CASE REPORT

Febrile ulceronecrotic Mucha-Habermann disease

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SUMMARY

The Mucha-Habermann disease is an inflammatory disease of the skin and is a variant of pityriasis lichenoides et varioliformis acuta. We describe the case of a 64-years-old woman who was admitted for erysipelas of the face. Despite treatment, evolution was marked by the appearance of a necrotising ulcerative area in the centre of the erysipelas associated with local oedema and headache. A skin biopsy revealed a pityriasis lichenoides et varioliformis acuta. Corticosteroids led to a rapid stabilisation of lesions, and after 6 months the patient shows only a small area of frontal hypopigmentation. The aetiology remains uncertain. There is no established standard treatment. We would like to draw attention of the medical and surgical specialists to this rare disease. The diagnosis should be considered in a necrotic lesion associated with rapid expansion of systemic and peripheral cutaneous signs. Diagnosis must be considered to avoid unnecessary debridement and extensive scars.

BACKGROUND

For this case, we wanted to draw attention to a rare disease whose diagnosis remains difficult. A multidisciplinary approach including dermatologist, anatomopathologist, oncologist and surgeon is necessary to treat this condition and also to manage its possible malignant transformation. Sometimes this pathology can be fatal. Diagnosis must be known to avoid unnecessary excessive debridement and residuals scars.

CASE PRESENTATION

A 64-years-old woman is admitted for an infection in the forehead area. The infection began 10 days ago and the patient mentions a possible spider bite. At first, she applied a cream containing urea on the lesion. Her medical history contains no dermatological diseases, but she has suffered from Crohn's disease with no event for 14 years and without treatment. Her medication is composed of an antidepressant (mirtazapine) and a proton pump inhibitor (rabeprazole).

At home, the initial treatment was composed of clindamycin (300 mg three times daily) and Fucicort cream (fusidic acid and betamethasone). The first clinical examination reveals an inflammatory wound with a diameter of 1 cm at the base of the scalp and frontal oedema extending to the eyes and nose.

INVESTIGATIONS

Her medical parameters were normal and laboratory tests revealed only a moderate inflammation (C reactive protein 15 mg/L normal value <5.0 mg/L). In front of the suspicion of erysipelas, antibiotic are

continued intravenously. The evolution was marked on the day of admission by the rapid extension of the lesion and the appearance of necrosis (figure 1). Antibiotic therapy is extended with vancomycin, meropenem and combination of acyclovir as an antiviral. The computed tomodensitometry did not reveal any pathological picture. Microbiology of the wound has detected penicillin-resistant *Staphylococcus epidermidis*. Despite combination therapy, necrosis (figure 2) extended and we noted the appearance of macula and papula away from the main lesion on the face and different parts of the body (back, abdomen and limbs). The palpation was painful but there was no sign of subcutaneous emphysema. The patient reported a headache, pruritus and a burning sensation. The general condition of the patient is preserved and its parameters are normal. She had no bowel disease symptoms. Given the lack of diagnosis and rapid progression of necrosis (figures 3 and 4), we performed a skin biopsy in different parts of the necrotic lesion and peripheral lesions. Histological examinations showed epidermal necrosis, the seat of many elements including inflammatory mononuclear cells, many neutrophils, eosinophils and a few red blood cells. The superficial dermis shows a lichenoid infiltrate invading the basal layer which shows apoptotic and necrotic keratinocytes. There are also signs of leukocytoclastic vasculitis (figures 5 and 6). The histopathological report concludes with a *pityriasis lichenoides et varioliformis acuta* (PLEVA). The immunohistology found intradermal and intraepidermal lymphocytes CD8 and some macrophages.

DIFFERENTIAL DIAGNOSIS

At the first examination of the patient, our diagnosis of exclusion was a bacterial infection progressing

Figure 1 One day after admission.

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Figure 2 Day 2.

Figure 4 Day 4.

to necrotising fasciitis. This condition is subject to a high rate of mortality.¹⁻⁶ Diagnosis and early surgical treatment are essential to improve the prognosis and reduce sequelae.^{1 2 7-10} Among the risk factors, we found in our patient an advanced age and the presence of a wound. Although rare in the face,⁷ our case showed clinical signs of necrotising fasciitis but distinguished by the absence of fever, impaired general condition, subcutaneous emphysema (confirmed by the scanner). The diagnosis is not always easy and in case of doubt, the surgeon's experience and histological examination play an important role.^{3 4}

There is no standardisation of symptoms in febrile ulceronecrotic Mucha-Habermann disease (FUMHD) and necrotising fasciitis.

