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Diagnosis at first sight

Fever and skin lesions in a patient undergoing chemotherapy

Fiebre y lesiones cutáneas en paciente en quimioterapia

Case report

We present the case of a 62-year-old male with a history of Waldenström macroglobulinaemia and large B-cell lymphoma who had been treated for three months with rituximab, cyclophosphamide, doxorubicin, vincristine and prednisone (R-CHOP). He also had a previous IgE-mediated allergy to rituximab, requiring a dose of 100 mg of prednisone for desensitisation after each chemotherapy administration.

The patient came to the emergency department with painful skin lesions which had developed 10 days after the last cycle and reported that, after receiving the treatment, lesions appeared first on his head and later on the rest of his body and oral mucosa. In addition, he had a fever of 38.6°C. He had no other accompanying systemic symptoms.

A physical examination of the patient revealed lesions on the head, trunk and extremities. These lesions had a necrotic centre and an inflammatory halo of a erythematous-violaceous colour (Fig. 1), interspersed with more vesicular lesions. Clustered vesicles and enanthem on his hard palate were seen in the oral mucosa (Fig. 1). The rest of the examination did not identify any other abnormalities or pathological lung sounds, and there was no palpable lymphadenopathy.

A sample was taken from one of the vesicles with a swab for a herpes virus polymerase chain reaction (PCR), a posterior-anterior and lateral chest X-ray was taken, and a blood test with a complete blood count, serologies and biochemistry with a liver and kidney profile. In addition, a *punch* biopsy was performed for pathology and microbiology.

Clinical course

The blood test highlighted a C-reactive protein of 161 mg/L and a neutrophil count of 2,200/mL. No pleuroparenchymal changes were seen on X-ray. Histopathology study showed a vesicle with necrosis of large and probably multinucleated keratinocytes, as well as acute vasculitis changes (Fig. 2). No fungi or bacteria were isolated in the culture of the sample sent to microbiology. Varicella zoster virus (VZV) was amplified by PCR taken from one of the vesicles. The serology result was of negative IgM and positive IgG against VZV. In view of the above, the patient was diagnosed with disseminated herpes zoster (DHZ).

He was started on treatment with intravenous acyclovir 10 mg/kg every eight hours, after which he made good clinical progress. He remained afebrile and asymptomatic during his admission, with the skin lesions evolving to a crusty state and his leucocyte count recovering. It was decided to discharge the patient home with acyclovir 400 mg every eight hours until completing the 10 days of treatment.

Closing remarks

Reactivation of VZV causes herpes zoster. It manifests with the appearance of grouped, painful vesicles on an erythematous base which follows the distribution of a dermatome. When more than 20 vesicles are present outside the dermatome or when two or more dermatomes are involved, it is classified as DHZ. Sometimes the presentation of DHZ is atypical. Our patient had a diffuse, non-metameric distribution with a predominance of necrotic lesions. This atypical presentation of disseminated herpes zoster is characteristic of immunosuppressed patients.[1]

Thrombocytopenia, anaemia, leucopenia and elevated liver enzymes are abnormalities which can be found in patients with DHZ,[2] so it is important to monitor these variables during the patient's admission. Another possible complication is pneumonia, so all patients should have an imaging test.[3] However, the most common complications are postherpetic neuralgia and superinfection of the lesions.[2] PCR is the method of choice for diagnosing VZV before vesicle rupture. Our patient had an atypical distribution, with cephalocaudal direction of spread, characteristic of chickenpox. To differentiate between primary infection and reactivation of the virus, serological tests must be performed to establish whether the immune response is primary, mediated by IgM, or secondary, mediated by IgG.

The treatment indicated in immunosuppressed patients is intravenous acyclovir 10–15 mg/kg every eight hours adjusted to renal function. Administration should be started as soon as possible.[2] In conclusion, the presentation of DHZ can vary significantly in immunosuppressed patients and early treatment is essential to prevent serious complications. A high degree of suspicion should be maintained in the case of newly appearing necrotic and vesicular lesions accompanied by fever in patients with compromised immunity. Monitoring of haematological and biochemical parameters, as well as performing imaging tests, are essential for comprehensive management of these cases.

