Case Report

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Internal Medicine Section

Cutaneous Small Vessel Vasculitis Complicating Sepsis and Inflammatory Bowel Disease in an Older Adult with Chronic Kidney Disease: A Case Report

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ABSTRACT

Cutaneous Small Vessel Vasculitis (CSVV) is an immune-complex-mediated dermal vasculitis associated with infections, medications, and systemic inflammatory diseases. A man in his 70s with Chronic Kidney Disease (CKD), diabetes mellitus, and hypothyroidism presented with abdominal pain, diarrhoea, vomiting, Acute Kidney Injury (AKI), and palpable purpura involving all four limbs after receiving an undocumented parenteral drug. Investigations revealed Enterococcus faecalis Urinary Tract Infection (UTI), elevated inflammatory markers, severe renal dysfunction, and negative antineutrophil cytoplasmic antibodies. Skin biopsy confirmed Leukocytoclastic Vasculitis (LCV). Colonoscopy with histology and markedly elevated faecal calprotectin confirmed Inflammatory Bowel Disease (IBD), while renal biopsy showed Acute Tubular Injury (ATI) with class III diabetic nephropathy. Sepsis-associated CSVV was considered most likely, with IBD-related immune activation and drug-induced vasculitis as alternative contributors. Management prioritised infection control, pathogen-directed antibiotics, haemodialysis, and delayed corticosteroid therapy after microbiological control was achieved. The patient improved clinically but was discharged against medical advice at his own request before completion of the planned course of treatment, with scheduled outpatient follow-up and long-term outcome information was unavailable.

Keywords: Enterococcus faecalis, Inflammation, Leukocytoclastic

CASE REPORT

A man in his 70s presented with a three-day history of diffuse, dull abdominal pain radiating to both loins, accompanied by approximately two to three episodes per day of foul-smelling, non-bloody watery diarrhoea, vomiting, reduced oral intake, and a generalised non-pruritic purpuric rash involving all four limbs. One week before admission, he had experienced similar gastrointestinal symptoms and had received an undocumented parenteral medication at a nearby clinic. Within a few days of receiving the medication, he developed the purpuric rash without mucosal involvement [Table/Fig-1]. He denied fever, rigours, chest pain, palpitations, dyspnoea, syncope, headache, focal neurological symptoms, and oliguria. His medical history included CKD, Type 2 Diabetes Mellitus (T2DM), systemic hypertension, hypothyroidism, seizure disorder, vestibulobasilar insufficiency, a chronic right occipital infarct, previous traumatic urethral injury, and a resolved hepatic abscess. He had no previous history of cutaneous vasculitis or a similar purpuric eruption. His regular medications included antihypertensive, antidiabetic, antiepileptic, and thyroid-replacement therapies. He was a former alcohol user and denied smoking or recreational drug use.

On examination, the patient was conscious and oriented but pale, with bilateral upper- and lower-limb oedema and purpuric lesions. Cutaneous examination revealed multiple palpable, non-blanching, non-pruritic lesions distributed over both upper and lower limbs, without ulceration, necrosis, blistering, or mucosal involvement; ecchymotic patches subsequently appeared over both forearms during the hospital stay. He was afebrile at presentation. His BP was 120/80 mmHg, pulse rate was 73 beats/minute, respiratory rate was 18 breaths/minute, capillary blood glucose was 158 mg/dL and oxygen saturation was 100% on room air. Cardiovascular examination was unremarkable. Respiratory examination revealed bilateral basal crepitations. The abdomen was soft, with generalised tenderness and left renal-angle tenderness, without guarding or rebound tenderness. Neurological examination showed a Glasgow Coma Scale score of 15/15 with no focal neurological deficit. The provisional diagnosis was an infective gastrointestinal illness with UTI and sepsis-associated AKI superimposed on CKD, together with a concurrent vasculitic cutaneous eruption.



[Table/Fig-1]: Non-pruritic purpuric rash developed in all four limbs.

