# Painless perianal growth in an elderly male

Dear Editor,

A 61-year-old male presented with insidious onset of a lobulated perianal mass for the past 5 months. It was progressively increasing in size without any associated bleeding, pain, discharge or alteration in bowel habits. The patient complained of malaise and occasional palpitations. The case was previously clinically diagnosed as a Buschke-Lowenstein tumour by another physician and had undergone one cycle of cryosurgery. Clinical differentials considered were Buschke Lowenstein tumour, perianal squamous cell carcinoma and perianal amyloidosis.

The vital examination was normal except for an elevated blood pressure of 150/90 mm of Hg. There was no lymphadenopathy. Cutaneous examination revealed a single, 13 × 8 cm skin-coloured, firm, non-tender fleshy lobulated mass with cobblestoning of surface, spanning across the perineal raphe and eczematisation of surrounding skin [Figure 1]. There was no involvement of anal mucosa on per-rectal examination and proctoscopy.

Routine investigation revealed haemoglobin of 9.9 gm/dL with normocytic, normochromic RBCs and occasional anisocytosis. Erythrocyte sedimentation rate was 5 mm/hour, and 24-hour urinary protein levels were 2 gm/day. Serum creatinine was 5 mg/dL with hyperphosphatemia, hypocalcaemia, and metabolic acidosis. Serum protein, albumin and globulin levels were 5.5, 3.26 and 2.24 gm/dL, respectively, with an albumin/globulin ratio of 1.45. Electrocardiogram showed sinus tachycardia, nonspecific ST depression, and intraventricular conduction block. Leukocyte and platelet counts, coagulation profile, serologies for human immunodeficiency virus, syphilis, and hepatitis B, C and X-rays were normal.

Biopsy from the lesion stained with haematoxylin-eosin showed marked diffuse deposition of fibrillary, pale pink material in the dermis and subcutis. [Figures 2a, 2b] Bright green fluorescence was detected on staining with thioflavin T, leading to the diagnosis of amyloidosis [Figures 2c, 2d]. Immunohistochemistry showed positivity for amyloid light-chain protein, λ type of light chains. This prompted us to do a serum and urine protein electrophoresis, which showed hypo-proteinemia and hypogammaglobulinemia with oligoclonal bands. Immunotyping revealed the absence of an M band/monoclonal gammopathy. Urinary Bence Jones proteins were absent. The patient was advised to undergo a bone marrow examination and further management in liaison with a haematologist, to which he did not agree and resorted to alternative Ayurvedic medicinal care. A total of 6 months later, the patient returned with florid purpuric

