DOI: 10.7860/JCDR/2026/80049.23261

Case Report

Internal Medicine Section

Acute Hepatitis A Virus-induced Cutaneous Necrotising Vasculitis: A Case Report with Literature Review

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ABSTRACT

Cutaneous Necrotising Vasculitis (CNV), a rare dermatological condition, is characterised by inflammation and necrosis of small blood vessels, often presenting as palpable purpura. While viral aetiologies have been identified, Hepatitis A Virus (HAV)-induced CNV is seldom reported. A 41-year-old male presented with palpable papular rash involving both upper and lower limbs, abdomen and back region over the past 15 days. The rash was associated with high-grade fever, diarrhoea and pain in both knee and ankle joints. He had a recent history of consuming outside food and drinks. Total leukocyte count, C-Reactive Protein (CRP) levels, total anti-HAV antibody titres, and anti-HAV IgM were elevated. Liver function test was slightly deranged with raised alkaline phosphatase (104 U/L), aspartate aminotransferase (34 U/L) and alanine aminotransferase (77 U/L) and normal serum bilirubin levels. Skin biopsy demonstrated necrotising vasculitis with fibrinoid necrosis of the vessel wall. The patient was treated with systemic steroids, antihistaminic agents and topical lotion. On four weeks follow-up, rash disappeared spontaneously. Thus, viral aetiologies should be considered in patients presenting with cutaneous vasculitis to facilitate timely diagnosis and management.

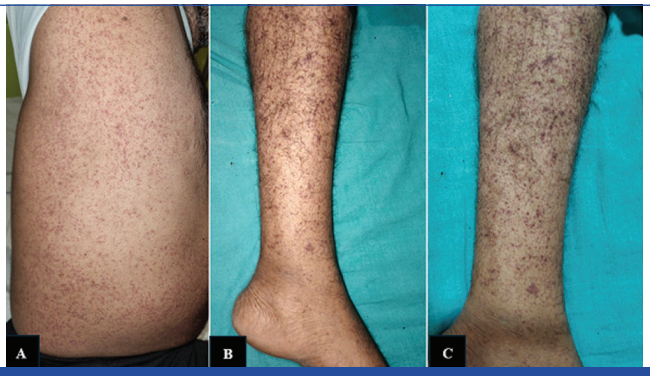
Keywords: Cutaneous vasculitis, Fibrinoid necrosis, Systemic steroids

CASE REPORT

A 41-year-old male presented with the complaint of reddish purple papular rash involving both upper and lower limbs and back region over the past 15 days. The rash was gradual in onset, progressive in nature, and associated with high-grade fever (39°C) over the last 15 days. The patient had complaints of loose motion for the last 15 days, which was watery in consistency, followed by decreased stool frequency over the past seven days. He complained of pain in both knee and ankle joints. He had a recent history of consuming outside food and drinks following which he developed loose motion. He was not on any medication and had no history of recent travel, chronic liver disease, high-risk sexual behaviour, or illicit drug abuse.

On examination, vital parameters were normal. The patient had extensive palpable purpura involving both lower limbs and abdomen [Table/Fig-1a-c] and marked swelling of the knees filling the suprapatellar pouch. He was oriented, with no signs of meningitis, encephalopathy, or endocarditis. Routine laboratory tests showed raised total leucocyte count (14700/mm^3) and slightly decreased haemoglobin (12.1 gm%), while other parameters on complete blood count were normal. Peripheral smear and routine urine examination were normal. The liver function test was deranged with slightly raised levels of Alkaline Phosphatase (ALP, 104 U/L), Aspartate Aminotransferase (AST, 34 U/L) and Alanine Aminotransferase (ALT, 77 U/L) with normal total serum bilirubin (0.5 mg/dL). Rheumatoid factor levels were normal, while levels of C-Reactive Protein (CRP, 32.45 mg/L) and Lactate Dehydrogenase (LDH, 241 U/L) were raised together with raised Erythrocyte Sedimentation Rate (ESR, 55 mm/hr).