Laboratory investigations showed leucocytosis of 13,300 cells/mm^3, blood urea nitrogen of 207 mg/dL, serum creatinine of 6.23 mg/dL, erythrocyte sedimentation rate of 80 mm/hour, C-reactive protein of 55.65 mg/L, and procalcitonin of 4.20 ng/mL. Urine analysis demonstrated bacteria, blood, leukocytes, and 6-8 pus cells/high-power field. Urine culture grew *Enterococcus faecalis*, whereas two sets of blood cultures were sterile. Serial laboratory investigations during hospitalisation are summarised in [Table/Fig-2]. The patient had an indwelling urethral catheter in situ, retained following his earlier traumatic urethral injury during previous hospital stay (3 weeks), re-catheterised by urology team after admission and advised to retain foley's for three more weeks. The diagnosis of catheter-associated UTI was therefore based on left renal-angle tenderness, pyuria, bacteriuria, haematuria, leucocyte esterase positivity on urinalysis, together with the growth of *E. faecalis* on urine culture and elevated inflammatory markers and procalcitonin, in the absence of an alternative focus on imaging.

Parameter (reference range)	Day 1 (admission)	Day 3	Day 8	Day 12	Day 16 (pre-discharge)
Haemoglobin (13-17 g/dL)	10.1	6.5	7.3	7.2	8.4*
Total leucocyte count (4,000-10,000 cells/mm^3)	13,300	10,300	11,500	6,400	3,900
Platelet count (150,000-450,000/mm^3)	370,000	273,000	213,000	132,000	86,000
Blood urea (17.1-49.2 mg/dL)	207	197	99	96	139
Serum creatinine (0.8-1.3 mg/dL)	6.23	8.49	4.87	5.32	4.66
Sodium (136-143 mEq/L)	130	132	137	138	135
Potassium (3.5-5.1 mEq/L)	3.3	3.2	3.4	3.3	4.3
Serum albumin (3.2-4.6 g/dL)	1.8	2.3	2.5	–	–

[Table/Fig-2]: Serial laboratory investigations during hospitalisation.
*After transfusion of packed red blood cells. Key one-time investigations: ESR 80 mm/hour; CRP 55.65 mg/L; procalcitonin 4.20 ng/mL; admission coagulation profile deranged (prothrombin time 156 s, INR 17.0, aPTT 72.6 s), normalising after vitamin K and fresh frozen plasma (INR ≈1.2); D-dimer 10,000 ng/mL; LDH 847 U/L, declining to 211 U/L before discharge; reticulocyte count 3.5%, later 0.7%; direct Coombs test positive (IgG-positive, C3d-negative); complement C3 100.41 mg/dL (normal), C4 9.77 mg/dL (borderline low); serum vitamin B12 450.1 pg/mL, serum folate 1.94 ng/mL (low); faecal calprotectin >800 µg/g; urine analysis: protein 1+, blood 3+, leukocyte esterase 3+, bacteria 3+, pus cells 6–8/HPF; urine culture: Enterococcus faecalis; two sets of blood cultures sterile; stool occult blood positive

Abdominal computed tomography demonstrated cystitis, mild ascites, focal hepatic steatosis without an active hepatic abscess, and a left adrenal adenoma. Punch biopsy of a skin lesion confirmed LCV. Antineutrophil cytoplasmic antibodies against myeloperoxidase and proteinase-3 were negative; complement C3 was normal and C4 was borderline low. Renal biopsy subsequently demonstrated ATI superimposed on class III Renal Pathology Society (RPS) diabetic nephropathy, without pauci-immune glomerulonephritis. Because diarrhoea persisted, faecal calprotectin was measured and was markedly elevated at >800 µg/g; colonoscopy with histopathological examination subsequently confirmed IBD. Upper gastrointestinal endoscopy demonstrated erosive gastritis. The principal differential diagnoses were sepsis-associated CSVV, drug-induced vasculitis related to the undocumented parenteral medication, IBD-associated vasculitis, and primary antineutrophil cytoplasmic Antibody-Associated Vasculitis (AAV). Primary microscopic polyangiitis was excluded because of negative serology, absence of pulmonary or upper-airway involvement, and absence of pauci-immune glomerulonephritis. The final diagnosis was antineutrophil cytoplasmic antibody-negative CSVV, most likely triggered by *E.faecalis* UTI and sepsis, with IBD-related immune activation as a possible concurrent contributor. Drug-induced vasculitis remained possible but could not be confirmed because the administered medication was unknown. The differential diagnosis with the present case was tabulated [Table/Fig-3].