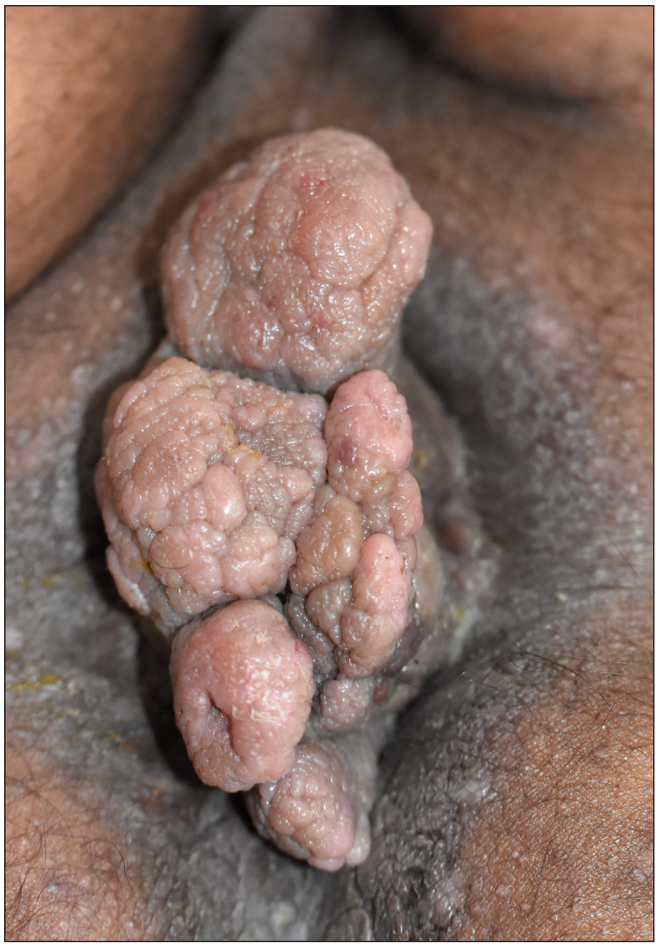

**Figure 1:** Fleshy, lobulated perianal mass with cobblestoned appearance of surface.

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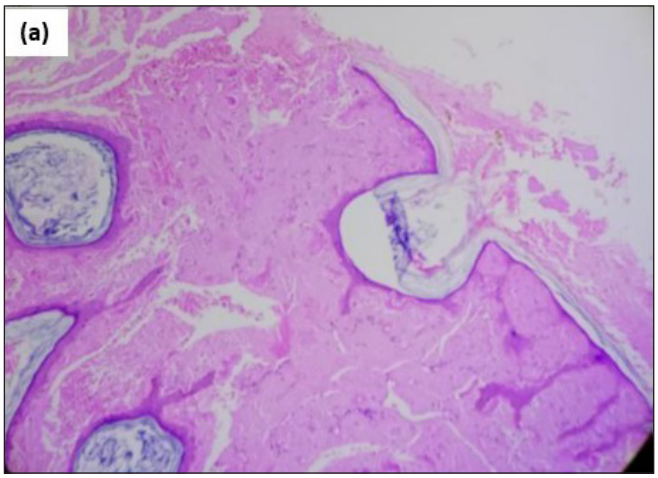


**Figure 2a:** At 100× magnification.

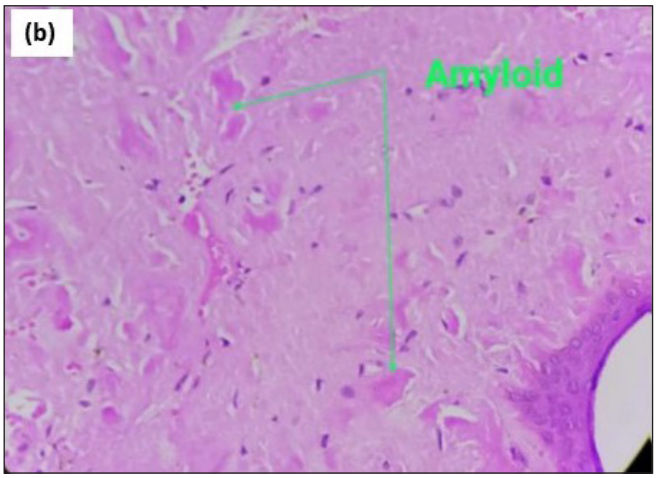


**Figure 2b:** At 400× magnification.

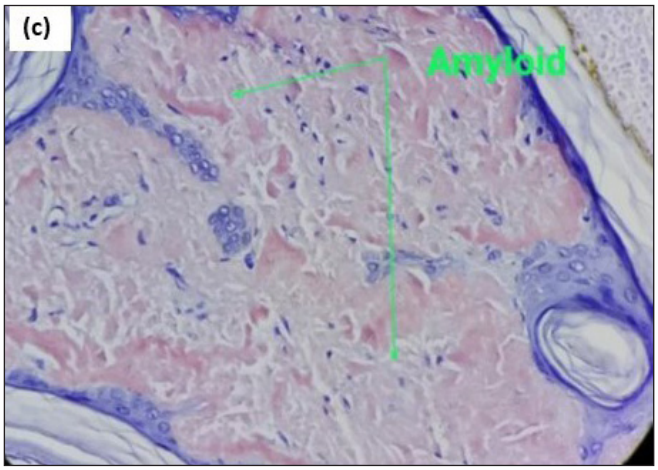


**Figure 2c:** Congo red staining at 400× magnification.

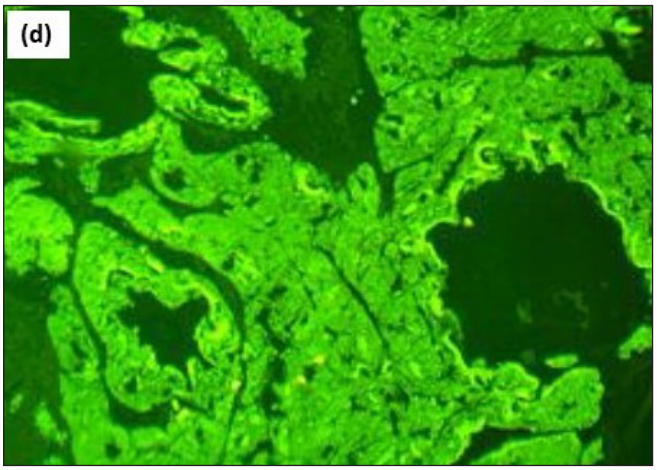


**Figure 2d:** Bright green florescence with thioflavin T, showing amyloid positivity.

papules, plaques and waxy infiltrated nodules all over the body, macroglossia and splinter haemorrhages in nails with breathlessness at rest, and pedal oedema as anticipated in the progressive course of systemic amyloidosis. Bortezomib induction therapy with autologous stem-cell transplant was planned for the patient. However, he shortly succumbed to cardiac complications, which had progressed due to a delay in treatment initiation.

