

# Purpuric pityriasis rosea in patients with anorexia nervosa

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## Abstract

Pityriasis rosea (PR) is an exanthematous disease associated with the endogenous systemic reactivation of human herpesvirus (HHV)-6 and/or HHV-7. Variants of PR characterized by atypical

lesion morphology, eruption course, and severe symptoms have already been described, but their prevalence is probably underestimated. We report herein two patients suffering from anorexia nervosa (AN) who developed a very rare form of purpuric PR. So far, no cases of PR in AN patients have been reported in the literature.

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## Introduction

Pityriasis rosea (PR) is an exanthematous disease associated with the endogenous systemic reactivation of human herpesvirus (HHV)-6 and/or HHV-7.<sup>1</sup> Although classical presentations are well documented, atypical forms – including those with unusual lesion morphology, prolonged duration, or more intense symptoms – are likely under-recognized.<sup>1,2</sup> In this report, we present two cases of purpuric PR occurring in patients diagnosed with anorexia nervosa (AN), a combination not previously described in the existing medical literature.

In AN, impaired cell-mediated immunity may favor the endogenous systemic reactivation of HHV-6 and/or HHV-7, causing PR. Furthermore, an increased level of vascular cell adhesion molecule 1, interleukin 6, and tumor necrosis factor- $\alpha$  has been demonstrated in AN patients, which may be responsible for their increased vascular dysfunction.

## Case Reports

### Case 1

A 17-year-old male presented with a 3-week history of an asymptomatic skin eruption preceded a few days earlier by a sore throat and generalized arthromyalgia. Medical history is remarkable for AN, which started three years earlier. He did not take any medication and was on a program of behavioral psychotherapy for AN. Physical examination showed maculopapular, purpuric lesions of a few millimeters in diameter, sometimes covered with fine scales, coalescing to form annular lesions on the cleavage lines of the trunk (Figure 1 A,B). An erythematous-purpuric scaly plaque (herald patch) on the abdomen had preceded the eruption. Punctate hemorrhages were present on the oral mucous membrane. There was neither lymphadenopathy nor hepatosplenomegaly. Routine blood investigations were normal. The calibrated quantitative real-time polymerase chain reaction (CQ-PCR) detected Epstein-Barr virus DNA (215,000 copies/mL) and HHV-6 (320 genome equivalents/mL) plasma viremia, whereas HHV-7 and cytomegalovirus DNA were not detected in plasma.

### Case 2

A 27-year-old female presented with a 2-week history of a severe itchy eruption over the trunk. The eruption was preceded by a few days of headaches and difficulty concentrating. She had been suffering from AN and had been undergoing psychotherapy

for five years. She was not taking any drugs. Physical examination showed an erythematous macular and purpuric eruption and widespread scratching lesions over the trunk (Figure 1C). Routine laboratory investigations were normal, and no virus was found in the plasma by CQ-PCR.

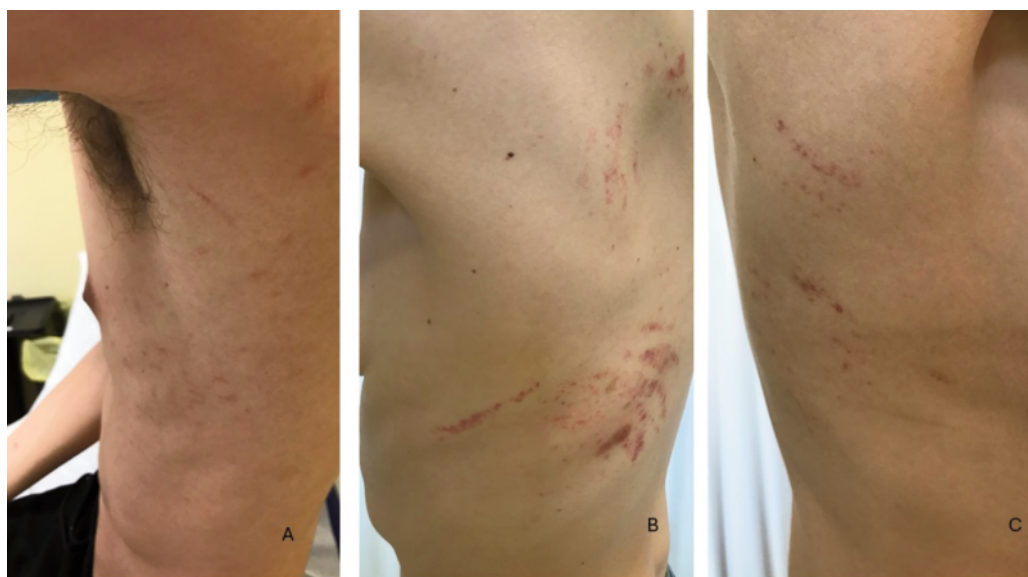
## Discussion and Conclusions

Histopathology of a skin biopsy showed *foci* of spongiosis and a perivascular infiltrate of lymphocytes in both patients, with prominent erythrocyte extravasation in the papillary dermis (Figure 2). A diagnosis of purpuric PR was made in both patients.