The antinuclear antibody and serum IgA titres were normal, with no evidence of IgA paraprotein. Blood was negative for cryoglobulins, thereby ruling out cryoglobulinaemic vasculitis. As the association of skin purpura and cytolytic hepatitis was strongly suggestive of a viral aetiology, tests for emergent viruses were performed. The panel for anti-HAV total antibody (5.25 S/CO) and anti-HAV IgM (4.52 S/CO) was raised, while the panel for human immunodeficiency virus I/II, Hepatitis C Virus (HCV), Hepatitis B surface Antigen (HBsAg) and Hepatitis E Virus (HEV) were negative. Moreover, stool routine microscopy was normal and stool culture did not demonstrate growth of organisms [Table/Fig-2]. Thus, the diagnosis of HAV infection was reached.



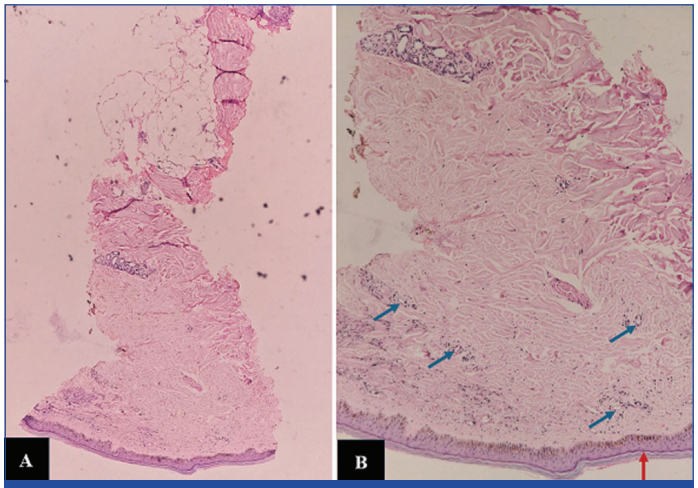
[Table/Fig-1]: Reddish purple papular rash involving abdomen and back (A) as well as both lower limbs (B and C).

Tests	Values
Total WBC count	14700/mmcu
Total RBC count	3.97 lac/mmcu
Total platelet count	5.91 lac/mmcu
Haemoglobin	11 gm%
Total serum bilirubin	0.5 mg/dL
Alkaline phosphatase	104 U/L
Aspartate aminotransferase	34 U/L
Alanine aminotransferase	77 U/L
Total protein	6.1 g/dL
Serum urea	26 mg/dL
Serum creatinine	0.8 mg/dL
Serum sodium	136 mEq/L
Serum potassium	4.8 mEq/L
Lactate Dehydrogenase (LDH)	241 U/L
ESR	55 mm/hr
CRP	32.447 mg/L

RA factor	8.6
Urinary Protein Creatinine Ratio (UPCR)	0.94
C3 levels	129 mg/dL
C4 levels	44.2 mg/dL
Antinuclear Antigen (ANA)	Normal
HIV, HBsAg, HCV rapid card test	Negative
Anti-HEV total, Anti-HEV IgM, Anti-HEV IgG, Anti-Hep B Core AG, Hep C	Negative
Hepatitis A Virus (HAV) total antibody	5.25 S/CO
Anti-Hepatitis A Virus (HAV) (IgM)	4.52 S/CO
Anti-Hepatitis A Virus (HAV) (IgG)	Negative
Stool routine microscopy	Normal
Stool culture	No growth of organisms
Skin biopsy	Necrotising vasculitis

[Table/Fig-2]: Diagnostic work-up of the patient.

For rash, a dermatologic consultation was done, and injection hydrocortisone 100 mg BID, tablet desloratadine 10 mg HS, and a lotion (calamine, pramoxine hydrochloride, and aloe vera) were prescribed. Skin biopsy from the lesion demonstrated thickened and hyalinised basement membrane of blood vessels of superficial dermis with deposition of fibrin and chronic inflammatory cells around the blood vessels suggesting necrotising vasculitis [Table/Fig-3a,b]. Evaluation of renal function test, 24-hour urinary protein, urine protein/creatinine ratio and complement (C3 and C4) levels revealed normal values, suggesting normally functioning kidneys.