To assist in diagnosis, table 1 presents the main differences and similarities between FUMHD and necrotising fasciitis.

TREATMENT

Over the lack of response to treatment, on the day of the biopsy, systemic corticosteroids were started, intravenous methylprednisolone (120 mg) for 5 days relayed by oral form.

OUTCOME AND FOLLOW-UP

The follow-up was marked by rapid stabilisation of the lesions after 1 week and local improvement after 1 month. Her home medical treatment consisted of a regression therapy of methylprednisolone (Medrol) and ointment including hydrocortisone butyrate (Locoid Lipocream). Six months later, the patient presents only with a skin hypopigmentation area in the forehead (figure 7).

DISCUSSION

Although Mucha-Habermann disease is a severe variant of PLEVA, it differs by rapid and painful evolution associated with systemic signs. In the acute form, this inflammatory dermatosis is characterised by the appearance of a rash initially composed of macula, papula topped by scales. These papula become vesicular-haemorrhagic pustules progressing to necrosis and ulceration. The ulceronecrotic form is characterised by rapid progression of necrotic papules that coalesce into large patches ulcerated. In general, lesions predominate on the trunk, roots and the ends of the limbs, but whole of the body can be affected including the mucous membranes. Systemic symptoms in the literature includes: high fever,

Figure 3 Day 3.

Figure 5 Seven months after treatment.

Figure 6 H&E stain histological staining $\times 20$: biopsy specimen showing acute findings consistent with Mucha-Habermann disease.

Figure 7 H&E stain histological staining $\times 20$: biopsy specimen showing acute findings consistent with Mucha-Habermann disease.

neurological signs,⁵ asthenia, alteration of the clinical status, abdominal pain,^{5 6} diarrhoea,¹¹ interstitial lung disease,¹² cardiomyopathy,¹³ rheumatological manifestations,^{14 15} megaloblastic anaemia,^{6 16 17} and death.^{18–22} The causes of death are due to: pneumonia,¹⁶ pulmonary thromboembolism,^{18 23} cardiac arrest,²⁴ sepsis,^{20 22 23 25} hypovolemic shock²¹ and massive thrombosis of the superior mesenteric artery.²³ The patient often reports of burning and pruritus. Healing occurs after several weeks or months,^{26–28} and there are varioliform scars and hyperpigmentation or hypopigmentation areas. Biological analyses are not specific and may show an increase of leukocytes,^{15 29 30} the C reactive protein,^{15 19 22} liver enzymes,^{15 29} lactate dehydrogenase,¹⁹ sedimentation rate^{15 19 29} but also anaemia²² and hypoproteinaemia.¹⁷ In a recent article, 42 cases of FUMHD were found in the

literature.³¹ The majority of cases occur in children and young adults with an average age 27.4 years.^{32 33} In FUMHD or PLEVA histological signs reveal at the dermoepidermal, parakeratosis areas often associated with scales containing nuclei of neutrophils. There is moderate acanthosis with necrotic keratinocytes over the entire height of the epidermis. The basal layer is vacuolated with a marked lymphocytic exocytosis. The superficial dermis is oedematous and the seat of a diffuse inflammatory infiltrate lichenoid type (neutrophils and eosinophils). These elements also infiltrate the skin appendages and vessels of the middle and deep dermis, with signs of leukocytoclastic vasculitis. There are extravasated red blood cells with intradermal haemorrhages.³⁴ The diagnosis may be difficult when the lymphocytic infiltrate highlights atypical lymphocytes, posing a problem of differential diagnosis with mycosis fungoides or lymphomatoid papulosis.³⁴ The immunohistochemical analysis allows identification of inflammatory cells. Most studies indicate that the proportion of CD8 T cell predominates in the PLEVA^{35–38} and CD4 T cells predominate in the pityriasis lichenoides chronica (PLC).^{38 39} Several authors have published cases of PLEVA where there was a clonal rearrangement of the T cell receptor with an increased risk to develop lymphoma.^{40 41} The receptor gene rearrangement invite to a prolonged monitoring of patients.

