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The Issue of Post-COVID Syndrome in Children (PIMS-TS)

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Abstract

Paediatric intermittent multisystemic inflammatory syndrome temporarily associated with SARS- CoV-2 infection (PIMS-TS) is a systemic disease affecting children and adolescents, associated with prior infection with SARS-CoV-2 virus. Although the proportion of children among those infected during the pandemic was relatively low (approximately 1.7%) and the course of infection itself was mostly asymptomatic or mild, an increase in complications in the young, approximately 2-4 weeks after infection, has been reported. Due to the similarity of symptoms, PIMS-TS is compared to Kawasaki disease, rheumatic fever or toxic shock syndrome. Fever, rash, gastrointestinal and respiratory complaints are most commonly observed; in severe cases, toxic shock and heart failure may occur. The pathophysiological basis of the inflammatory process remains incompletely understood. A dysregulation of the immune system due to previous Covid-19 is suggested as the main cause. Despite the potentially severe course, mortality in patients remains low. Treatment is based on the administration of immunoglobulins, glucocorticosteroids and biologic drugs. This article, based on a review of the information known to date about PIMS-TS, provides a definition of the condition, epidemiological data, description of pathophysiological mechanisms, clinical manifestations, diagnosis and management of patients.

Keywords: PIMS, SARS-CoV-2, COVID-19, Inflammatory Syndrome.

Introduction

In 2019, the world first heard about the SARS-CoV-2 virus (severe acute respiratory syndrome coronavirus 2), which spread rapidly around the world as the etiological agent of COVID-19 (coronavirus disease 2019). The scale of the outbreak took the form of a pandemic in the first quarter of 2020 and was designated as such by the World Health Organization [1]. Of the more than 630 million cases confirmed by November 2022, children and adolescents accounted for only about 1.7% [2, 3], and the course of the disease in this age group was often described as asymptomatic or mild, rarely requiring hospitalization and generally with a good prognosis [4-7].

Nevertheless, the first reports of severe cases among young people were reported as early as April 2020 in the United Kingdom, followed by the United States, France, and Italy [8]. Pediatricians reported hospitalizations of children with fever and multisystem inflammation, some of whom required intensive care due to shock and multiple organ failure [9-11]. Approximately 81% of patients in serious condition tested positive for IgG against SARS-CoV-2, and 37% tested positive for viral nucleic acid. Many large-scale studies also revealed a history of infection or contact with SARS-CoV-2 2-6 weeks before the onset of the disease. In light of the above, in May 2020, the World Health Organization named the condition: multisystem inflammatory syndrome in children and adolescents temporarily related to COVID-19 (MIS-C) (1), also known as pediatric inflammatory multisystem syndrome temporally associated with SARS-CoV-2 (PIMS-TS).

The course of the disease can vary from very mild to very severe, depending on the symptoms. Fever is characteristic, occurring much more frequently than in the course of COVID-19 [12]. In addition, PIMS-TS is similar to the clinical picture of other known diseases, such as Kawasaki disease, rheumatic fever, hemophagocytic syndrome, toxic shock syndrome, and sepsis

[13,14]. The diagnosis of the syndrome is based on the following factors: positive serological tests, RT-PCR, recent infection or exposure to COVID-19, and clinical symptoms [15].

To date, more than 9,000 children worldwide have been diagnosed; mainly children aged 6-12 are affected, with the average age of patients being 9.3 years. Predisposing factors include gender (56.8% of patients are male), race (Latinos and Blacks account for 34.6% and 31.5% of patients, respectively) [16-18] and obesity [19]. However, the syndrome is less common in children of Arab and Asian descent [20-23]. The influence of specific genes on PIMS-TS specific immune pathways requires further research [23]. Despite the potentially severe course of the disease, the mortality rate remains low at around 0.8%. However, it should be remembered that the lack of timely and effective treatment significantly worsens the prognosis in patients, which indicates the need for continuous improvement of diagnostic and therapeutic methods for PIMS-TS.

Aim of the Study

The aim of the study is to present the results of research from the latest publications on PIMS-TS, and thus to discuss the potential mechanisms of development of this condition, present the clinical picture, therapeutic management, and recommended prevention.

Research Methods

The PubMed and Google Scholar databases were analyzed for articles published since the creation of these databases until January 2024, describing the issue of post-COVID syndrome in children and young people. Substantive consultations were also held with specialists in the field of pediatrics.

