Sir,

An 80-year-old male with no prior comorbidity presented with the complaints of multiple fluid-filled lesions with few areas of erosions over the body of five days duration.

It started on the trunk with few vesicles over the chest and upper back which progressed to involve the rest of the body over the next two to three days. There was history of fever associated with the skin rash. Patient also complained of mild pain over the lesions. The vesicles and bullae burst open to leave behind erosions with hemorrhagic crusting which healed over the next five to six days. There was a history of similar lesions in the relatives of the patient. There was no history suggestive of any systemic infection in the form of headache, breathlessness, or loss of appetite. There was intake of drugs upto six weeks prior to the onset of symptoms. There was no history of atopy. On examination, the patient was poorly built (BMI: 16.2 kg/m²). Pallor was present, however there was no lymphadenopathy. Cutaneous examination revealed multiple, well-defined, discrete to grouped polysized hemorrhagic vesicles with crusting distributed over the entire body with involvement of scalp, palms, and soles, associated with mild tenderness over the involved skin and with sparing of the mucosal surfaces [Figure 1].

Tzanck smear from the vesicle revealed a hemorrhagic fluid which demonstrated multi-nucleated giant cells on microscopy [Figure 2]. Chest x-ray revealed multiple ill-defined and nodular opacities in bilateral upper zones suggestive of an infective pathology, most likely viral etiology due to the bronchopneumonia pattern followed. Mantoux test to rule out tuberculosis was negative. Routine hematological and biochemical parameters were within normal limits apart from mild anemia (Hb 10 gm/dl). Peripheral smear showed a microcytic hypochromic picture suggestive of iron deficiency anemia, further confirmed on iron studies (serum iron, TIBC, and serum ferritin). Varicella IgM antibodies were found to be positive. The coagulation parameters (PT, PTTK, and INR) were found to be normal. Screening for internal malignancy was done and tumor markers (LDH, prostate-specific antigen (PSA), and CA 125) were found negative.

Patient was diagnosed as a case of hemorrhagic varicella based on history, clinical examination, and investigations.

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He was given tab acyclovir 800 mg five times/day for 7 days, along with topical therapy in the form of potassium permagnate soaks and framycetin cream. Patient responded well to the therapy with healing of lesions with crusting and later few hypopigmented macules [Figure 3].

Varicella is a benign viral exanthematous disease of childhood and less commonly adults, caused by varicella zoster virus. Rarely, it can lead to complications such as hemorrhagic varicella, most commonly in immuno-compromised patients. Case reports of fatal hemorrhagic chicken pox have been reported in children with steroid-dependent asthma, nephrotic syndrome, and chronic liver disease.[1]

Adults with underlying disease such as leukaemia, on prolonged steroids, chemotherapy or bone marrow transplant recipients are also at an increased risk of developing varicella complications.[2]

Most complications of varicella can be grouped into following categories: (a) bacterial superinfection by Staphylococcus aureus or Streptococcus pyogenes; (b) varicella-associated Reye’s syndrome; (c) central nervous system complications such as cerebellar ataxia and encephalitis; (d) pulmonary complications such as pneumonitis; and (e) hemorrhagic complications like hemorrhagic rash and disseminated intravascular coagulation.[3,4]

Coagulopathies are frequently associated with varicella infection and a variety of etiological mechanisms have been described, ranging from secondary thrombocytopenia to disseminated intravascular coagulation. Hemorrhagic complications can manifest as petechiae, ecchymosis or with features of internal organ bleeding along with the hemorrhagic rash in the patients.[5] Our patient only had hemorrhagic rash with no evidence of cutaneous or internal organ bleeding.

Acyclovir which is life-saving in such circumstances should be given as early as possible to be effective. Intravenous foscarnet may also be given in patients not responding to acyclovir, but our patient responded well to oral acyclovir.

Varicella in elderly has been rarely reported due to effective antibody formation post varicella vaccination in countries where this vaccine is regularly administered. However, considering old age being an immunocompromised state, development of hemorrhagic varicella can be rarely encountered. Our patient is a rare example of an elderly male with no underlying immune-suppression developing hemorrhagic varicella.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.
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Conflicts of interest
There are no conflicts of interest.

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