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Cutaneous Vasculitis Attributable to Post-COVID Syndrome

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Abstract

While the acute phase of the COVID-19 pandemic has largely come to pass, the chronic physiologic effects of the coronavirus continue to unfold. Specifically, the number of COVID-19-associated cutaneous vasculitis cases has steadily increased since the onset of the pandemic. Data have shown that vasculitis may develop less than two weeks after COVID-19 or during a later onset of the disease. At this time, research has demonstrated that the novel coronavirus invades more than just the lungs; it can also attack the nervous system, cardiovascular system, kidneys, and cutaneous vasculature. In addition, there is a greater understanding of the pathogenesis regarding COVID-19-induced vasculitis via humoral immunity and immune complex disease. Recent case reports have shown an association between COVID-19 and secondary cutaneous vasculitis. This presentation discusses case reports and data that suggest that COVID-19 may lead to specific cutaneous vasculopathies. More research needs to be performed on this association to aid in diagnosis and treatment.

Keywords: nervous system; cardiovascular system; kidneys; and cutaneous vasculature

Introduction

As of now, COVID-19, as reported by the WHO, has afflicted over 770 million individuals globally and claimed 6.95 million lives, persisting as an enduring global crisis [1]. The virus's capability to invade various organs, including the lungs, heart, and kidneys, has led to a plethora of complications. An escalating concern noted in the literature involves the direct and indirect consequences of COVID-19, as well as secondary manifestations following COVID-19 infection or vaccination, impacting the skin and blood vessels, ultimately giving rise to cutaneous leukocytoclastic vasculitis. Vasculitis, defined as inflammation of blood vessel walls, can affect vessels of various sizes, be they arteries or veins [2]. This inflammation poses a risk by potentially obstructing blood flow or promoting clot formation, which can restrict organ perfusion in the case of larger vessels or cause symptoms like palpable purpura in small cutaneous vessel occlusion [3]. Vasculitis can be triggered by autoimmune disorders, trauma, certain medications, and infections [2]. Among the different types of vasculitis, those more commonly associated with COVID-19 include Cutaneous Leukocytoclastic Vasculitis (CLCV), Kawasaki disease, and IgA vasculitis. The pathophysiology underlying CLCV appears to originate from the deposition of immune complexes formed in response to the Coronavirus within the walls of small blood vessels. Consequently, this triggers the complement cascade, leading to tissue damage, recruitment of neutrophils, and the release of pro-inflammatory cytokines such as IL-1, IL-6, and IL-8 [3]. Subsequently, fibrinoid necrosis occurs within the vessel walls, allowing red blood cells to extravasate. Confirmation of examination findings can be obtained through histological biopsy, along with immunofluorescence or light microscopy, which provides diagnostic insights based on the observed features. This diagnostic approach can also shed light on the underlying disease or triggering factor [4].

Currently, there is no well-defined treatment protocol for COVID-19-associated cutaneous vasculitis. The management approach largely depends on the severity and type of vasculitis, with options ranging from no intervention or supportive care to the use of potent immunosuppressive agents and extended courses of corticosteroids. While many cases seem to resolve spontaneously without significant long-term consequences, this literature review has uncovered case reports indicating fatalities resulting from severe vasculitis associated with COVID-19 [5,6]. These findings underscore the urgent need for a clear and effective treatment strategy, emphasizing the importance of further research to prevent such devastating outcomes. The COVID-19 pandemic has largely passed, but the virus infection rate persists with

no clear end in sight. This had led to continuous emergence of complications and emphasizes the pressing need for ongoing research to fully understand the virus's primary and secondary effects. Among these complications, the topic of cutaneous vasculitis secondary to COVID-19, while relatively rare, is an emerging sequela. This literature review aims to provide insights based on the latest case reports, case series, and systematic reviews linking COVID-19 to vasculitis. It also explores the treatment strategies employed to manage affected patients. By sharing this information, we seek to keep the medical community and physicians updated on the potential consequences they may encounter when dealing with patients who have a known history or current COVID-19 infection and vaccination.

