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Kawasaki-like disease associated with COVID-19 (Kawa-COVID-19); an overview of recent evident

Paniz Pourpashang[1,2*]

[1]Department of Pediatric Nephrology, Bahrami Hospital, School of Medicine, Tehran University of Medical Sciences, Tehran, Iran

[2]Pediatric Chronic Kidney Disease Research Center, Gene, Cell and Tissue Research Institute, Tehran University of Medical Sciences, Tehran, Iran

Correspondence to:
Paniz Pourpashang, Email: paaniz.p@gmail.com

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Abstract

Kawa-COVID-19 represents a new pediatric inflammatory syndrome associated with SARS-CoV-2 infection that mimics but is distinct from classic Kawasaki disease. Kawasaki-like disease associated with COVID-19 is a serious inflammatory condition that emerged during the COVID-19 pandemic. In Kawa-COVID-19, autoantibodies specifically target endothelial cells by recognizing both constitutive and induced endothelial antigens, often due to molecular mimicry, leading to complement activation, immune-mediated cytotoxicity, and endothelial dysfunction. These processes collectively contribute to vascular inflammation, coagulopathy, and the multisystem manifestations of the disease. This description incorporates the current evidence on how autoantibodies target endothelial cells in Kawa-COVID-19, highlighting key immunopathological mechanisms underlying vascular injury. Additionally, acute kidney injury, in this disease can be prerenal due to hypoperfusion or heart failure or intrinsic renal AKI from tubulointerstitial nephritis, hemolytic uremic syndrome, immune-complex nephropathy, or causes related to Kawasaki disease shock syndrome.

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Introduction

Kawasaki-like disease associated with COVID-19, which is also named as Kawa-COVID-19 or multisystem inflammatory syndrome in children (MIS-C), is a post-infectious hyper-inflammatory condition detected in children some weeks after SARS-CoV-2 infection (1). It shares clinical features with Kawasaki disease, however this disease exhibits separate characteristics and severity (2). This disease presents with persistent fever, maculopapular rash, conjunctivitis, cracked lips, erythematous palms, and lymphadenopathy (3). In addition, gastrointestinal symptoms consisted of nausea, vomiting and abdominal pain occur in several cases, often without respiratory involvement (4). This condition is also associated with myocarditis, coronary artery abnormalities, pleural effusion, and hepatosplenomegaly (5). Prior studies detected that elevated inflammatory markers like C-reactive protein, ferritin, leukocytosis, anemia, and hypoalbuminemia are accompanied with this condition (6). This mini-review sought to review the most recent findings in Kawasaki-like disease associated with COVID-19.

Key point

Kawa-COVID-19 primarily affects children and young adults several weeks after SARS-CoV-2 infection, characterized by systemic hyperinflammation and multisystem involvement resembling but distinct from traditional Kawasaki disease. Renal pathology in Kawasaki disease encompasses a range of inflammatory, immune-mediated, and vascular lesions affecting the kidneys and urinary tract. Clinical manifestations include persistent fever, rash, conjunctivitis, mucous membrane changes like strawberry tongue, swollen lymph nodes, and signs of shock or cardiac dysfunction in severe cases.

Search strategy

For this review, we searched PubMed, Web of Science, EBSCO, Scopus, Google Scholar, Directory of Open Access Journals (DOAJ) and Embase, using different keywords like Kawa-COVID, SARS-CoV-2, Kawasaki disease, autoantibody and Kawasaki-like disease.

Overview of Kawa-COVID-19

Kawa-COVID-19 is characterized by hyperinflammation known as a cytokine storm that can lead to multi-organ dysfunction, including cardiovascular failure (7). It overlaps with features of macrophage

activation syndrome and toxic shock syndrome, which necessitates exclusion of bacterial sepsis and other infections during diagnosis (8). Unlike classic Kawasaki disease, this condition often requires intensive care, with treatments including intravenous immunoglobulin (IVIg), corticosteroids, and plasmapheresis (8,9). Though, adult cases of Kawasaki-like multisystem inflammatory syndrome linked to COVID-19 have also been reported; however, it is rare, and often carry severe complications requiring aggressive treatment (10,11).