The patient was transferred to the medical intensive care unit and initially treated with empirical intravenous antibiotics (meropenem and metronidazole), intravenous fluids, and strict fluid-balance monitoring. After the urine culture result became available, antimicrobial therapy was changed to intravenous linezolid in accordance with the culture and sensitivity report. Worsening azotaemia necessitated seven sessions of haemodialysis. Anaemia, hypoalbuminaemia, and coagulopathy were managed with packed red blood cell transfusions, intravenous albumin, vitamin K, and fresh frozen plasma. Colchicine was initiated for recurrent cutaneous lesions, while systemic corticosteroids were deferred during active infection. After microbiological control had been achieved, intravenous dexamethasone was commenced and subsequently changed to a short tapering course of oral prednisolone. Erosive gastritis was managed with proton-pump inhibitor therapy, and the urinary catheter was replaced following accidental removal and urethral injury. After approximately three weeks of hospitalisation, the patient was haemodynamically stable and symptomatically improved; however, he requested discharge against medical advice before completing the antibiotic course and repeat abdominal imaging.

Differential diagnosis	Key supporting features	Arguments against	Histology pattern	Final disposition
Sepsis-Associated CSVV (Primary Working Diagnosis)	E. faecalis CAUTI confirmed; temporal link between infection onset and purpura; ANCA negative; improved with antibiotics and source control prior to immunosuppression	Cannot fully exclude concurrent IBD or drug trigger; does not explain initial prior rash episode	LCV on punch biopsy; pattern non-specific, consistent with any secondary CSVV	ACCEPTED as primary contributor; antibiotic-first strategy validated by clinical recovery
Drug-Induced CSVV	Rash onset temporally following undocumented parenteral drug at external clinic one week prior; drug-induced LCV is a leading cause of CSVV	Drug identity unknown; cannot confirm or refute; no re-challenge possible; no eosinophilia documented	LCV pattern on biopsy; identical to other secondary CSVV; cannot histologically distinguish from other aetiologies	POSSIBLE but unverifiable; acknowledged as a diagnostic limitation; warrants documentation of all prior drug exposures at follow-up
IBD-Associated Vasculitis (Extra-intestinal Manifestation)	Colonoscopy and histology confirmed IBD; faecal calprotectin >800 µg/g indicating active intestinal inflammation; IBD-associated CSVV is a recognised EIM	IBD confirmed after rash; cannot establish temporal primacy; bowel inflammation may be a parallel rather than causal driver	LCV on biopsy; DIF not reported separately; IgA deposition would strengthen IBD-EIM link but is non-specific	ACCEPTED as parallel contributor; colchicine commenced for recurrent ecchymosis; short prednisolone course initiated after infection control
Primary ANCA-Associated Vasculitis (AAV/MPA)	Renal dysfunction (AKI on CKD); palpable purpura; systemic inflammatory burden	ANCA negative (both MPO and PR3); no pauci-immune GN on renal biopsy (ATI+diabetic nephropathy); no pulmonary haemorrhage, ENT, or peripheral nerve involvement; clear infective precipitant	Renal biopsy: Acute Tubular Injury (ATI) with class III RPS diabetic nephropathy; no crescentic GN or immune deposits consistent with AAV	EXCLUDED; MPA label incorrect and retracted; replaced with ANCA-negative CSVV. EULAR 2022 guidelines criteria not met

Septic/Embolic Vasculitis	Active systemic infection; bacteraemia theoretically possible; purpuric lesions mimicking septic emboli	Both sets of blood cultures sterile; no vegetations on echocardiography documented; lesion distribution and morphology favoured immune-complex CSVV over embolic occlusion	LCV pattern; no intravascular organisms or fibrin-platelet thrombi described	EXCLUDED on clinical and microbiological grounds
IgA Vasculitis (Henoch-Schönlein Purpura)	Palpable purpura on lower limbs; abdominal pain; renal involvement; GI symptoms	Age >70 (HSP rare in adults); no DIF reported showing IgA deposits; no arthritis; clinical context dominated by sepsis and IBD	DIF would be required to confirm IgA deposits; not performed or not reported	NOT CONFIRMED; DIF should be obtained at follow-up biopsy if lesions recur