Although the pathogenesis of PLC remains unclear, several hypotheses have been proposed. One of the dominant explanations in the literature is a possible reaction to an infectious agent.^{5 6 15 42} In our case, the spider bite could be the trigger. The possible causes include: *Toxoplasma gondii*,^{43–47} Epstein-Barr virus,^{48–51} HIV,^{52–54} cytomegalovirus,¹⁵ varicella-zoster virus,⁵⁵ parvovirus B19,^{56 57} *Staphylococcus*^{58 59} and the measles vaccine.⁶⁰ Some drugs appear to be the cause of the disease. For example, chemotherapy agents (Tegafur),⁶¹ oestro-progestogenic contraceptives,⁶² antihistaminic drug,⁶³ and some medicinal plants.⁶⁴ Some authors believe that the PLC is a T cells lymphoproliferative disorder, while others believe that it's an immune complex disorder.^{65–68} Owing to the large number of possible aetiologies, many treatments have been used and their effectiveness varies from one patient to another. Among the treatments oral antibiotic use is the most common. Several authors have published cases of complete healing with the use of tetracycline,^{58 69} erythromycin or^{70 71} ciprofloxacin.⁵⁹ It is important to note that the efficiency can range from complete remission to no improvement. Methotrexate is also effective in some patients with PLEVA⁷² or FUMHD.^{6 19 29 71 73} Finally, the efficient use of dapsone (antileprosy) has also been described.^{74–76} The use of phototherapy ultraviolet light A or B^{77–79} as well as psoralen

Table 1 The main differences and similarities between FUMHD and necrotising fasciitis

	FUMHD	Necrotising fasciitis
Local signs		
Pain	Common and moderate	Common and intense
Oedema	Rare (present in our case)	Frequent
Bullae/blisters	Rare (present in our case)	Frequent
Skin colour	Violet	Violet
Hypoesthesia	Rare	Frequent
Necrosis	Frequent	Frequent
Ulceration	Frequent	Frequent
Crepitations	Rare	Frequent with <i>Clostridium</i>
Bleeding	Present	Absent
Rash (macula—papula)	Frequent	Rare
Lividity	Rare	Frequent
Burning	Frequent	Rare
Pruritus	Frequent	Rare
Satellite lesions	Frequent	Absent
General signs		
Fever	Frequent	Frequent
Shock	Rare	Frequent
Blood analysis		
Leukocytosis	Frequent	Frequent
C reactive protein	High	High

FUMHD, febrile ulceronecrotic Mucha-Habermann disease.

+UVA treatment (PUVA) therapy^{80–83} have demonstrated their effectiveness, with a few cases of recurrence. Systemic corticosteroids are indicated in febrile ulcerative necrotic forms¹⁰ and associated with topical corticosteroids, they can decrease symptoms (pruritus and burning). In chronic forms, application of immunosuppressive ointment (tacrolimus) has proved its effectiveness. Effective treatment of ulcerative necrotic forms by oral administration of ciclosporin^{84–86} has been published. Treatment with inhibitor of tumour necrosis factor α has also been reported.³² Finally, some cases with important skin necrosis necessitate surgical debridement associated with a skin graft.

The choice of treatment depends on the severity of the pityriasis and the patient's age. Most of the time a combination of several treatments is necessary to control the disease. In chronic forms, minimally symptomatic, treatment is not always necessary but sometimes antibiotics, phototherapy or topical corticosteroids are needed. On the other hand, for ulceronecrotic forms, antibiotics and corticosteroids must be started with or without PUVA (except children). In case of resistance to the first treatment, the use of dapsone, methotrexate or ciclosporin may be necessary. In children, the risk–benefit ratio must be taken into account. With this case, we wanted to draw attention to a rare disease whose diagnosis remains difficult. A skin biopsy is the best way to confirm the PLC and monitor, but there is no definite guideline for treatment. A multidisciplinary approach including dermatologist, anatomopathologist, oncologist and surgeon is necessary to treat this condition and its possible malignant transformation. Sometimes this pathology can be fatal. Diagnosis must be known to avoid unnecessary excessive debridement and subsequent scars.

Learning points

- All suspicious skin lesions of the FUMHD requires a biopsy.
- Several treatments is necessary to control the disease.
- Diagnosis must be known to avoid unnecessary excessive debridement.

Competing interests None.

Patient consent Obtained.

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