 10.7%. His C-peptide (< 0.1 ng/ml) and glutamic acid dicarboxylase (<1 U/ml) levels were low. The child was initiated on insulin glargine and insulin lispro. He responded to the treatment. He was discharged on insulin glargine, inslin lispro and low dose aspirin (5 mg/kg/day) and kept on follow up. Occurrence of IDDM after KD is rare; we could find any association between two. The cause of diabetes in our patient remains unclear, as does the cause of KD.*

Date of Submission: 08-12-2021 Date of Acceptance: 23-12-2021

## I. Introduction:

Kawasaki disease (KD) is an acute systemic necrotising panvasculitis affecting medium-sized arteries, particularly coronary arteries. It is characterised by prolonged fever of at least five days duration and other clinical features including rash, non-purulent conjunctivitis, oropharyngeal changes, lymphadenopathy and changes to the extremities[1]. KAWA 2.Other clinical features, such as extreme irritability and re-inflammation of a recent BCG scar may aid the diagnosis[2,3]This multisystem disease is believed to be caused by a yet unidentified agent[4,5].In the vast majority of cases, insulin-dependent diabetes mellitus (IDDM) is an autoimmune disease possibly triggered by a viral infection in a genetically susceptible individual[6,7]

Although there have been limited published reports to link KD with IDDM, there is evidence to support pancreatic involvement in KD. Stoler and collaborators[8] in 1987 reported 2 cases of pancreatitis in children shortly after they developed KD. Lanting et al[9] reported a 5 1⁄2-year-old child who presented with pancreatitis 4 days before developing symptoms of KD.The association of KD with a variety of primary immunodeficiency disorders has previously been reported[10.11]. However reports of IDDM as a sequela of KD are probably rare. In this case report we are presenting a patient in whom IDDM was temporally related with the development of KD.

## II. Case Report:

A 4-years-old male child presented with increased frequency of micturition and polydipsia for 7 days. He developed pain abdomen, vomiting and fast breathing 1 day prior to admission. One month back, the child had fever for 6 days, had generalized maculopapular rash (Fig. 1A) conjunctival injection, strawberry tongue, red cracked lips, edema of hands and feet. On examination, he was lethargic, had pulse rate 110/min, respiratory rate 36/min, blood pressure 100/74 mm Hg. He had periungual and sheet like desquamation on soles and palms (Fig. 1B,C and D). Rest of the examination was unremarkable. Laboratory investigations showed hemoglobin 11.5 g/L, total leucocyte count 20.9x $10^9$/L, platelets 501x $10^9$/L, serum sodium 132 meq/L, serum potassium 3.87 meq/L, blood sugar 511 mg/dl, HbA1C 10.7%. His C-peptide (< 0.1 ng/ml) and glutamic acid dicarboxylase (<1 U/ml) levels were low. Urine examination revealed glycosuria and ketonuria. Arterial blood gas (ABG) analysis showed severe metabolic acidosis.

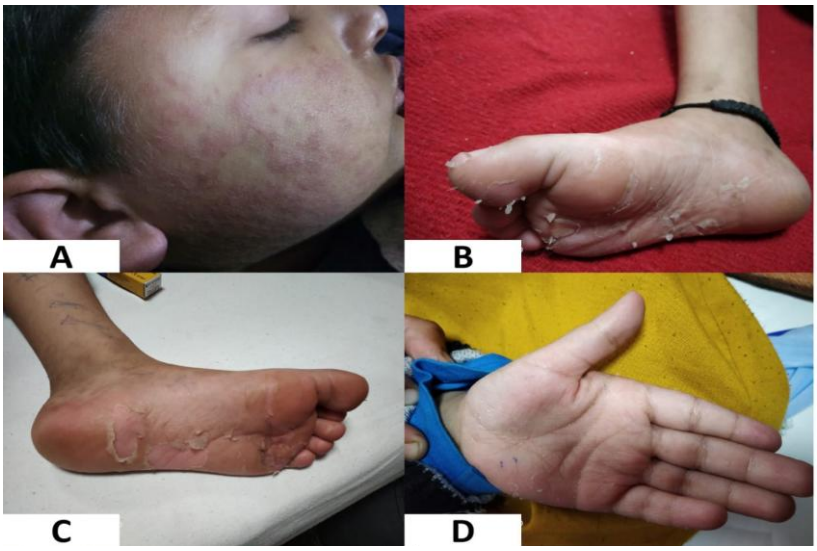


**Figure 1:** A, generalized maculopapular rash; B and C, characteristic sheet like desquamation on both soles; D, periungual and palmar desquamation.

## III. Results:

Diagnosis of type 1diabetes mellitus (DM) with diabetic ketoacidosis with history of KD was considered. The child was initiated on Milwaukee protocol for first 24 hours followed by regular insulin for next 4 days. After 4 days, the child was initiated on insulin glargine and insulin lispro. He responded to the treatment. Two-dimensional echocardiography for coronary arteries was normal. He was discharged on insulin glargine, inslin lispro and low dose aspirin (5 mg/kg/day) and kept on follow up.

## IV. Discussion

KD is an acute, multisystem inflammatory disease that mainly affects infants and young children. Clinical and epidemiologic data suggest an infectious cause of KD, but the cause and pathogenesis of the illness still remain unclear[4,5]. Cardiovascular pathology of KD has been well described[12] but much less information exists regarding pathologic changes in nonvascular tissues[13]. Patient in this report fulfilled the diagnostic criteria of KD.Within a month of probable diagnosis of KD, patient presented with DKA. Patient did not go through a honeymoon phase. Immune markers for type I diabetes were negative with almost undetectable levels of C-peptide in patient at the time of DKA. The cause of diabetes in this patient remains obscure. It is highly unlikely that pancreatitis caused diabetes in this patient. In a study of 14 patients, Rowley et al[13] observed and demonstrated pancreatic infiltration of IgA and plasma cells in periductular distribution. Five of the 14 patients showed extension of IgA and plasma cell infiltration into the pancreatic parenchyma, but the islet cells were not affected. Therefore, this process, where cells are spared, is also unlikely an etiologic factor in the pathogenesis of diabetes. IDDM has been linked to infection with "diabetogenic viruses," which may trigger an autoimmune process. It may also destroy cells by direct cytotoxic and cytopathic effects[6]. It is presumed that KD may be caused by an infectious agent[4,5] and it is also possible that the same agent that caused KD may have a direct cytotoxic and cytopathic effect on pancreatic cells in genetically susceptible individuals, resulting in complete destruction of those cells within a short span. Our patients did not go through a honeymoon phase, which is usually common in this age group with autoimmune diabetes, further suggesting a rapid total destruction of cells. Our patient required a progressive increase of insulin dose to control hyperglycemia. Therefore, the process may not be immune-mediated.

## V. Conclusion:

The index child had KD and later developed DM. In addition to being coincidental, it is certainly possible that, the co-occurrence could be due to same infectious trigger or KD may be an implicating factor to DM because of pancreatic beta cells involvement due to vasculitis. Additional studies are needed to determine whether an association exists between KD and the development of IDDM.

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Dr Naveen kumar, et. al. "Diabetes Mellitus following Kawasaki Disease: Is there a link to common etiology?." *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, 20(12), 2021, pp. 48-50.