Discussion

Literature Research (Methodology) and Study Selection

Using the PubMed database and search engine, we performed a search of the English language literature by combining the term COVID-19 with each term for specific forms of cutaneous vasculitis from January 2020 through December 2022. Supporting research and additional information surrounding studies on the topic of cutaneous vasculitis was also gathered using the PubMed database and searching for terms related to specific forms of cutaneous vasculitis.

Summary of Current Studies

Cutaneous vasculitis (CV) is an inflammatory vascular disease limited to the skin that affects the dermal and/or the hypodermal blood vessel wall. Vasculopathies found within a cutaneous distribution are a pathological process notably characterized by inflammation affecting the vessel wall, both arterial and venous, of any location on the body [7]. Following the Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) pandemic in 2020, cases of cutaneous vasculitis induced by both active Coronavirus Disease 2019 (COVID-19) infection and post-COVID syndrome have been reported throughout the literature. Cutaneous vasculitis induced by SARS-CoV-2 warrants further explanation for insights into the pathogenesis of inflammatory processes within the vasculature with post-COVID syndrome and long COVID. Preliminary research has found that patients previously diagnosed with COVID-19 experience additional clinical manifestations and cases of isolated vasculopathy [8]. The association between SARS-CoV-2 direct or indirect vasculopathy and a correlation with disease prognosis has not yet been fully discovered. It has recently been proven that COVID-19 directly causes vasculitis via a specific immune response. Notably, research has provided scientific evidence that in COVID-19 vasculitis, there appears to be a life-threatening escalation from type 2 T-helper immune response (humoral immunity) to type 3 hypersensitivity (immune complex disease) [9]. The deposition of immune complexes was seen inside vascular walls that induced a severe inflammatory state, and the immune complexes within the vasculature trigger an immune response and lead to varying types of vasculitis throughout the body.

Recent evidence has suggested cutaneous vasculitis (CV) may occur during or after the COVID-19 infection. Cutaneous small-vessel vasculitis has been the most commonly reported dermatological vasculitis in COVID-19 patients [10]. Cutaneous vasculitis, previously known as leukocytoclastic vasculitis, occurs both with COVID-19 infection, vaccination, and post-COVID syndrome [10]. The mechanism of action seen in vasculopathy on the histology of patients with severe infection appears to be endothelial injury due to immune thromboembolic mechanisms. The pathogenic mechanisms are not entirely understood; however, a hyperactive immune response, complement activation, and microvascular injury are hypothesized to contribute to cutaneous vasculitis [10]. Common cutaneous vasculitides include cutaneous small-vessel vasculitis, urticarial vasculitis, skin-limited IgA vasculitis, and lymphocytic vasculitis [11]. In a case series, Corrá et al. reported that the timeline from having COVID-19 to developing vasculitis varies, ranging from within a few days to over 30 days [12]. While recent studies have discovered associations between prior COVID-19 infection and cutaneous vasculitis and vasculopathy, further research is needed to determine the pathophysiology of these findings. The skin is noted as one of the most affected organs in vasculitides. Cutaneous vasculitis may be a cutaneous presentation of systemic vasculitis, or it may be a skin-limited variant of systemic vasculitis. When there is no systemic involvement and only the skin is affected, this is referred to as single-organ vasculitis (SOV) [12]. Inflammation within the vessel wall occurs alongside an intravascular infiltrate, which may disrupt the integrity of the vasculature and promote leakage of red blood cells. Cutaneous

vasculitides have been characterized by the 2012 Chapel Hill consensus conference nomenclature of vasculitides, dividing by diameter of the impacted vessel: Large Vessel Vasculitis and Medium Vasculitis leading to skin necrosis, ulceration, and livedo reticularis and Small Vessel Vasculitis leading to purpura and vesiculo-bullous lesions [13].