[Table/Fig-3]: Differential diagnosis summary: Cutaneous Small Vessel Vasculitis (CSVV) in the context of sepsis, Inflammatory Bowel Disease (IBD), and Chronic Kidney Disease (CKD).
CSVV: Cutaneous small vessel vasculitis; CAUTI: Catheter-associated urinary tract infection; ANCA: Antineutrophil cytoplasmic antibody; AAV: ANCA-associated vasculitis; MPA: Microscopic polyangiitis; CKD: Chronic kidney disease; AKI: Acute kidney injury; IBD: Inflammatory bowel disease; EIM: Extra-intestinal manifestation; LCV: Leukocytoclastic vasculitis; DIF: Direct immunofluorescence; GN: Glomerulonephritis; ATI: Acute tubular injury; RPS: Renal pathology society; HSP: Henoch-Schönlein purpura; MPO: Myeloperoxidase; PR3: proteinase-3

Follow-up after one week was planned in the general medicine, dermatology, and nephrology clinics, with full blood count and renal-function monitoring and all the laboratory parameters were normal, the pruritus started to resolve and the symptoms of the IBD also started to resolve. Long-term outcomes regarding renal recovery, vasculitis recurrence, and IBD activity were unavailable. [Table/Fig-4] provides the clinical timeline for the case.

DISCUSSION

The CSVV is an immune complex-mediated vasculitis of the dermal small vessels that may be triggered by infections, medications, autoimmune disorders, or systemic inflammatory diseases [1-3]. Histopathologically, it is characterised by LCV, although this pattern is not specific for a particular aetiology. Consequently, identification of the underlying trigger requires careful correlation of clinical chronology, drug exposure, microbiological findings, serology, and histopathology [2,3]. This distinction is particularly important in older adults with CKD, in whom premature immunosuppression during an active infection may result in substantial harm [4,5].

The principal diagnostic consideration in this case was sepsis-associated CSVV. The patient had a CAUTI caused by *E.faecalis*, elevated inflammatory markers and procalcitonin, AKI superimposed on CKD, and a temporally associated purpuric eruption. Skin biopsy confirmed LCV. The clinical improvement following pathogen-directed antimicrobial therapy and infection control supported infection-associated CSVV as the most likely diagnosis. However, drug-induced CSVV could not be excluded because an undocumented parenteral medication had been administered

