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Atypical kawasaki disease in a 10-month-old infant

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Typical clinical signs of Kawasaki disease are a fever lasting more than 5 days, conjunctiva, lymphadenopathy, skin rash, changes in extremities, and mucosal changes (1). In some cases, patients do not meet the classic criteria for Kawasaki disease and are then considered to have an incomplete (atypical) course. According to a recent Australian study, this occurs in 9.6 % of cases (2). The atypical course of the disease occurs more often in infants younger than 6 months and older children. It is suspected based on a fever lasting at least 5 days and only two or three main clinical criteria. Therefore, it is important to suspect Kawasaki disease and prescribe an echocardiogram in all children younger than 6 months of age with a fever of unknown etiology lasting at least 7 days with laboratory signs of systemic inflammation.

We present a clinical case of a 10-month-old boy who was admitted to the infection department of Ternopil Regional Children's Hospital with fever up to 39°C, diarrhea, excitation, conjunctivitis, and a macular papular rash on the trunk, arms, and legs. Manifestations of catarrh were minor. The first symptoms were noted seven days before admission and included a fever up to 39°C, which was accompanied by sweating, rash, and diarrhea. The decrease in temperature after intake of the non-steroidal anti-inflammatory drug (NSAID) was not observed. At the time of hospitalization, the general condition of the child was of medium severity. A periodic (every 6 - 6.5 hours) increase in temperature was maintained. The skin was pale, and elements of papular rashes on the legs remained. Diffuse erythema of the oral mucosa or oropharynx was weakly expressed. The mucous membrane of the conjunctiva was hyperemic, the tongue was bright, the diffuse injection of the sclera was preserved, catarrhal manifestations were insignificant, cervical lymph nodes were sized around 1 cm in diameter during palpation, and painless. Breathing rate was 38-42/minute, and heart rate was 132-138/minute. The stomach was soft, the liver was +1,5 cm, and elastic. Defecation occurred twice a day, the stool was a yellow-green color with a liquid consistency. Leukocytosis was noted (24.5x109/L), neutrophilia (30%), increased erythrocyte sedimentation rate (ESR) up to 20 mm/h, thrombocytosis (420x109/L), anemia (hemoglobin 110 g/L, hematocrit 31%). Biochemical blood testing revealed hypoproteinaemia (46.0 g/L), albumin level was not determined, C-reactive protein (CRP) was - 162,9 mg/L, level of alanine aminotransferase (ALT) was 17,6 IU/L. In the urine analysis traces of protein were present, leukocytes 3-5 in the field of vision. Echocardiography: apical defect of the interventricular septum - 1 mm, patent foramen ovale (PFO) - 4,5 mm, coronary vessels were unchanged. X-ray of chest organs showed signs of peribronchial infiltration. Taking into account a fever of more than 5 days, conjunctiva, and macular papular rash on the trunk, arms, and legs - a

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diagnosis was made - atypical Kawasaki disease. The patient received (IVIG) intravenous immunoglobulin 2 g/kg for 12 hours, aspirin 30 mg/kg/d for 3 days, then 5 mg/kg/d for 2 months. The peculiarity of this case was the presence of an atypical course of Kawasaki disease in a 10-month-old boy then when according to literature data the atypical course of the disease occurs more often in infants less than 6 months and older children (2). It was examined whether there is a significant difference in detecting anomalies of coronary arteries in the cases of Kawasaki disease and atypical Kawasaki disease. The frequency of coronary artery dilatation is greater in atypical Kawasaki disease compared to classic Kawasaki disease (3). Based on other studies, atypical Kawasaki disease accounts for a large proportion of the total number of cases (4, 5, 6). Although this may lead to a delay in diagnosis, it has not been definitively established whether incomplete imaging is a risk factor for coronary artery anomalies (CAAs). It was noted that coronary artery dilatation was more common in patients who did not meet AHA criteria (3). Is it because coronary artery dilatation may be developing slightly earlier in incomplete cases as compared to classical KD where we have the opportunity to start treatment early? Or is it because the incomplete KD is a virulent subgroup of Kawasaki disease with more predilections for coronary arteries?

It was impossible to find published reviews corroborating this and the number of our cases is too small to demonstrate statistical significance. Studies have shown that the incidence of Incomplete Kawasaki disease is high, and the incidence of coronary artery dilatation is higher in these children (3). Therefore, clinicians should continue to maintain high levels of suspicion, even in the absence of a complete clinical picture of Kawasaki disease.

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