

focal B (CD20+) and dispersed T (CD45RO+) lymphocytes. CDw75 antibody, which stains germinal centre B lymphocytes, was negative. Ki-67 was negative. Kappa chain-positive lymphocytes and lambda chain-positive lymphocytes were observed almost equally. The lesion has shown no recurrence for at least 3 years after the surgical removal.

DISCUSSION

APACHE has been reported to arise mostly in childhood, and to present as unilateral eruptions of multiple angiomatic papules, mostly on acral sites of the hands and feet (1–4). Histologically, APACHE has been documented to correspond to pseudolymphoma (1–4). Immunohistochemical examination of infiltrating lymphocytes did not reveal any monoclonal proliferation of T or B cell subsets nor aberrant expression of surface markers (1–4). The present case is regarded as belonging to the category of APACHE in both histological and clinical features, except for the site of the lesion.

As regards the term “APACHE”, the disorder has not shown any histological features of angiokeratoma (1–4), with the exception of one case of a lichenoid reaction (2). It arises not just in childhood (1–4) but

also in adulthood (3), and is seen not just on the digits (1–4) but also on the back (3), and, as in the present case, on the wrist. The term “APACHE” does not therefore seem suitable for the condition. Recently, the terms “small lymphoid papules of the extremities” (3) and “small papular pseudolymphoma” (3) have been put forward for the condition. A kind of mechanical stimulation might be a factor inducing the condition, as suggested by the acral localization of lesions in most cases of this skin disorder.

REFERENCES

1. Ramsay B, Dahl MCG, Malcolm AJ, Wilson-Jones E. Acral pseudolymphomatous angiokeratoma of children. *Arch Dermatol* 1990; 126: 1524–1525.
2. Hara M, Matsunaga J, Tagami H. Acral pseudolymphomatous angiokeratoma of children (APACHE): a case report and immunohistological study. *Br J Dermatol* 1991; 124: 387–388.
3. Kaddu S, Cerroni L, Pilatti A, Soyer HP, Kerl H. Acral pseudolymphomatous angiokeratoma: a variant of the cutaneous pseudolymphomas. *Am J Dermatopathol* 1994; 16: 130–133.
4. Ito K, Fujiwara H, Ito M. Acral pseudolymphomatous angiokeratoma of children (APACHE) (in Japanese). *Hifubyo Shinryo* 2000; 22: 979–982.

Prurigo Pigmentosa in Association with *Helicobacter pylori* Infection in a Caucasian Turkish Woman

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Sir,

Prurigo pigmentosa (PP) is a rare inflammatory dermatosis of unknown origin characterized by intensely pruritic papular eruption which resolves leaving gross reticulate hyperpigmentation. The majority of cases are young Japanese women (1–4). To date, only 17 cases have been reported outside Japan (2, 5, 6). This is a report on a case of PP associated with gastritis due to *Helicobacter pylori* in a young Caucasian Turkish woman.

CASE REPORT

An 18-year-old female presented with a 1-year history of intensely itching, recurrent papular eruption leaving gross reticular pigmentation on her back. The condition had not responded to previous treatments with antihistamines, antidepressants, sulfamethaxazole/trimethoprim, fluconazole, ciprofloxacin or topical cortico-

steroids. She had had no past or current medical problem except mild dyspepsia. On examination, there were erythematous papules, some of which were excoriated and ulcerated, on the background of hyperpigmented keratotic plaques distributed in reticular pattern covering the middle portion of the back and scapulas symmetrically.

A punch biopsy from a papule revealed orthokeratosis, spongiosis, intraepidermal vesicles with accumulation of neutrophils and eosinophils, focal vacuolar basal layer degeneration, perivascular lymphohistiocytic infiltrate, melanophages and upper dermal oedema. Direct immunofluorescence was negative for IgA, IgG, IgM and C3 deposits. With a diagnosis of PP, the patient was started on doxycycline, 200 mg daily. However, on the third day of therapy, she experienced a phototoxic reaction, which resolved with topical steroids, and her gastric complaints became more severe. Doxycycline was therefore discontinued. Laboratory

analyses, including full blood cell count, biochemical and serologic tests, were either negative or normal except the high titres of *H. pylori*-specific IgG and IgM antibodies. She was referred to a gastroenterologist, and antral gastritis due to *H. pylori* was diagnosed by gastroscopic biopsy.