Timepoint	Clinical events	Investigations and decisions
~1 week pre-admission	Prior clinic visit: similar diarrhoea and abdominal complaints. Received undocumented parenteral drug. Developed non-itchy maculopapular purpuric rash over all limbs within days. No mucosal involvement noted.	Drug identity: unknown and unrecorded. Drug-induced CSVV cannot be excluded (see Differential Diagnosis).
Day 0 - ED presentation	Emergency department attendance: 3-day history of dull abdominal pain, foul-smelling watery diarrhoea, vomiting, reduced oral intake, and generalised rash. Vitals: BP 120/80 mmHg, HR 73/min, CBG 158 mg/dL. Bilateral basal crepitations. Generalised abdominal tenderness, left renal angle tenderness.	Bloods: WBC 13,300 cells/mm^3, creatinine 6.23 mg/dL, BUN 207 mg/dL, ALP 182 U/L. Urinalysis: 3+ bacteria, leukocytes, blood, 6-8 pus cells/HPF. ESR 80 mm/hr, CRP 55.65 mg/L. Bedside USG: cortical thinning right kidney, collapsed bowel loops, mild-to-moderate ascites.
Day 0 - MICU transfer	Transferred to medical ICU given sepsis and pre-existing renal dysfunction. Empirical i.v. Cefoperazone-Sulbactam 1.5 g BD commenced. IV fluids, pantoprazole, nifedipine continued.	Impression: UTI with AKI on background CKD. CT abdomen: focal hepatic steatosis (no active abscess), cystitis, mild ascites, left adrenal adenoma. Surgical opinion: metronidazole added empirically given prior history of hepatic abscess and bowel pathology (CT showed no active abscess).
Days 1-2	Urine and blood cultures processed. Dermatology review: punch skin biopsy performed. Antimicrobial therapy de-escalated to i.v. Linezolid 600 mg BD on culture result.	Urine culture: Enterococcus faecalis (sensitive). Blood cultures ×2: sterile. Biopsy result: Leucocytoclastic Vasculitis (CSVV confirmed). ANCA (MPO+ PR3): negative.
Days 2-3	Creatinine rose to 8.49 mg/dL. Hypoalbuminaemia (1.8 g/dL): 2 units IV albumin 20% administered. Nephrology review: Haemodialysis (HD) sheath inserted, PRBC transfusion initiated. Procalcitonin 4.20 ng/mL: consistent with sepsis-driven AKI.	Haemoglobin fall 10.1→6.5 g/dL. Platelets 273×10^9/L. LDH 211 U/L during the hospitalisation around 3rd day. Autoimmune workup requested given AKI + vasculitis. Complement: C3 100 mg/dL (normal), C4 9.77 mg/dL (borderline low).
Days 3-5	Stool occult blood positive ×2. Coagulopathy identified (PT >2 min, aPTT 95.7 sec, D-dimer 10,000 ng/mL). i.v. Vitamin K 10 mg/day ×3 days, 4 units FFP. Folic acid 5 mg OD and IV thiamine 100 mg OD commenced. General surgery review: upper GI endoscopy.	UGI endoscopy: erosive gastritis. Biopsy deferred (bleeding risk). Folate 1.94 (low), B12 450.1 pg/mL.
Days 5-7	Persistent diarrhoea and vomiting despite antibiotics and antiemetics. Faecal calprotectin sent. Dermatology: new ecchymosis over bilateral forearms. Colchicine commenced by dermatologist (rationale: suspected neutrophilic dermatosis component/recurrent cutaneous inflammation pending immunosuppression initiation).	Faecal calprotectin: markedly elevated (>800 µg/g), consistent with active intestinal inflammation - prompting colonoscopy.
Days 7-10	Colonoscopy and colonic biopsy: confirmed IBD. Lactulose commenced for bowel regulation. Histology review of skin biopsy: ANCA-negative CSVV (original MPA label revised - see Discussion). i.v. Dexamethasone 8 mg OD commenced after infection control confirmed; transitioned to oral prednisolone with tapering plan.	IBD diagnosis established. Diagnosis revised from MPA to ANCA-negative CSVV, most likely secondary to sepsis with IBD-related immune activation as a parallel contributor.
Days 10-14	Urology: Foley catheter re-catheterised after accidental removal with urethral injury; retained for 3 further weeks. Neurology: CT brain - chronic right occipital infarct, small-vessel ischaemic change; EEG normal. Antiepileptic continued.	Ongoing intermittent HD: 7 sessions total. Renal biopsy: Acute Tubular Injury (ATI) with class III RPS diabetic nephropathy.
Day ~21 (DAMA)	Patient haemodynamically stable and symptomatically improved. Opted for discharge against medical advice (DAMA) prior to: (1) completion of antibiotic course; (2) repeat imaging to confirm resolution of prior hepatic finding; (3) completion of vasculitis follow-up plan.	Discharge regimen: prednisolone taper, PPI, lactulose, ongoing comorbidity medications. Outpatient review planned at 1 week: FBC, RFT, dermatology, nephrology. Limitations: outcome of hepatic steatosis/prior abscess site unknown; long-term vasculitis and IBD activity unconfirmed.

[Table/Fig-4]: Clinical timeline: chronological sequence of events.
ED: Emergency department; MICU: Medical intensive care unit; CSVV: Cutaneous small vessel vasculitis; ANCA: Antineutrophil cytoplasmic antibody; MPA: Microscopic polyangiitis; IBD: Inflammatory bowel disease; CAUTI: Catheter-associated urinary tract infection; AKI: Acute kidney injury; CKD: Chronic kidney disease; HD: Haemodialysis; DAMA: Discharge against medical advice; USG: Ultrasonography; CT: Computed tomography; BP: Blood pressure; HR: Heart rate; CBG: Capillary blood glucose; WBC: White blood cell count; BUN: Blood urea nitrogen; ALP: Alkaline phosphatase; ESR: Erythrocyte sedimentation rate; CRP: C-reactive protein; HPF: High-power field; FFP: Fresh frozen plasma; PRBC: Packed red blood cells; EEG: Electroencephalography; RPS: Renal pathology society; ATI: Acute tubular injury; PPI: Proton-pump inhibitor

at an external facility shortly before the onset of the rash [2,4,5]. Medications are well-recognised triggers of LCV, but the inability to identify the administered drug precluded definitive assessment of causality [1-3].

