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VASCULITIS (L ESPINOZA, SECTION EDITOR)

Vasculitides in HIV Infection

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Abstract

Purpose of Review To review the spectrum of vasculitides in HIV-infected patients and to identify the clinical features that characterize vasculitis in sero-positive HIV.

Recent Findings Epidemiological studies conducted in the post-HAART era described the rarity of vasculitis in the setting of HIV-infected patients. A study identified histopathological features such as leukocytoclastic vasculitis of the vasa vasorum and adventitial inflammation in the large artery pathology of HIV-positive patients compared with HIV-negative patients with critical lower limb ischemia. A recent retrospective cohort study reported that HIV-positive patients with LVV developed more vascular complications, responded less to antiretroviral therapy, and had worse outcome than HIV-negative patients with LVV.

Summary Vasculitides continue to be a rare disease in patients with HIV. The spectrum of vasculitis ranges from life-threatening conditions to relatively mild skin conditions. Recognizing vasculitis in the setting of HIV-positive patients is important because sometimes it require immunosuppressive treatment.

Keywords HIV infection · Vasculitides · Polyarteritis nodosa · Kawasaki-like syndrome · Large-vessel vasculitis

Introduction

At the time of writing, the world grapples with the pandemic of COVID-19, each day passing continues to increase the number of lives lost and the human suffering. Nearly 40 years have elapsed since the occurrence of HIV pandemic which caused high mortality among affected population [1]. An analogous situation such as stigmas, diagnostic testing, and treatment almost similar to the beginning of the HIV pandemic is the one we are seeing in this new global medical emergency due to the pandemic caused by infection with SARS-CoV-2 coronavirus (COVID-19) [2]. The immunodeficiency status of patients with HIV/AIDS was accompanied by the occurrence of inflammatory and autoimmune diseases and is an issue that needs to be kept in mind and that their development still is an enigma.

This article is part of the Topical Collection on *Vasculitis*

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Epidemiology

Reliable studies on the prevalence of HIV-associated vasculitis are scarce because there are few high-quality, descriptive, longitudinal, and cohort studies. There are prevalence data from studies that have been conducted in hospitals, but this is not a true representation of what occurs in the general population.

Vasculitis has been uncommonly associated with HIV infection, and the literature have reported a frequency < 1% [3].

Contrary to these data, Zhang et al. in a study conducted in Asian patients infected with HIV/AIDS showed higher prevalence of vasculitis in 20 cases of vasculitis (20.41%) in 98 inpatients with HIV/AIDS, including Behçet-like disease (n = 15), Henoch-Schonlein purpura (n = 2), digital gangrene (n = 2), and central nervous vasculitis (n = 1). This raises the question of whether ethnicity plays an important role in the development of prevalent rheumatic manifestation [3, 4•]. Yao et al. have not reported cases of vasculitis in a retrospective record review of 888 inpatients with HIV/AIDS [5]. Yen et al. and Lebrun et al. conducted prospective longitudinal cohort study

during highly active antiretroviral therapy (HAART) era, and both did not show development of vasculitis cases [6•, 7•].

J. Reveille (personal communication, May 24, 2020) reported a retrospective chart review of HIV-infected patients evaluated at an outpatient rheumatology clinic from 1994 to 2019.The frequency of vasculitides was 0.9%. The spectra of vasculitides were as follows: systemic vasculitis/polyarteritis nodosa ($n = 3$), granulomatosis with polyangiitis ($n = 2$), primary angiitis of the CNS ($n = 2$), MPO-associated vasculitis ($n = 1$), IgA vasculitis ($n = 1$), and HCV-associated cutaneous vasculitis ($n = 1$).

In the last 10 years of the post-HAART era, there has been an absence of new cases of vasculitis and this is probably due to a more effective antiretroviral treatment [4•].

Mechanisms to Induce Vasculitis in HIV Infection

The pathogenic mechanisms of these diseases are not completely understood; however, there are proposed mechanisms that may cause vascular damage: (1) direct action, vascular wall injury by HIV replication or opportunistic microorganism; (2) indirect action, formation and depot of immune complexes (molecular mimicry); (3) the aberrant immune activation which characterizes the HIV infection could act indirectly in the development or maintenance of the vascular inflammatory reaction [8, 9]; and (4) other mechanism is immune restoration inflammatory syndrome during HAART that suggests that immunopathology results from restoration of protective pathogen-specific cellular immune response [3, 10] (see Fig. 1).

Clinical Manifestations

The clinical manifestations of patients with HIV vasculitis are wide and depend of the size of the compromised blood vessel and can occur at any stage of HIV infection; therefore, the spectrum and severity of vasculitides range from life-threatening disease to relatively minor skin disease.