Cutaneous vasculitis is primarily a small-vessel vasculitis that affects the dermal and hypodermal capillaries and venules with histopathologic findings consistent with leukocytoclastic vasculitis[14]. Leukocytoclastic vasculitis is often characterized by erythrocyte extravasation, fibrinoid necrosis of vessel wall, and neutrophilic infiltrate with degeneration [12]. Direct immunofluorescence is helpful in the diagnosis of cutaneous vasculitis, and it aids in accurate diagnosis even if histological changes are quite minimal [15,16]. While it is impossible to assess a definite disease-promoting factor in cases of isolated cutaneous vasculitis, past research points to the well-known triggering factors of CV as immunopathogenic mechanisms secondary to viral infections [17,18]. Since the COVID-19 pandemic, cases of COVID-19-associated CV have increased steadily. Manifestations of the COVID-19 infection of the skin show a wide variety of presentation and symptoms. Specifically, cutaneous findings often present in classes of pseudo-chilblain lesions, urticarial rash, vesicular eruption, maculo-papular rash, and vaso-occlusive lesions [19]. Case reports of cutaneous vasculitis identified in post-COVID syndrome include both new onset and flares. In one case, a previously healthy 49-year-old male presented to a dermatology department with a chief complaint of skin lesions on his lower limbs. This patient had had COVID-19 a few weeks prior and developed rashes along with his cough and myalgia. Histopathologic findings of this patient revealed mild hyperkeratosis of the epidermis, moderate neutrophilic infiltration, extravasated red blood cells, and a few lymphocytes around superficial and mid dermal vessels all consistent with leukocytoclastic vasculitis [20]. The skin lesions worsened and became intensely painful and pruritic. Purpuric lesions covered the flexor and extensor surfaces of his legs, thighs, and abdomen. This patient was determined to have cutaneous leukocytoclastic vasculitis secondary to a prior COVID-19 infection.

Another case documented a previously healthy 30-year-old male who presented to the dermatology clinic with asymptomatic rashes on his leg following a two-week long COVID-19 infection. He began testing negative for COVID-19 a few days after his two-week infection but noticed the skin rashes at a later date. His physical examination showed non-blanchable erythematous papules and plaques on his thighs and legs in a linear arrangement consistent with the appearance of the Koebner phenomenon. Histopathological examination showed features of perivascular neutrophilic infiltrate and fibrinoid degeneration [21]. This patient was given a diagnosis of cutaneous small-vessel vasculitis secondary to prior COVID-19 infection. Vasculitis flares of preexisting conditions have also been documented in the literature through case reports. One review specifically discusses how COVID-19 is related to autoimmune diseases and is known to trigger endothelial dysfunction and the appearance of autoantibodies. Patients with history of COVID-19 are found to have vasculitis-like phenomena and leukocytoclastic vasculitis. This literature review of IgA vasculitis discovered two cases where patients who previously had well-controlled IgA vasculitis developed a renal and cutaneous flare of vasculitis after mild COVID-19 infections [22]. One patient even experienced new-onset ANCA vasculitis. Both patients were treated with glucocorticoids and immunosuppressants with successful response. COVID-19 was found in these case reports to not only trigger vasculitis but also to provoke flares of preexisting autoimmune conditions and diseases.

A fatal, critical case of cutaneous leukocytoclastic vasculitis secondary to COVID-19 infection has been documented in a patient who experienced extensive skin necrosis from his vasculopathy. While fatal cases of cutaneous vasculitis following COVID-19 infection are rare, this 93-year-old male survived the course of his COVID-19 infection only to develop a fulminant cutaneous leukocytoclastic vasculitis (cLcV) leading to extensive skin necrosis and tissue damage that preceded his death [6]. This patient had no other triggers for vasculitis, and his cLcV was determined to be a secondary manifestation from post-COVID syndrome. This pathophysiologic rationale was hypothesized based on SARS-CoV-2-induced endotheliitis, complement activation, and interleukin 6 dominant intra- and perivascular inflammation [6]. The first case of a generalized purpuric eruption showing typical microscopic features of leukocytoclastic vasculitis in the setting of COVID-19 was seen in a 59-year-old male. The patient experienced severe respiratory failure and was intubated following hospital admission. The patient