Mechanistic impact of Kawa-COVID-19

Kawa-COVID-19, or Kawasaki-like disease associated with COVID-19, is triggered by an abnormal immune response following SARS-CoV-2 infection rather than direct viral damage (12). Its mechanistic impact involves post-infectious hyperinflammation driven by immune dysregulation, adaptive immune activation, and cytokine storm, which leads to multisystem inflammation and cardiovascular complications (13-15). Several studies found that Kawa-COVID-19 typically develops 4–6 weeks after SARS-CoV-2 infection, suggesting a delayed post-infectious process initiated by the adaptive immune response triggered by viral antigens or antibodies (16,17). This presentation is distinct from acute COVID-19 infection, where innate immunity plays a more dominant role (18). This syndrome explains by a significant cytokine storm with elevated pro-inflammatory cytokines such as interleukin 6, tumor necrosis factor, and IL-10, which address systemic inflammation and tissue damage, similar to what occurs in classic Kawasaki disease along with more severe intensity (19). In this syndrome, elevated ferritin levels, a marker for macrophage activation syndrome, are common, indicating severe immune activation (20). Likewise, autoantibodies targeting endothelial cells, mucosal tissues, and cardiac antigens have been identified, suggesting immune complexes contribute to vascular inflammation and vasculitis characteristic of Kawa-COVID-19 (21). These autoantibodies may engage Fcγ receptors on neutrophils and macrophages, triggering pro-inflammatory cytokine secretion that further tissue injury (22). Recent studies found genetically predisposed children may develop exaggerated immune responses to SARS-CoV-2, involving heightened mucosal T cell activation and Th17 responses, which amplifying inflammation through IL-17 production (23). Additionally, upregulation of Toll-like receptors like TLR-7 and oligoclonal IgA responses in vascular tissues suggest antigen-driven immune activation akin to classical Kawasaki disease (24). Finally, this syndrome exhibits multisystem involvement with predominant cardiovascular effects, sparing the lungs, contrasting with adult COVID-19 hyperinflammation, which primarily causes respiratory distress syndrome due to innate immune dysregulation and neutrophil-driven cytokine storm (25). The sustained hyperinflammatory state leads to myocarditis, pericarditis, coronary artery abnormalities, and shock (4). Moreover, the vasculitis and endothelial injury from immune complexes and cytokines cause widespread organ dysfunction (4,13). Some studies suggested that the delay between infection and symptom onset reflects immune-mediated tissue injury rather than active viral replication (26).

Focus on autoantibodies in Kawa-COVID-19

Autoantibodies generated during or after SARS-CoV-2 infection can recognize and bind to endothelial cell antigens, leading to endothelial cell activation and damage (27). This immune complex formation triggers complement activation and induces pro-inflammatory signaling, which compromises vascular integrity and promotes vasculitis characteristic of Kawa-COVID-19 (28). Certain autoantibodies, especially anti-cytokine autoantibodies, can exacerbate systemic inflammation by modulating cytokine activity, leading to an amplified cytokine storm that drives vascular inflammation (29). This hyper-inflammatory environment recruits immune cells to vascular sites, intensifying tissue injury (30). Furthermore, IgM autoantibodies against angiotensin-converting enzyme 2, as a receptor for SARS-CoV-2, have been shown to activate the complement system, which plays a direct role in endothelial perturbation and vascular inflammation (31). Complement activation also contributes to a pro-thrombotic state, promoting microclots and vascular occlusions seen in severe cases (32,33). Recent investigations demonstrated that autoantibodies arise due to molecular mimicry, where viral antigens that share epitopes with host vascular proteins, causing cross-reactive immune responses that target blood vessel components, further driving inflammation and damage (34). Eventually, the vascular inflammation mediated by autoantibodies could be due to the multisystem inflammatory syndrome, manifesting as cardiac complications like myocarditis and coronary artery aneurysms, shock, and vasculitis in Kawa-COVID-19 patients (4,35).