Small Vessel Vasculitis

The common denominator of these affectations is usually cutaneous involvement presenting as palpable purpura and less frequently extracutaneous involvement, which is less severe compared with other vasculitis forms.

Hypersensitivity Vasculitis

The reported cases are most often induced by antiretroviral drugs and less post infectious [11]. Because HIV/AIDS patients take a combination of antiretrovirals, often determining the cause of a cutaneous eruption can be difficult.

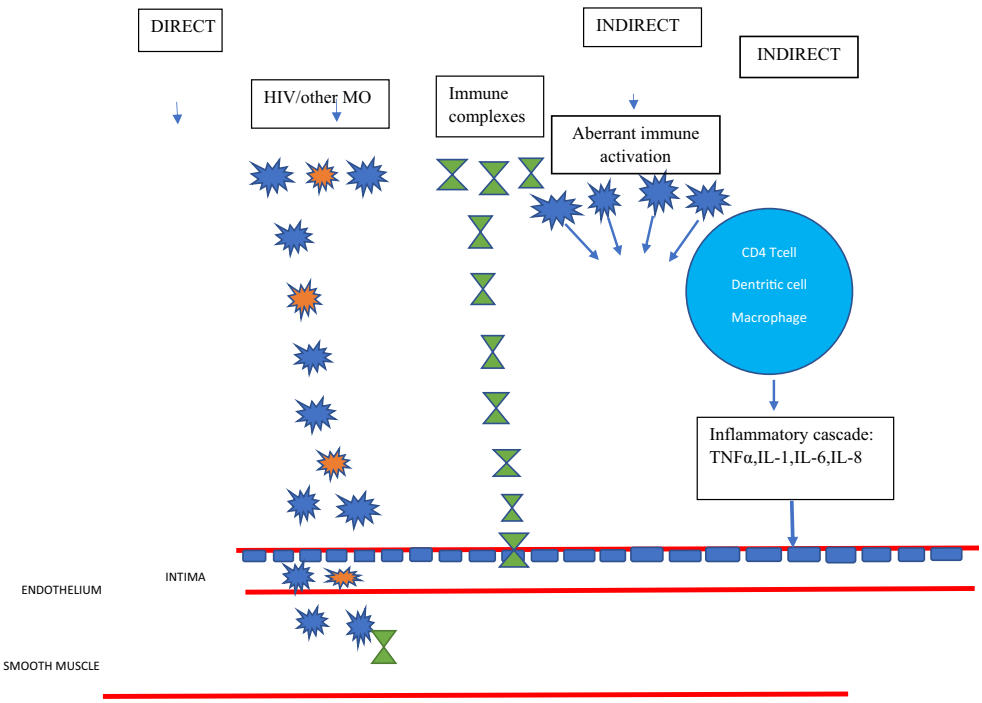

Fig. 1 Mechanisms to induce HIV infection

Erythema Elevatum Diutinum

A rare form of leukocytoclastic vasculitis of the skin is EED associated with progressive fibrosis. HIV disease is likely to predispose to this disorder. Many published literature report associations between CD4 and EED. CD4 count of less than 300 cells/μl. to be a risk factor for EED. The lesions initially are soft, non-fixed red/violaceous, or brown papules and nodules that later become firm because of fibrosis [12–15]. Patient responded well to dapsone treatment [15].

Mixed Cryoglobulinemia

HIV infection is a recognized cause of mixed cryoglobulinemia. Previous studies have reported the prevalence of cryoglobulins varying from 17 to 27% [16]. MC is frequently associated with infectious agents such as CMV, EBV, and hepatitis C virus. Clinical manifestations are like HIV-negative individuals, but many times, vasculitis purpura is not seen, and the clinical picture is predominantly neurologic [17].

Vasculitis of the Central Nervous System

Vasculitis of the CNS is an uncommon disease. In the setting of HIV/AIDS, CNS vasculitis may be primary or are associated with concomitant infection by opportunistic or no-opportunistic microorganisms. Primary vasculitis of the CNS is a diagnosis of exclusion [18]. Melica et al. reported cases of primary vasculitis of CNS, in African patients suggesting ethnic predisposition. The patients showed persistently low CD4 cell counts, and two of these patients had been receiving HAART and had partial or good control of viral replication, suggesting persistent immune activation [19]. Benjamin et al. conducted a study of adult ischemic stroke. They made comparison between HIV-infected and non-infected HIV patients and found that HIV-associated vasculitis was one of the etiologies. Patients were younger, at late stage of HIV/AIDS, and have fewer risk factors for stroke. Most cases started on HAART suggestive of immune reconstitution inflammatory syndrome [20]. It has been described coinfection with CMV, VZV, toxoplasmosis [21, 22].