received empirical broad-spectrum antibiotics including cefepime, linezolid, and gentamicin while in the hospital. He had no known drug allergies. Detection of SARS-CoV-2 by reverse transcription polymerase chain reaction assay of a throat swab was positive. Skin biopsy found superficial and deep perivascular neutrophilic infiltrate with sparse leukocytoclasis, red blood cell extravasation and fibrinoid necrosis of vessel walls. This patient developed neurologic complications as well. It cannot be proven, however central nervous system vasculitis due to SARS-CoV-2 could be hypothesized. The case is an example of COVID-19 infection potentially inciting severe drug-related cutaneous leukocytoclastic vasculitis along with systemic vasculitis [23].

In a case reporting urticarial vasculitis, an elderly female presented with painful erythematous patches that resulted in residuary purpura on the hips and trunk after arriving at the emergency department for bilateral pneumonia and positive COVID-19 testing. Cutaneous biopsy displayed histologic changes consistent with small-vessel vasculitis including blood extravasation and neutrophilic perivascular inflammation with prominent karyorrhexis. Abrupt worsening of her respiratory condition contributed to the patient's death; therefore, no treatment had been initiated [24]. Another case of urticarial vasculitis was reported for a 32-year-old female with COVID-19 who presented with a uticariform rash. The rash appeared six days after the onset of her viral symptoms. Biopsy displayed perivascular infiltrate of lymphocytes, eosinophils, and oedema of the upper dermis. Oral antihistamines were implemented resulting in both clinical and symptomatic improvement within five days [25]. Another review documents 14 patients who tested positive for COVID-19 infection using PCR. They displayed skin changes a few days after viral symptoms began. Inflammatory lesions were reported in seven patients, exanthema in four patients, chicken pox-like vesicles in two patients, and cold urticaria in one [26]. Vascular lesions including violaceous macules with "porcelain-like" appearance, livedo, non-necrotic purpura, necrotic purpura, chilblain appearance with Raynaud's phenomenon, chilblain and eruptive cherry angioma were distributed in different variations among the other seven patients. While the pathophysiology was not proven, it is hypothesized to include vasculitis, immune dysregulation, vessel thrombosis or neoangiogenesis [27].

Another study reported chilblain lesions in people who had close contact to COVID-19 + patients but who did not themselves have COVID-19 PCR confirmation or general symptoms of COVID-19 infection. This led to a proposal of three hypotheses. First, it was suggested that the lesions could be from a confounding factor other than COVID-19. Second, they could also be from a post-viral immunological reaction that comes from asymptomatic forms of COVID-19. The third hypothesis was that the chilblain lesions represent a skin presentation of COVID-19 infection in a subgroup of patients with peculiar immune anti-viral response [27].

Treatments

Treatment for Covid-19-induced cutaneous vasculitis hinges on two pivotal factors: the specific subtype of vasculitis triggered and the severity of the condition. Fortunately, the literature has indicated that cutaneous vasculitis typically follows a self-resolving course, sparing individuals from enduring permanent effects [28]. In instances where spontaneous resolution does not occur, the treatment approach primarily relies on clinical judgment, considering the patient's medical history as well as their individual physiological attributes such as age and associated risk factors. Given its propensity for self-resolution, the initial consideration leans towards symptomatic treatment. This encompasses the use of analgesics, such as acetaminophen or ibuprofen, to alleviate joint pain or muscle aches. Additionally, antihistamines can reduce the transmigration of immunoglobins across blood vessels limiting further progression of the disease [28]. In cases of IgA vasculitis this becomes especially important as compression therapy and antihistamines were shown to reduce the formation of new lesions by way of inhibiting vascular dilatation [29]. In the event that the vasculitis presents as Kawasaki's disease (KD), treatment follows a more well-defined path, thanks to a wealth of available data on KD management. The foremost treatment strategy involves intravenous immunoglobulin (IVIG) and aspirin, a regimen applied even in pediatric cases. For patients with IVIG-resistant disease, a combination therapy approach can be considered, incorporating IVIG, tumor necrosis factor (TNF) inhibitors, steroids, calcineurin inhibitors, or anakinra [30].