[Table/Fig-3]: Histopathological examination of biopsy specimen (2a: 4× H&E and 2b: 20× H&E) illustrating thickened and hyalinised basement membrane (red arrow) of blood vessels of superficial dermis with deposition of fibrin and chronic inflammatory cells around the blood vessels (blue arrow) suggesting necrotising vasculitis.

The patient was discharged with a final diagnosis of HAV-induced CNV and was prescribed tapering doses of tablet prednisolone (40 mg once daily for 5 days) and followed-up after four weeks. During this period, the purpuric rash disappeared spontaneously and liver function tests returned to normal. Two months post-diagnosis, physical examination and laboratory values were normal.

DISCUSSION

Cutaneous Necrotising Vasculitis (CNV) is a rare dermatological condition characterised by inflammation and necrosis of small blood vessels. It often presents as palpable purpura. Though the exact incidence of CNV is not known, it is estimated that CNV is diagnosed in 155.5 cases per million individuals annually. CNV is multifactorial in origin, including drug reactions, viral infections and systemic diseases, thus complicating the diagnosis [1]. Though viral hepatitis is known to present with extrahepatic manifestations, HAV infection seldom presents with rash and transient arthralgia [2]. To the best of our knowledge, only four cases with HAV-induced CNV has been reported in literature [3-6].

CNV is often idiopathic (≈60%), but may be caused by several conditions, including autoimmune disorders, hypersensitivity drug reactions, lymphoproliferative disorders, malignancies and infections (≈40%) [1]. In addition to hepatic manifestations, the patients with HAV, HBV and HCV present with various extrahepatic signs and symptoms [7]. CNV occurs as a consequence of the inflammation of vessel walls due to direct infection, type II or immune complex-mediated reaction or cell-mediated hypersensitivity [8]. Similar to our patient, two cases of cutaneous leukocytoclastic vasculitis have been reported by Dan M and Yaniv R in women aged 31 years and Inman RD et al., in a woman aged 26 years [5,6]. Moreover, Press J et al., described a case of CNV during the acute phase of HAV in a two-year-old girl [3]. The cases reported by Ilan Y et al. and Inman RD et al., demonstrated cryoglobulinaemia, which was absent in our patient, highlighting heterogeneity in immune-mediated responses to HAV [4,6]. In our patients and that reported by Press J et al., and Dan M et al., vasculitis was associated with deposition of antibodies and complements without cryoprecipitates [3,5]. In our patient, the association of skin purpura and cytolytic hepatitis was strongly suggestive of viral aetiology. Our patient is unusual because CNV, although reported with other hepatotropic viruses, especially during the acute phase of hepatitis A, has been reported as an isolated association with HAV infection along with other viral infections (Hepatitis B and E) [2-6,9].

HAV is usually transmitted via the faeco-oral route [2]. Our patient had a history of consumption of outdoor food and drinks, which was probably contaminated and subsequent onset of diarrhoea confirmed the transfer of HAV via food. HAV infections occur worldwide and usually affect children without producing symptoms, but in adults it causes clinically apparent disease, often with jaundice [10]. The usual presentation includes fever, jaundice, diarrhoea, weight loss, pruritus, dark urine and hepatomegaly [7]. Approximately 30% of patients aged less than six years present with non-specific symptoms without jaundice and 70% adults present with non-specific and specific symptoms, including jaundice [11]. Though jaundice is one of the most common presenting symptoms in a HAV-infected patient, it is not always present, as observed in our patient who had high-grade fever and diarrhoea. Painful knee and ankle joints were attributed to reactive arthritis due to current illness. Moreover, laboratory evaluation revealed slightly raised liver enzymes and elevated levels of HAV total antibody and HAV IgM. Similarly, Dan M et al., and Inman RD et al., reported HAV IgM positivity [5,6]. Thus, the diagnosis of HAV infection was reached based on history of exposure to outside food, clinical presentation, though atypical and laboratory investigations.

