CT Angiogram showed upper periaortic soft tissue surrounding the origin of the coeliac axis and superior mesenteric artery (SMA) causing marked stenosis of the SMA.

A working diagnosis of an inflammatory or infective Aortitis involving the proximal intra-abdominal Aorta, extending from the diaphragm to the renal vessels was made.

An extensive infectious work up was performed which proved negative including exclusion of tuberculosis.

C3 and IgA were marginally raised. Serum Anti Nuclear Antibodies, Anti-Double Stranded DNA Antibodies, Rheumatoid factor and urinary catecholamines were all within normal limits.

PET scan confirmed uptake in the proximal abdominal aorta with associated periaortic soft tissue suggestive of Aortitis.

A diagnosis of Takayasu arteritis- large vessel granulomatous vasculitis, was made.

Treatment was instigated with high dose intravenous methylprednisolone for three days followed by high dose oral prednisolone and subcutaneous methotrexate at a dose of 15 mg/m² weekly.

Inflammatory markers slowly began to normalise with immunosuppressive treatment. Follow up ultrasound at one month showed interval improvement in the aortic mass with increase in the aortic lumen size. On corticosteroid wean a month showed interval improvement in the aortic mass with immunosuppressive treatment. Follow up ultrasound at one week showed interval improvement in the aortic mass with increase in the aortic lumen size. On corticosteroid wean a month showed interval improvement in the aortic mass with immunosuppressive treatment. Follow up ultrasound at one week showed interval improvement in the aortic mass with increase in the aortic lumen size. On corticosteroid wean a month showed interval improvement in the aortic mass with immunosuppressive treatment. Follow up ultrasound at one week showed interval improvement in the aortic mass with increase in the aortic lumen size. 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after birth from respiratory failure secondary to the narrow chest cavity and hypoplastic lungs. It was learned that she had applied to the hospital with the complaint of absence of urine output nine years ago. She had been diagnosed with atypical hemolytic uremic syndrome. ADAMTS activity could not be studied at that time. Fresh frozen plasma (FFP) infusion was applied and hemodialysis was performed three times. Then the patient was improved. However, the patient did not come for follow-up.

The laboratory findings of the patient in the latest emergency presentation were: urea: 211 mg/dl, cre: 8.56 mg/dl, LDH: 5388 U/L, PLT: 103×10^9/μl, and coagulation values were normal. Direct coombs test was negative, and peripheral blood smear revealed schistocytes and fragmented erythrocytes.

The patient was diagnosed with TTP due to low ADAMTS 13 activity (<0.2%). Plasmapheresis and dialysis treatment was performed and also started pulse prednisolone treatment. The platelet count increased to over 150,000 on the 5th day and urine output improved on the 10th day. The patient was discharged with an oral steroid therapy.

Conclusion In this case report, we want to emphasize that severe AKI may rarely occur in TTP patients in childhood. We should evaluate the ADAMTS level of each patient with AKI and hemolytic anemia and regulate the treatment.