In the instance of cutaneous vasculitis previously known as leukocytoclastic vasculitis, the majority of cases will resolve spontaneously with mild supportive measures, including leg elevation, rest, compression stockings, and antihistamines. If an episode persists so

long that it is determined as chronic or resistant, a four- to six-week tapering dose of corticosteroids can be used [3]. Rarely, immunosuppressive steroid-sparing agents such as methotrexate, azathioprine, mycophenolate mofetil, dapsone, cyclophosphamide, and intravenous immunoglobulin may be needed [3]. Lastly, it is important to note that the resolution of vasculitis associated with COVID-19 may be intricately tied to the treatment of the underlying COVID-19 infection itself. Given that COVID-19 remains a relatively novel virus with limited established treatment protocols in the literature, the management of these cases often enters the realm of experimentation guided by the attending physician's best clinical judgment. The pathophysiology of COVID-19 is comparatively better understood in the medical literature. Due to its capacity to trigger a pronounced and potentially devastating inflammatory cascade in affected patients, regardless of age, a common approach involves the utilization of anticoagulants, immunosuppressants, and anti-inflammatory agents. These therapeutic interventions are frequently employed in the treatment of severe COVID-19 cases. After a comprehensive review of numerous case reports in the current medical literature, it becomes evident that the majority of management strategies include a combination of blood thinners, corticosteroid therapy (such as methylprednisolone), intravenous immunoglobulin (IVIG), hydroxychloroquine, or other less conventional antiviral or biologic agents [4, 5, 31, 32, 33, 34, 35, 36].

Limitations

This extensive review of the current literature included studies published between January 2020 and December 2022, using keywords of COVID-19 and specific types of cutaneous vasculitis. All referenced manuscripts were available within the PubMed database. Although the information provided in this literature review included all appropriate studies in the PubMed database, the collected information was limited to one database. Additional information may be accessible in other research databases and in manuscripts published after December 2022. It is possible that some manuscripts meeting our criteria were excluded due to the use of keywords other than what was searched. The clinical presentation of cutaneous vasculitis associated with current or previous COVID-19 infection varies greatly, making it challenging to diagnose. From 2020-2022, global attention was given to SARS-CoV-2, a novel coronavirus. Information known about the virus during that time period was limited but progressively updated; thus, it is not unreasonable to assume that the associated vasculitis could have been misdiagnosed and attributed to another disease process, diagnosed as a flare of a preexisting condition, or classified as an isolated vasculopathy. This is supported by the fact that the number of COVID-19-associated cutaneous vasculitis cases steadily increased over the course of the pandemic as more information was available about the virus.

Areas for Future Research

At this time, the pathogenesis and prognosis of SARS-CoV-2-associated cutaneous vasculitis remains unclear. The condition appears to be highly variable in both the timing of symptom onset and clinical presentation making it difficult to predict who may be affected. Studies have suggested immunopathogenic mechanisms and inflammation may trigger the development of this condition, but further research is needed to identify the pathogenesis and any genetic factors, inciting events, or medications that may predispose individuals to post-COVID-19-associated cutaneous vasculitis. SARS-CoV2 has been shown to invade almost every organ system of the human body with emphasis on pulmonary involvement. Future studies may be able to identify why certain patients develop an isolated cutaneous vasculitis whereas others have widespread systemic symptoms.

Conclusion

Cutaneous vasculitis has been reported in cases of patients with post-COVID syndrome throughout the literature. Cases of cutaneous vasculitis included both self-limiting skin forms without systemic involvement and cases with regional or widespread systemic involvement. Additional research is recommended on this topic to gain a better understanding of the pathophysiology of inflammation leading to cutaneous vasculopathies and pathogenetic mechanisms contributing to the origin of vascular disease. With patients presenting with vasculopathies attributed to post-COVID syndrome and long COVID, it remains imperative to carefully monitor, recognize, and diagnose patients with early signs of cutaneous vasculitis.

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