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The first case of pediatric inflammatory multisystem syndrome (PIMS-TS) in Poland, complicated by giant coronary artery aneurysms

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Short title: Giant coronary artery aneurysms in a patient with PIMS-TS

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On May 3rd, a 2-year-old boy in good condition, with no significant past medical history, was admitted to our hospital in the 10th day of fever (up to 40°C) after ineffective empiric oral antibiotic therapy with amoxicillin with clavulanic acid. During the first 2 days of the disease, he complained about feeling joint pain and having a mild diarrhea. No other signs or symptoms were reported. About 5 weeks earlier (at the beginning of the COVID-19 epidemic in Poland), he and his family suffered from mild upper respiratory tract infection with low grade fever and rhinosinusitis. No tests were performed at the time.

On admission, differential diagnosis did not indicate any typical cause of fever of unknown origin. COVID-19 polymerase chain reaction (PCR) nasopharyngeal swab test was negative twice; Anti-SARS-CoV-2 ELISA IgG was positive, IgM negative (Euroimmun assay).

Laboratory test results are presented in table 1. No abnormalities were found on chest X-ray and abdominal ultrasound. Transthoracic echocardiography revealed left main coronary artery (LMCA) aneurysm of 4.3 mm (5.67z), left anterior descending artery (LAD) aneurysm of 6.3 mm (13.17z) and right coronary artery (RCA) aneurysm of 5.4 mm (5.67z) (echocardiography pictures in Supplementary material, Figure S1), without any myocardial contractility disorders or pericardial effusion.

Except for prolonged fever, none of the typical Kawasaki disease symptoms were present, but because of laboratory findings and coronary arteries aneurysms (CAA), KD treatment was administered. Fever resolved after intravenous immunoglobulin (IVIG) administration (2g/kg). In addition, antiplatelet agent (ASA) and warfarin were introduced. The patient was discharged home in good condition. CAA persisted in a follow up echocardiography performed after a period of 3 months.

The presented patient was, to our knowledge, the first case of Pediatric Inflammatory Multisystem Syndrome (PIMS-TS) in Poland. At that point, except for Royal College of Pediatrics and Child Health (RCPCH) recommendations, no information on this phenomenon
was available in medical literature. Since then, several definitions of PIMS (called in USA MIS-C) were established [1-3].

Reports from various countries [1, 4, 5] have provided a growing set of data about children presenting symptoms and signs overlapping with Kawasaki Disease (KD) and toxic shock syndrome (TSS), with frequent cardiac involvement and progression to shock. Compared to this data, our patient was younger than the median age for KD-like PIMS-TS and more typical of KD. His course of disease wasn’t complicated with most commonly described cardiological presentation – myocarditis and acute heart failure, but he developed CAA – the next common PIMS complication. The size of CAA > 10 z-score was observed in minority of PIMS-TS cases [5]. His clinical symptoms didn’t correspond to the severity of CAA, but no clinical or laboratory CAA risk factors have been found so far [5]. Laboratory tests [tab.1] results shared few features with those most commonly indicated in PIMS: high markers of inflammation, anemia, lymphopenia and low sodium concentration. IL-6 concentration levels were not tested, but another biomarker related to inflammatory response – CRP and erythrocyte sedimentation rates were significantly increased.

Different from most PIMS cases, ferritin concentration and NT-proBNP were in norm.

CAA can complicate all phenotypes of PIMS-TS, not only KD-like disease. Our case supports the conclusion arising from the current data, that all children with suspicion of PIMS-TS should have cardiological evaluation. Further investigation is necessary to establish best therapeutic options for these patients, as it may be different than in KD known from before the COVID-19 pandemic [5].
References:


<table>
<thead>
<tr>
<th>Laboratory tests</th>
<th>Value</th>
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<tbody>
<tr>
<td>White blood cell count</td>
<td>17.8 K/ul [6-17]</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>9.2 g/dl [10.9-13.8]</td>
</tr>
<tr>
<td>Platelets count</td>
<td>862 K/ul [210-490]</td>
</tr>
<tr>
<td>CRP&lt;sup&gt;1&lt;/sup&gt;</td>
<td>78.12 mg/l [0-2.8]</td>
</tr>
<tr>
<td>ESR&lt;sup&gt;2&lt;/sup&gt;</td>
<td>92 mm/h [&lt; 10.00]</td>
</tr>
<tr>
<td>Sodium</td>
<td>132 mmol/l [134-143]</td>
</tr>
<tr>
<td>Albumine</td>
<td>3.1 g/dl [3.4-4.2]</td>
</tr>
<tr>
<td>Ferritin</td>
<td>96.9 ng/ml [20-200]</td>
</tr>
<tr>
<td>Troponine T</td>
<td>&lt; 3 pg/ml [0-13]</td>
</tr>
<tr>
<td>NT-proBNP&lt;sup&gt;3&lt;/sup&gt;</td>
<td>134.9 pg/ml</td>
</tr>
</tbody>
</table>

Table 1.

<sup>1</sup>CRP- C-reactive protein

<sup>2</sup>ESR - Erythrocye sedimentation rate

<sup>3</sup>NT-proBNP - N-terminal pro-brain natriuretic peptide