Right to privacy and informed consent

The patient's consent was obtained for this case report and the hospital's protocols on the treatment of patient information were followed.

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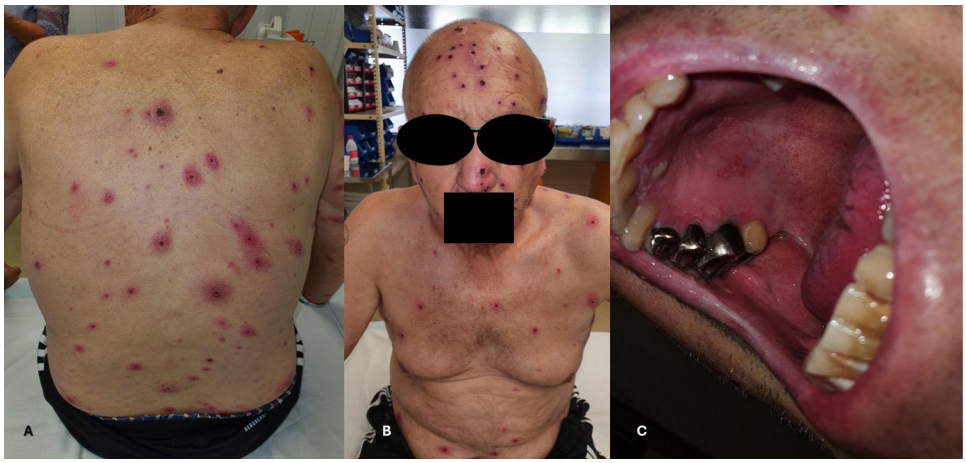

Figure 1. (A) Papules with a necrotic, crusty centre and an erythematous halo over the head and back. (B) Papules with a necrotic, crusty centre with an erythematous halo in the facial region. (C) Clustered vesicles on the hard palate along with enanthem.

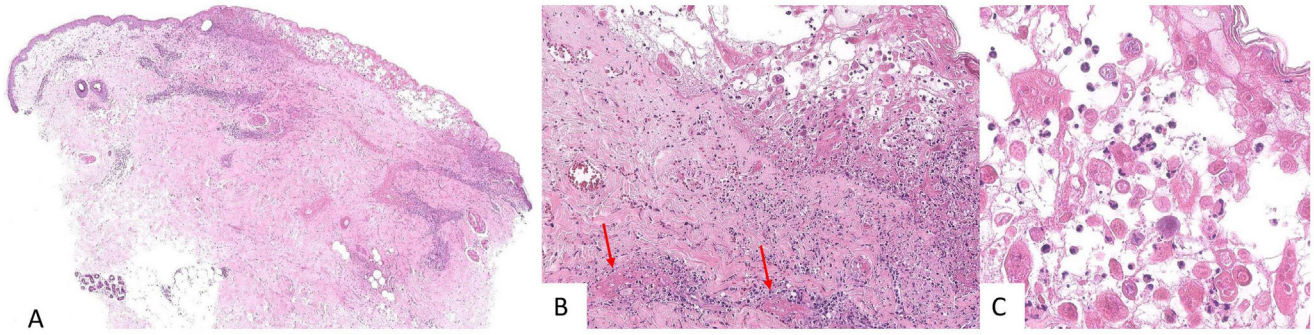

Figure 2. (A) (HE, 2×): at low magnification, a skin *punch* with epidermal lesion with blister formation can be seen, as well as a superficial perivascular inflammatory component. (B) (HE, 10×): at higher magnification, signs of vasculitis are identified, with fibrin deposition in the vascular walls (red arrows). (C) (HE, 20×): detail of the epidermal lesion where large multinucleated elements with intranuclear inclusions stand out. HE: haematoxylin-eosin staining.

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Declaration of competing interest

The authors have no conflicts of interest to declare.

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