Gastritis was treated with 14-day therapy using clarithromycin, omeprazole and aluminium hydroxide gel. At the end of therapy, *H. pylori*-specific IgM antibody levels gradually decreased. Interestingly, pruriginous papules disappeared, leaving residual reticular pigmentation, and pruritus resolved completely during eradication treatment. No recurrence was observed within the following 6 months.

DISCUSSION

PP is a peculiar inflammatory dermatosis which has an active phase consisting of erythematous and pruriginous papules and a resolution phase characterized by a marbled-like reticulated pigmentation. The frequency of the initial cases from Japan suggested an ethnic predisposition or environmental cause (1, 2). Some cases seemed to be caused by exogenous factors, including contact allergens such as chromium, trichlorophenol and para-amino compounds, and bismuth subsalicylate ingestion (2, 4, 5). It has recently been suggested that ketonemia resulting from diabetes mellitus, fasting, dieting or pregnancy may play a significant role in the pathogenesis (3). However, other reported cases of PP have had no apparent association with endogenous or exogenous factors (2, 5, 6). PP is typically unresponsive to systemic or topical corticosteroids.

Several treatment regimens have been successful, including dapsone, sulfamethoxazole, potassium iodide, minocycline, doxycycline and sun exposure (2, 5). *H. pylori* is the major cause of gastritis and plays a key role in the etiology of peptic ulcer. Based on an increasing number of reports, a possible relationship between *H. pylori* infection and certain dermatological diseases, particularly rosacea and chronic urticaria, has been proposed (7). A pathogenic correlation between prurigo

and *H. pylori* infection has also been suggested recently, as eradication treatment had led to improvement of skin lesions (8, 9). In the current case, clarithromycin and/or omeprazole might have induced resolution of the skin lesions, probably by *H. pylori* eradication. However, the association with *H. pylori* infection may be purely coincidental. As the cases of PP are usually responsive to antibiotics such as minocycline, doxycycline or sulfamethoxazole, clarithromycin used in eradication therapy may also have similar effects, irrespective of the effects on *H. pylori*. Macrolide antibiotics, having both anti-inflammatory and antibacterial effects, have recently been demonstrated to be beneficial in patients with PP who were previously unresponsive to other drugs (10). The present case suggests that macrolide antibiotics deserve consideration as an alternative treatment for PP.

REFERENCES

1. Nagashima M. Prurigo pigmentosa. Clinical observation of our 14 cases. *J Dermatol* 1978; 5: 61–67.
2. Schepis C, Siragusa M, Palazzo R, Cavallari V. Prurigo pigmentosa: a misdiagnosed dermatitis in Sicily. *Cutis* 1999; 63: 99–102.
3. Teraki Y, Teraki E, Kawashima M, et al. Ketosis is involved in the origin of prurigo pigmentosa. *J Am Acad Dermatol* 1996; 34: 509–511.
4. Tani T, Kono T, Katoh J, et al. A case of prurigo pigmentosa considered to be contact allergy to chromium in an acupuncture needle. *Acta Derm Venereol* 1991; 71: 66–67.
5. Yanguas I, Goday JJ, Gonzalez-Guemes M, et al. Prurigo pigmentosa in a white woman. *J Am Acad Dermatol* 1996; 35: 473–475.
6. Gurses L, Gurbuz O, Demircay Z, Kotiloglu E. Prurigo pigmentosa. *Int J Dermatol* 1999; 38: 924–925.
7. Rebora A, Drago F, Parodi A. May *Helicobacter pylori* be important for dermatologists? *Dermatology* 1995; 191: 6–8.
8. Neri S, Ierna D, D'Amico RA, et al. *Helicobacter pylori* and prurigo nodularis. *Hepatogastroenterology* 1999; 46: 2269–2272.
9. Ohtsuka T, Yamakage A, Yamazaki S. A case of prurigo and lichenified plaques successfully treated with proton pump inhibitor. *J Dermatol* 1999; 26: 518–521.
10. Yazawa N, Ihn H, Yamane K, et al. The successful treatment of prurigo pigmentosa with macrolide antibiotics. *Dermatology* 2001; 202: 67–69.