Endothelial cell injury in Kawa-COVID-19

As mentioned above, autoantibodies in Kawa-COVID-19 specifically target endothelial cells by recognizing and binding to endothelial cell surface antigens or proteins expressed or exposed during the inflammatory process triggered by SARS-CoV-2 infection (36). In fact, autoantibodies are directed against a heterogeneous group of endothelial cell antigens, including; 1) constitutively expressed surface proteins on endothelial cells (37). 2) Cytokine-induced or inflammation-induced neoantigens that appear on endothelial cells during vascular inflammation. These autoantigens become targets once exposed or upregulated during endothelial activation or damage in the context of COVID-19-related inflammation (38). In addition, SARS-CoV-2 infection can induce molecular mimicry, where viral proteins share structural

similarities with endothelial proteins (39). This similarity causes the immune system to generate autoantibodies that cross-react with endothelial cell components, leading to endothelial injury and dysfunction (40). Another pathway is autoantibody-mediated endothelial activation and cytotoxicity; since, upon binding to endothelial antigens, autoantibodies can activate complement pathways, leading to membrane attack complex formation and endothelial cell lysis (41). These autoantibodies also promote antibody-dependent cellular cytotoxicity involving immune cells that kill antibody-coated endothelial cells (42). Meanwhile, these autoantibodies trigger endothelial cell activation, resulting in the release of pro-inflammatory cytokines, adhesion molecule upregulation, and a pro-coagulant state (40). It should be noted that the binding of autoantibodies to endothelial cells leads to vasculitis alongside of thrombotic complications by inducing pro-thrombotic endothelial phenotypes seen in Kawa-COVID-19 patients (4,13).

Renal pathology in Kawasaki disease

In general, kidney and urinary tract involvement in Kawasaki disease contains pyuria, prerenal acute kidney injury (AKI), renal AKI caused by tubulointerstitial nephritis, hemolytic uremic syndrome (HUS), immune-complex mediated nephropathy, acute nephritic syndrome, nephrotic syndrome, renal tubular abnormalities, imaging abnormalities, and renal artery lesions (43-46). These lesions are believed to arise primarily from vasculitis, immune complex deposition, and T-cell mediated immune dysregulation (43-46). Tubulointerstitial nephritis is characterized by interstitial infiltration of mononuclear and polymorphonuclear leukocytes and is considered a common cause of acute kidney injury in this disease, linked to T-cell activation (45). Hemolytic uremic syndrome is rare but reported in this disease, possibly resulting from endothelial injuries caused by vasculitis. In patients with Kawasaki disease shock syndrome, acute renal failure often results from acute tubular necrosis secondary to multiple organ dysfunction (45, 46). Meanwhile, immune-complex deposition in glomeruli has been observed, especially in cases presenting with acute nephritic syndrome, manifesting as hematuria, proteinuria, edema, and hypertension (45-47). In fact, decreased complement levels in some acute nephritic syndrome patients suggest activation of the classical complement pathway. Several studies showed that nephrotic Syndrome is rare but documented in this condition. The underlying mechanism may involve immune complex injury or T-cell dysregulation similar to minimal change disease (14, 48). Elevated urinary markers such as lysozyme, β2-microglobulin, N-acetyl-β-D-glucosaminidase, and interleukin-6 indicate tubular inflammation or damage during the acute phase of Kawasaki disease. These abnormalities reflect inflammatory processes within the renal parenchyma, possibly related to tubulointerstitial nephritis (49). Imaging by ultrasonography and renal scintigraphy often reveals nephromegaly, increased cortical echogenicity, and inflammatory foci in kidneys of Kawasaki disease (44). Likewise, renal inflammatory foci detected by scintigraphy may lead to scarring, indicating potential long-term renal sequelae (50). Kawasaki disease vasculitis can affect renal arteries, resulting in aneurysms or stenosis, occasionally causing renovascular hypertension (51). Renal artery aneurysms are less common but have been reported alongside coronary artery aneurysms. Renal artery stenosis cases have sometimes required interventions like percutaneous transluminal renal angioplasty or surgery (52). The pathogenesis of renal involvement in this situation is not fully understood but involves systemic vasculitis affecting medium-sized arteries, immune complex deposition, and T-cell mediated inflammatory responses (44,53,54).

Conclusion

The mechanistic impact of Kawa-COVID-19 involves a post-infectious, adaptive immune-mediated hyperinflammatory syndrome triggered by SARS-CoV-2. Immune dysregulation, cytokine storm, autoantibody production, and immune complex formation lead to multisystem inflammation, primarily affecting the cardiovascular system in genetically susceptible children. This understanding informs treatment strategies focusing on immunomodulation with IVIg, corticosteroids, and other agents.

Conflicts of interest

The author declares that she has no competing interests.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the authors utilized Perplexity to refine grammar points and language style in writing. Subsequently, the authors thoroughly reviewed and edited the content as necessary, assuming full responsibility for the publication's content.

Ethical issues

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the author.

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