Medium-Sized Vessel

Polyarteritis Nodosa

A form similar has been described in patients infected with HIV and named polyarteritis nodosa-like disease (PAN-LD). The classic PAN is associated with hepatitis B virus and is characterized by inflammation and necrosis predominantly medium-sized vessels, typically involving renal and other visceral arteries while sparing the pulmonary circulation, while HIV-associated PAN compromises smaller and/or medium-sized vessels of the skin, muscle, nerves, and gastrointestinal tract with rare involvement of the renal arteries [23, 24]. PAN can occur at any stage of HIV/AIDS disease and at any count of CD4. The clinical course of HIV-related PAN exhibits major differences in comparison with classic HBV-related PAN (see Table 1). Presence of anti-neutrophil cytoplasmic antibodies, especially pANCA, is high (13–42%), but its clinical significance is not well defined.

Kawasaki-Like Syndrome

Classic Kawasaki (KD) disease is a multisystem disorder, affects most frequently children, and is rarely seen in adults [25, 26]. This disease commonly compromise coronary arteries and can lead to various complications like coronary artery aneurysm, thrombosis, stenosis and even sudden.

KLS occurs in association with moderate to severe immunodepression, low CD4 count, and high viral load [27•]. It is probably that KD and KLS are the same disease because they share inflammatory signature such as soluble tumor necrosis factor receptor (sTNFRII) and chemokines Cdl1, Cdl2, and Cxcl11 [28]. Compared with KD is not reported coronary aneurysm [29] (see Table 2). Also KLS has been described after the introduction of HAART, as a potential form of IRIS. The therapy is the same with classic KD [30].

Large-Vessel Vasculitis

The spectrum of large-vessel disease associated vasculitis consists of aneurysms and occlusive disease [31]. This disorder can occur in the absence of known risk factors such as

Table 1 Differences between HIV-PAN and classic PAN

	HIV-associated PAN	Classic PAN
Virus associated	None	HBV
Involvement	Rare multisystem	Multisystemic
Common symptoms	Peripheral neuropathy, rash	Fever, livedo reticularis, cardiac, gastrointestinal, and renal
Clinical course	Usually no life-threatening	Progressive o fulminant deterioration

Table 2 Comparison of clinical symptoms and laboratory abnormalities in Kawasaki disease of HIV-positive and HIV-negative adults and children

	HIV+	HIV-	Children
Conjunctivitis	+++++	+++++	+++++
Pharyngitis	+++	++++	++++
Cheilitis	+++	+++	++++
Stomatitis	+++	++++	+++++
Desquamation	+++++	+++++	+++++
Adenopathy	+++++	+++++	+++++
Arthralgia	++	++	++
Gastrointestinal	+++	++	++
Electrocardiogram abnormalities	+	+	+++
Coronary aneurysms	-	-/+	+++
Leukocytosis	+	++	
Thrombocytosis	+	+++	+++++
Risk of relapse	++	-/+	-/+

arterial hypertension, diabetes mellitus, and atherosclerosis. There is also evidence to suggest that HIV infection promotes the development of accelerated atherosclerosis due to endothelial damage occurring in a much younger group of patients and could be associated to vasculopathy [31].

LVV is uncommon. The patients more affected tend to be young, in advanced stages of diseases, and present more severe vascular complications such as aneurysms and occlusive disease [31]. The patients were predominantly black African. The aneurysms or occlusions are usually multiple [31, 32•]. A particularity is that the aneurysms are in atypical locations such as carotid, subclavian, femoral, and popliteal vessels [31, 32•]. The main histopathologic finding is leukocytoclastic vasculitis of the vasa vasorum and of periadventitial vessels [33•]. Other histopathologic findings will depend on the stage of disease (see Table 3). Clinical and histopathological characteristics overlap or are reminiscent with those seen in Takayasu arteritis (TA) [33•, 34, 35••]. Vasculitis resulting from IRIS is extremely rare, unless an associated opportunistic infection is present.

Robbs performed a study in 226 patients admitted with HIV vasculopathy and reported vascular complication such as aneurysms in 111 patients and occlusive disease in 115 ones. Patients were predominantly man, younger, and black African. The CD4 count was low (range 1–930 cells/mm^3) [36].