Pathomechanism of the Disease

PIMS-TS is a new type of manifestation of SARS-CoV-2 infection, characterised by an intensified and delayed inflammatory response in the body relative to previous COVID-19 (PMS-TS occurred on average 4-6 weeks after the peak of reported positive cases) [1,20,23-31].

Patients have elevated inflammatory parameters in their blood serum: high leukocytosis, high CRP, procalcitonin and ferritin. Most patients have elevated levels of pro-inflammatory cytokines: IL-1β, IL-6, IL-8, IL-20, IL-17, TNF-alpha and INF- gamma [25-26]. Some patients have elevated troponin and N-terminal natriuretic peptide concentrations [20-25]. Patients in the acute phase of PIMS-TS also had reduced levels of T, B and NK lymphocytes [26].

The exact pathomechanism of the disease remains unknown although hypotheses are based on post-infectious dysregulation of the immune system in patients. The role of the so-called cytokine storm, which activates the IL-1β pathway and increases the levels of pro-inflammatory cytokines, including TNF α and INF-γ, is also important [20, 25, 26].

After infection with SARS-CoV-2, the body attempts to limit the spread of the virus by activating the immune response. Cell apoptosis and T-helper cell activation are induced [1]. Potential mechanisms suggest an excessive T-cell response to the superantigen located on the surface of the SARS-CoV-2 capsid virion glycoprotein. This results in the secretion of very large amounts of pro-inflammatory cytokines [1, 23]. In addition, macrophages and monocytes are activated, producing additional amounts of cytokines. The increasing number of pro-inflammatory cytokines causes a cytokine storm in the body, which induces a very intense inflammatory response [1]. Abnormalities in the number and function of NK cells and T lymphocytes have been observed in patients with PIMS-TS. A reduced number of NK cells and dysregulation of the remaining cells are observed, which may result in excessive activity of CD8+ T lymphocytes. This leads to uncontrolled inflammation in patients. In addition, a reduced level of the CLL22 marker, an important mediator of T lymphocytes responsible for their function and migration, is found in the serum of patients, which may also contribute to the induction of uncontrolled inflammation. Patients with PIMS-TS also have abnormal B lymphocytes and reduced levels of effector and memory B lymphocytes [23, 32, 33]. In addition, the blood of patients has elevated levels of plasma cells. These are unstable and can produce antibodies directed against their own antigens [23, 34]. Some patients have IgG and IgA autoantibodies directed against cardiovascular, gastrointestinal and intraepithelial autoantigens [1,22,23,32,35,36]. Studies have shown that SARS-CoV-2 can directly attack endothelial cells, contributing to their damage, and endothelial damage can cause thrombosis. These effects can lead to organ dysfunction and symptoms of PIMS-TS.

Symptoms and diagnosis of PIMS-TS

Most children who contract COVID-19 do not show any symptoms or only mild ones. However, some develop PIMS-TS. Its symptoms vary, which is why three phenotypes of this disease are distinguished:

- shock form – similar to shock occurring in cardiovascular diseases,
- Kawasaki disease (KD)-like form,
- non-specific inflammation form – does not meet the criteria required to qualify as shock or KD-like [37].

The main symptoms of PIMS-TS in children are (in order of most common occurrence):

- fever,
- abdominal pain and gastrointestinal symptoms,
- rash and erythema,
- cardiovascular symptoms,
- conjunctivitis,
- cough, shortness of breath and sore throat [38, 39].

The diagnosis of PIMS-TS requires the following conditions to be met:

- age under 18 years,
- fever and signs of inflammation (increased CRP and neutrophils and decreased lymphocytes),
- dysfunction of at least one organ (respiratory system, circulatory system, excretory system, digestive system, nervous system or shock),
- positive test result for SARS-CoV-2 infection (PCR, serological test, antigen test) or exposure to SARS-CoV-2 within the last 4 weeks [37].

Additionally, when diagnosing PIMS-TS, the following should be ruled out:

- bacterial infection,
- sepsis,
- toxic shock syndrome (TSS),

- Ritter's disease (SSSS),
- Kawasaki disease (KD),
- viral infection,
- serum sickness,
- acute abdomen, appendicitis,
- neoplastic process [37].