Extrahepatic cutaneous manifestations are seldom observed and only four cases of HAV-induced CNV are reported [3-6]. Cutaneous vasculitis generally presents with a bilateral purpuric rash involving lower extremity that resolves within four weeks, usually leaving hyperpigmentation and/or atrophic scars. Though generally painless, rash may be associated with fever, burning sensation, itching, arthralgia, or myalgia [12]. In their case, Dan M et al., observed that the patient had erythematous papular rash with pruritic lesions primarily over the hips, but also involved arms and buttocks and arms, with occasional petechial rash. The rash was associated with malaise, fever, nausea and anorexia [5]. In another case, Inman RD et al., noted that erythematous papular rash with purpuric areas mostly present over the lower legs, with further involvement of the buttocks and arms. The patient also complained of swelling and painful movement of knees and ankles joints [6]. Similarly, our patient had reddish purple papular rash over both upper and lower limbs as well as back region associated with high-grade fever, diarrhoea and arthralgia.

The association between palpable purpura and HAV, leading to CNV, is an intriguing phenomenon that underscores the complex interplay between viral infections and immune-mediated skin disorders [2].

Our patient presented with extensive palpable purpura, diarrhoea, fever and joint swelling, which were indicative of an underlying inflammatory process. The elevated CRP, LDH and ESR further supported the presence of systemic inflammation. The exact link between HAV and CNV had not been fully elucidated; however, available literature suggests that HAV infection may trigger deposition of immune complexes, vascular inflammation and necrosis. Various factors that contribute to vasculitis in infection with hepatotropic viruses, including HAV, are systemic inflammation, cytokine release and complement activation. While HCV is predominantly associated with cutaneous vasculitis, the role of HAV as a potential trigger should not be overlooked [2,3,13].

The diagnosis of CNV is confirmed on histopathological examination of a biopsy specimen [1]. In our patient, skin biopsy illustrated necrotising vasculitis with fibrinoid necrosis of the vessel wall, mirroring the leukocytoclastic vasculitis reported by Dan M et al. and Inman RD et al., but without cryoglobulin or IgA paraprotein deposits [5,6]. These findings suggest immune complex deposition as a possible mechanism, though we could not perform direct immunofluorescence due to unavailability. The patient was not on medication, thereby ruling out hypersensitivity vasculitis. Cutaneous polyarteritis nodosa was excluded because necrotising vasculitis was not observed in the lower dermis and/or subcutaneous fat. Presence of normal serum IgA levels and the absence of other underlying conditions further support the notion that CNV was secondary to the acute HAV infection.

The majority of the patients (~90%) can be managed conservatively by eliminating the underlying causes (infection or drug), taking rest, elevation, compression and non-steroidal anti-inflammatory drugs for pain as well as topical antihistaminics or steroids for pruritus. In severe cases, systemic steroids are considered (e.g., prednisone in tapering doses). While patients with chronic or recurrent CNV (10%) are managed with immunosuppressive agents, including colchicine, dapsone, azathioprine and systemic steroids. Moreover, severe or refractory cases are treated with mycophenolate mofetil, methotrexate, hydroxychloroquine, cyclosporine and intravenous therapy of rituximab, infliximab, or immunoglobulin [14]. Immunosuppressive agents act by reducing inflammation and preventing further tissue damage. Antihistaminic agents reduce pruritus and block formation of endothelial gaps due to histamine with subsequent trapping of immune complexes [1]. HAV is self-limiting and recovery is spontaneous following HAV clearance [2]. Our patients had spontaneous resolution of the purpuric rash and normalisation of liver function within four weeks, aligning with the expected course of viral infections, reinforcing the link between HAV and CNV. In their cases, Dan M et al., and Inman RD et al., reported that the clinical and laboratory findings resolved spontaneously without therapy without any sequelae [5,6].