Ferfar et al. performed a retrospective cohort study with 93 patients affected of LVV. Eleven patients (12%) also had HIV infection [35••]. Patients were middle-aged adults. Most patients fulfilled Ishikawa or ACR criteria of TA diagnosis. Histopathologic examinations showed changes like TA. These patients were compared with their HIV-negative counterparts. Patients with LVV-HIV developed more vascular complications, 6 patients with aneurysms, and 5 with vascular occlusion, and there was significantly greater use of vascular procedures. They also indicated that corticosteroids and immunosuppressive therapy were less often prescribed in HIV-positive patients and had worse outcome than non-HIV patients with LVV.

The vasculitis may occur in all ranges of CD4, independent of viral load, and in patients after starting antiviral therapy suggesting an immune reconstitution inflammatory syndrome (IRIS) [35••, 36, 37]. A further observation was the lower use of conventional and biological DMARD in patients with HIV-associated vasculitis.

Other Vasculitis

Behcet's Disease The rare cases occurred in the setting of an acute HIV infection characterized by a high viral load, and these symptoms decreased following initiation of effective antiretroviral therapy and a rapid decreased of virologic load. Researchers have postulated that induction of Behcet's disease in HIV-associated cases might be a direct effect of viral replication or through HIV induction of autoimmune mechanisms or immune dysregulation [38, 39].

Therapy

1. Controlling the HIV Infection

HAART forms an essential part of the therapy to prevent disease progression. The early use of currently available antiretroviral drug (HAART) could avoid the catastrophic

Table 3 Histopathologic findings in LVV-associated vasculitis

Intima	Media	Adventitia
Duplication and fragmentation of the internal elastic lamina	Fragmentation of the elastic tissue	Marked leukocytoclastic vasculitis of the vasa vasorum and periadventitial vessels
	Medial necrosis	Proliferation of slit-like vascular channels
	Plasmolymphocitary and giant cell infiltrate	Chronic inflammation
	Fibrosis	Fibrosis

complications of large-vessel vasculitis such as aneurysms and occlusion.

2. Treating the Vasculitis

Deciding how to treat the HIV-associated vasculitis is not easy, because the pathogenesis is not completely understood.

HIV infection has significantly impacted the natural history and therapeutic intervention of autoimmune diseases due to the presence of underlying immunosuppression and the use of immunosuppressive drugs or biologic agents that may lead to serious complications including infections.

2.1. Management of Vasculitis Secondary to Drugs and Infectious Agents

The resolution occurs with discontinuation of the offending agent and treatment with oral corticosteroid in the first case and administration of antibiotics in the second case.

2.2. Management of Severe Vasculitis Diseases

The array of therapeutic agents is similar in HIV-positive and HIV-negative patients [40–42]. Patients afflicted with HIV-associated vasculitides may respond well to conventional therapy such as corticosteroids and DMARDs including methotrexate, leflunomide, mofetil mycophenolate, azathioprine, and cyclophosphamide, and for refractory cases may need the use of biological agents such as rituximab, tocilizumab. When considering immune suppressive therapy, it is important to keep in mind that CD4+ T cells are necessary in the control of intracellular and extracellular bacteria, parasites, and viruses. This therapy should be recommended when patients have CD4+ T cell counts above 200 cells/mm^3 and HIV viral activity completely suppressed to < 60,000 copies/mm^3 [43, 44]. In addition, they have been shown to be effective, safe, and well-tolerated [44].

Currently, however, there are no studies of good quality on the use of biologic therapy to vasculitis disorders in HIV-infected patients; therefore, we cannot conclude on efficacy and safety of biologic therapies in HIV-infected populations.

As part of the proposed measures to improve the management of rheumatic patients in times of COVID-19, it is not advisable to discontinue immunosuppressive treatment as it may be responsible for onset of clinical flare of rheumatic disease [45].

3. Managing the Aneurysms and Occlusive Disease

These complications are considered emergencies and therefore should be prioritized regardless of the immune status. Treatment should be individualized and prioritized to patients with symptomatic aneurysmal lesion and acute arterial occlusion [31, 32•, 36, 37]. Interventional or surgical therapy is indicated if a vascular lesion that persists despite medical therapy is either symptomatic or is associated with an increased risk of future complications.

The therapeutic decisions will always require the participation of a cardiovascular surgeon. Management of HIV-associated vasculitis requires a multi-disciplinary approach where intervening rheumatologist, vascular surgeon, and rehabilitation specialists.

Prophylaxis Consensus suggests that prophylaxis against opportunistic infection should be given to all HIV patients on immunosuppressive therapy due to an increase risk of infection.

Conclusions

Vasculitides is a group of rare diseases that affect large, medium, and small vessels. It is a clinically important complication that we must recognize because it requires immunosuppressive treatment. Interventional or surgical therapy will be reserved for patients with complicated large-vessel vasculitis.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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