The IBD-associated vasculitis was also considered because persistent gastrointestinal symptoms led to the detection of markedly elevated faecal calprotectin (>800 µg/g), followed by colonoscopic and histological confirmation of IBD. Vasculitis is an uncommon but recognised extra-intestinal manifestation of IBD and may involve small or large vessels through shared immune-mediated mechanisms [6-8]. Nevertheless, because IBD was diagnosed after the onset of the cutaneous eruption and active infection was concurrently present, a direct causal relationship between IBD and CSVV could not be established. IBD-related immune activation was therefore considered a possible parallel contributor rather than the sole precipitating factor.

Primary antineutrophil cytoplasmic AAV, particularly microscopic polyangiitis, was an important differential diagnosis because of the combination of palpable purpura, systemic inflammation, and renal dysfunction [9,10]. However, both myeloperoxidase- and proteinase-3-ANCA were negative, there was no pulmonary, upper respiratory tract, or peripheral neurological involvement, and renal biopsy demonstrated ATI with class III RPS diabetic nephropathy rather than pauci-immune necrotising glomerulonephritis. These findings substantially reduced the likelihood of primary AAV [11]. Although microscopic polyangiitis had been initial clinical working diagnosis during hospital stay, and it was subsequently excluded on the basis of negative AAV and the renal biopsy findings. Septic or embolic vasculitis was also considered but was less likely because blood cultures were sterile and skin histology showed LCV without reported intravascular organisms or thrombotic occlusion. IgA vasculitis could not be confirmed because direct immunofluorescence demonstrating IgA deposition was unavailable.

Similar presentations have been reported previously. Simonetti O et al., reviewed cutaneous vasculitides complicating IBD and emphasised that, although rare, they are an important cause of morbidity and a significant diagnostic challenge [12]. In the largest series to date, Sy A et al., described 32 patients with coexisting IBD and vasculitis, including isolated cutaneous vasculitis, and noted that IBD usually preceded the vasculitis [6]; in contrast, IBD was diagnosed after the cutaneous eruption in the present case, further arguing against IBD as the sole trigger. de Groot K highlighted that renal involvement in small-vessel vasculitis is a strong predictor of outcome and warrants early evaluation [13] in this patient, renal biopsy was pivotal in attributing the renal dysfunction to ATI superimposed on diabetic nephropathy rather than vasculitic glomerulonephritis. Drug-induced CSVV in patients with renal impairment is well documented: Carrasquel M et al., reported vancomycin-induced CSVV in a patient with end-stage renal disease that resolved rapidly after withdrawal of the offending drug [14], underscoring why the undocumented parenteral medication in our case could not be excluded as a trigger. Similarly, Yang M et al., described recurrent streptococcal infection-associated single-organ CSVV in which antimicrobial therapy formed the cornerstone of management before immunosuppression [15], supporting the infection-first approach adopted in the present case.

Several limitations should be acknowledged. The identity of the parenteral drug administered before admission was unavailable, preventing definitive exclusion of drug-induced CSVV. Direct immunofluorescence of the skin biopsy was not separately reported and could have provided additional information regarding immune-complex deposition, particularly IgA. Furthermore, the patient was discharged against medical advice before completion of therapy and in-hospital reassessment; therefore, long-term vasculitis recurrence, renal recovery, and IBD activity could not be determined. Overall, the findings favour sepsis-associated CSVV, with IBD-related immune activation as a possible concurrent contributor and drug-induced CSVV remaining an unconfirmed alternative. This case emphasises the value of sequential clinicopathological assessment and infection control before immunosuppression in patients with overlapping infectious, renal, gastrointestinal, and vasculitic manifestations.

CONCLUSION(S)

This case highlights the diagnostic challenge of CSVV occurring with concurrent sepsis, IBD, and CKD. An infection-first approach, supported by microbiological testing, skin biopsy, serology, and renal evaluation, helped avoid premature immunosuppression. Sepsis-associated vasculitis was considered the most likely diagnosis, with IBD-related immune activation as a possible contributor.

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