This is a case report of a single case and this itself is the limitation. Moreover, full-text case reports of Press J et al., and Ilan Y et al., were not available, so patient details could not be described completely [3,4].

CONCLUSION(S)

Even in the absence of typical hepatitis symptoms, physicians should consider HAV testing for patients who present with purpuric rash and cytolytic hepatitis because of the temporal association between HAV infection and CNV. In addition to hepatic involvement, HAV leads to various extrahepatic manifestations. Cutaneous manifestations could be the most significant, first, or even the only signs of the infection. The presence of palpable purpura and the histological findings of fibrinoid necrosis, coupled with the extracutaneous manifestation, could help the clinicians in correct diagnosis of the underlying HAV infection.

Acknowledgement

Authors would like to thank Dr. Vikas S. Sharma (MD), CEO, Maverick Medicorum® (India), for medical writing assistance in the preparation of this article.

REFERENCES

[1] Tsampau D, Buggiani G, Hercogová J, Lotti T. Cutaneous necrotizing vasculitis: A rational therapeutic approach. Dermatol Ther. 2012;25(4):335-39.
[2] Wang CR, Tsai HW. Human hepatitis viruses-associated cutaneous and systemic vasculitis. World J Gastroenterol. 2021;27(1):19-36.
[3] Press J, Maslovitz S, Avinoach I. Cutaneous necrotizing vasculitis associated with hepatitis A virus infection. J Rheumatol. 1997;24(5):965-67.
[4] Ilan Y, Hillman M, Oren R, Zlotogorski A, Shouval D. Vasculitis and cryoglobulinemia associated with persisting cholestatic hepatitis A virus infection. Am J Gastroenterol. 1990;85(5):586-87.
[5] Dan M, Yaniv R. Cholestatic hepatitis, cutaneous vasculitis, and vascular deposits of immunoglobulin M and complement associated with hepatitis A virus infection. Am J Med. 1990;89(1):103-04.
[6] Inman RD, Hodge M, Johnston ME, Wright J, Heathcote J. Arthritis, vasculitis, and cryoglobulinemia associated with relapsing hepatitis A virus infection. Ann Intern Med. 1986;105(5):700-03.
[7] Cozzani E, Herzum A, Burlando M, Parodi A. Cutaneous manifestations of HAV, HBV, HCV. Ital J Dermatol Venerol. 2021;156(1):05-12.
[8] Teng GG, Chatham WW. Vasculitis related to viral and other microbial agents. Best Pract Res Clin Rheumatol. 2015;29(2):226-43.
[9] Viallard JF, Vergier B, Lazaro E, Greib C, Pellegrin JL. Cutaneous necrotizing small-vessel vasculitis induced by acute hepatitis E. Clin Case Rep. 2019;7(8):1539-41.
[10] Koff RS. Hepatitis A. Lancet. 1998;351(9116):1643-49.
[11] Muñoz-Martínez SG, Díaz-Hernández HA, Suárez-Flores D, Sánchez-Ávila JF, Gamboa-Domínguez A, García-Juárez I, et al. Atypical manifestations of hepatitis A virus infection. Rev Gastroenterol Mex (Engl Ed). 2018;83(2):134-43.
[12] Alkhatib AA, Adler DG. Cutaneous necrotizing vasculitis associated with Hepatitis C virus infection. Dig Dis Sci. 2007;52(12):3438-39.
[13] Markiewski MM, Lambris JD. The role of complement in inflammatory diseases from behind the scenes into the spotlight. Am J Pathol. 2007;171(3):715-27.
[14] Micheletti RG. Treatment of cutaneous vasculitis. Front Med (Lausanne). 2022;9:1059612.

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PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Apr 25, 2025
- Manual Googling: Sep 02, 2025
- iThenticate Software: Sep 06, 2025 (8%)

ETYMOLOGY: Author Origin

EMENDATIONS: 6

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Apr 22, 2025**
Date of Peer Review: **Jul 04, 2025**
Date of Acceptance: **Sep 09, 2025**
Date of Publishing: **May 01, 2026**