During differential diagnosis, particular attention should be paid to differentiation from Kawasaki disease (KD) and toxic shock syndrome (TSS). First and foremost, in PIMS-TS, we do not find the presence of staphylococci or streptococci. In addition, patients with PIMS-TS (compared to those with KD or TSS) significantly more often present with gastrointestinal, neurological and cardiovascular disorders (occurring in approximately 50% of children with PIMS- TS).

Compared to multisystem inflammatory syndrome in adults (MIS-A), children are much more likely to experience cardiovascular symptoms, while pulmonary symptoms are less common. Children are also more prone to gastrointestinal complaints than adults with MIS-A.

These symptoms occur in children with milder forms of PIMS-TS and are often confused with appendicitis or peritonitis, with the correct diagnosis often only being made in the operating theatre [38,39]. When discussing the clinical picture of PIMS-TS, it is worth noting the cardiovascular symptoms. These include mild mitral valve dilation, aneurysm formation and endocarditis. It is assumed that mitral valve dilation is caused by excessive release of IL-6 during a cytokine storm [38]. Endocarditis occurs as a result of an acute immune response to a cytokine storm, endothelial damage and microcirculatory damage. Conduction blocks also occur occasionally [37]. Elevated NT-pro-BNP levels and a moderate increase in serum troponin indicate impaired ventricular systolic function. There are also cases of mitral valve regurgitation and pericardial effusion.

Management

The latest guidelines and research provide key information that can help optimize the therapeutic approach to PIMS-TS. Understanding the pathogenesis, effective treatments, and the impact of vaccination on reducing the risk and severity of the disease is essential for clinicians managing this condition.

In patients with PIMS-TS whose phenotype resembles Kawasaki disease, immunomodulation and management according to established guidelines for this disease are recommended. Immunoglobulin therapy (IVIG) and high-dose intravenous methylprednisolone pulse therapy are recommended for patients with PIMS-TS in shock. In contrast, in patients with a nonspecified inflammatory presentation, the administration of immunoglobulins (IVIG) or prednisolone may be considered [37,38]. In all patients with confirmed PIMS-TS treated with steroids, it is recommended to gradually reduce the dose of the drug over the next 2–6 weeks, taking into account the clinical response and laboratory test results [37]. If the response to initial treatment is insufficient, other forms of immunomodulation, such as anakinra, tocilizumab, or infliximab, should be considered.

Current guidelines recommend monitoring patients with PIMS-TS in hospital settings [40,41]. After discharge from hospital, periodic follow-up examinations are recommended to detect possible long-term complications, particularly cardiovascular ones [42,43].

There are studies showing that full (two-dose) vaccination with a recombinant mRNA vaccine against SARS-CoV-2 is associated with a reduced risk of PIMS-TS [44, 45]. It has been shown that full vaccination provided 91% protection against PIMS-TS [44]. In addition, vaccination ensured a milder manifestation of the syndrome in patients with COVID-19 [45]. Vaccination results in the body producing antibodies against the RBD protein (receptor-binding domain in the S1 subunit of the SARS-CoV-2 surface protein S) of the virion. The virus uses this protein to bind to the angiotensin-converting enzyme 2 (ACE2) receptor on host cells, which triggers the body's defensive response and the onset of COVID-19 symptoms [46]. Anti-RBD antibodies may contribute to neutralizing the SARS- CoV-2 virus by preventing it from binding to the ACE2 receptor. Elevated levels of anti-RBD antibodies have been found in unvaccinated children with severe PIMS-TS

[44, 47, 48]. It has been observed that both natural SARS-CoV-2 infection and vaccination result in the production of antibodies [44,49]. Studies demonstrate a beneficial impact of vaccination on the incidence and severity of PIMS-TS, but we do not have sufficient data on the pathogenesis of the syndrome to understand its mechanisms in more detail [44].

Conclusions

For nearly five years, the global scientific and medical communities have been continuously addressing the consequences of the COVID-19 pandemic, with PIMS-TS being one of the most significant challenges. Research on this syndrome has deepened understanding of its pathomechanism, clinical manifestations, and treatment strategies, emphasizing the importance of early diagnosis for the implementation of effective therapeutic intervention. The development of treatment strategies based on immunomodulation and experience with diseases with a similar clinical picture is an important step forward. COVID-19 vaccinations play a special role in the prevention of PIMS-TS, as they appear to reduce the incidence and severity of the syndrome. Nevertheless, further research is needed to fully understand the long-term effects of PIMS-TS and to optimize treatment methods. This will be crucial to minimizing the impact of this disease on the health of children and adolescents in the future.

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