Primary systemic amyloidosis can present with mucocutaneous features in 30–40% of cases which, when identified early, can prompt rapid institution of therapy.[1] Other early findings like weight loss and fatigue are nonspecific. Classical cutaneous features like periorbital purpura, waxy infiltrated nodules and macroglossia result from infiltration of amyloid in vessel walls, dermis and subcutis.

Atypical presentations resembling hand bruises, cutis verticis gyrata, localised nodulo-ulcerative growth on the tongue mimicking squamous cell carcinoma, and acquired digital cutis laxa-like picture and chronic paronychia are described in the literature.[1–5] We report a novel presentation as an isolated perianal mass where clinical-pathological correlation led us to evaluate for systemic amyloidosis. In our case, evidence of frank plasma cell clonality was not found at presentation, which is often the case before amyloid-related organ dysfunction ensues.[6]

The role of a dermatologist in the multidisciplinary care of primary systemic amyloidosis is pivotal. By the recognition of early and unconventional cutaneous features, there is an opportunity to make a timely diagnosis and initiate appropriate referrals before the onset of organ failure, thereby potentially prolonging the survival of such patients.

## Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

## Conflicts of interest

There are no conflicts of interest.

***Deval Mistry*, *Shikha R. Shah*, *Harshita R. Vyas*, *Bela J. Shah***

Department of Dermatology, BJ Medical College, Ahmedabad, Gujarat, India.

**Corresponding author:**
Dr. Harshita R. Vyas,
Department of Dermatology, BJ Medical College, Ahmedabad, Gujarat, India.
dr.harshitavyas@gmail.com

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# Amivantamab (JNJ-61186372)-induced adverse cutaneous reaction

Dear Editor,

The Food and Drug Administration (FDA) granted accelerated approval to amivantamab for adult patients with locally advanced or metastatic non-small cell lung cancer with epidermal growth factor receptor (EGFR) exon 20 insertion mutations, whose disease has progressed on or after platinum-based chemotherapy. Activating EGFR mutations render resistance to treatment with EGFR tyrosine kinase inhibitors. This resistance is mediated by multiple mechanisms, which include secondary EGFR mutation and activation of the c-mesenchymal-epithelial transition factor pathway. Amivantamab is a bispecific monoclonal antibody that targets both EGFR and mesenchymal-epithelial transition factor.[1] Acting on both targets provides amivantamab an "avidity effect," which is superior to poziotinib or cetuximab in terms of therapeutic efficacy.[2] We report a case of adverse cutaneous reaction following amivantamab treatment.

A 53-year-old man without a history of skin disease was diagnosed as right lung adenocarcinoma with multiple mediastinal lymph node metastases. His genetic testing showed epidermal growth factor receptor (EGFR) exon 20 insertion mutations and hence was treated with amivantamab (JNJ61186372)—targeted therapy.The dosage of amivantamab was 1050 mg weekly for the first four weeks, and then every two weeks thereafter. He began to develop facial skin lesions that gradually extended to the scalp, buttocks and lower limbs after three weeks of treatment. He complained of skin pain but without any accompanying constitutional symptoms like fever, breathlessness, facial oedema, jaundice, etc. Dermatological examination showed purulent crusts on the scalp, [Figure 1] scattered erythematous papules on the face, legs and scrotum along with scattered crusts on the face [Figures 2–4]. Erosive erythema was seen on the perianal area [Figure 5]. Nails showed paronychia-like changes [Figures 6]. The cutaneous adverse events were grade 3 in severity. The pulse, blood, pressure, respiratory rate, temperature, lymph nodes were normal. Scalp pus culture showed *S. aureus* infection, with sensitivity to amoxicillin, gentamicin and tetracycline. His haemogram, renal and liver function tests, absolute eosinophil count and peripheral smear analysis were within normal limits. He was treated with kangfuxin liquid, compound polymyxin b ointment, compound neomycin ointment and recombinant human epidermal growth factor gel without systemic steroids and continued amivantamab treatment. Skin lesions improved after a week. He received 22 more injections of amivantamab, and the rash appeared basically similar to the initial manifestation after about a week of each infusion. In subsequent recurrences, the pus culture and sensitivity did not reveal any pathogens and each time,

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