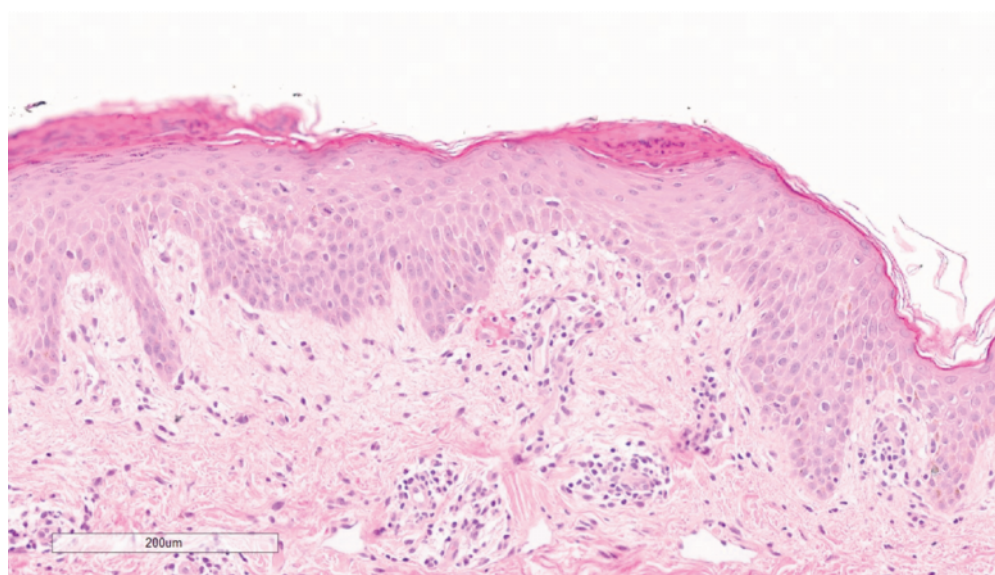
The eruption faded in 6 weeks without treatment.

As with other exanthems, in addition to the typical presentation of PR, atypical forms have also been reported.<sup>1,2</sup> Purpuric PR was first described by Hartman in 1944,<sup>3</sup> and only a few cases have been documented since then.<sup>4-6</sup> In purpuric PR, the erythrocyte's extravasation into the papillary dermis is significant and gives rise to the purpuric morphology. The presence of a "curtain theatre" pattern and the absence of histopathological signs of true vasculitis, namely fibrinoid degeneration, neutrophilic infiltrate, and nuclear dust, differentiates this form from pigmented purpuric dermatosis and cutaneous vasculitis.<sup>3</sup>

AN is a psychiatric disorder characterized by malnutrition and



**Figure 1.** A) Purpuric PR in the first reported patient: maculopapular, purpuric lesions of a few millimeters in diameter, sometimes covered with fine scales, coalescing to form annular lesions on the cleavage lines of the trunk ("theatre curtain distribution"); B, C) erythematous macular-purpuric eruption and widespread scratching lesions over the trunk in the second described patient with purpuric PR.



**Figure 2.** Histopathology shows an epidermis with patchy parakeratosis, irregular acanthosis, and mild spongiosis. In the papillary dermis, a perivascular inflammatory infiltrate and extravasated erythrocytes are visible (hematoxylin and eosin, 20x).

is frequently associated with medical complications. Cell-mediated immunity is usually altered in AN, as reflected by lymphocyte subset counts and the response to delayed hypersensitivity tests; leukocyte, lymphocyte, and T-cell counts have been shown to be depleted in AN patients.<sup>4</sup> T-lymphocytes correlate negatively with body mass index and are critically low in patients with severe malnutrition.<sup>7</sup> However, the consequences of these changes remain controversial regarding the susceptibility to infections.<sup>7</sup> In AN, the impaired cell-mediated immunity may favor the endogenous systemic reactivation of HHV-6 and/or HHV-7, causing PR. A decreased ability of the immune system to control other latent infections, such as Epstein-Barr virus (EBV),<sup>8,9</sup> has been hypothesized to be responsible for other AN comorbidities like chronic fatigue syndrome, celiac disease, rheumatoid arthritis, and other autoimmune diseases.<sup>8,10</sup> Furthermore, an increased level of vascular cell adhesion molecule 1, interleukin 6, and tumor necrosis factor- $\alpha$  has been demonstrated in AN patients, which may be responsible for their increased vascular dysfunction.<sup>11</sup> Microvascular damage may, therefore, be the basis for the greater presence of erythrocyte extravasation into the papillary dermis, which is the histopathological hallmark of the purpuric PR.

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