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Dr. Messaritakis and co-workers comment:

We have read with interest the letter from Dr. Gaisin and co-workers, and in reply would like to make some remarks.

Leiner's disease, as described initially by Leiner, is a severe condition with a rather standard pattern of symptoms and a fatal outcome if left untreated. Our cases have no clinical similarity with Leiner's disease except for the generalized seborrhoea.

On this occasion we should like to describe briefly the results of laboratory investigations, which were not mentioned in our paper. Complement assays have been done and were found to be within normal limits. Furthermore, IgA, IgM, IgG, and IgD immunoglobulins were normal except for 2 cases complicated by severe staphylococcal infection in which increased IgM was found. Haemoglobin and haematocrit values were found to be below normal levels in 20 patients. After detailed haematological and biochemical investigation anaemia was classified as normocytic, slightly hypochromic, noniron deficient, probably due to mild or severe skin infection. Decreased haemoglobin and haematocrit values were restored to normal 20 to 30 days after the start of treatment. Skin cultures have been taken from the more severely affected areas.

Atopic dermatitis or psoriasis was not traced in the family histories of our patients, but we cannot anticipate the occurrence or not of various dermatoses in these patients for the next 5–15 years. Anyway, in follow-up periods of from 4–27 months there were no recurrences.

We should not like to argue the various opinions on the relation between diaper dermatitis and predisposition to atopic dermatitis, psoriasis, or seborrhoea because it seems unpractical, as we should not easily come to an end.

We would stress here that most of our patients were referred to us because of failure of various treatments applied by both paediatricians and dermatologists. This had caused great anxiety to the parents. Moreover, at least 2 of the patients were brought with severe staphylococcal infection threatening their lives.

In conclusion, whatever the mechanisms of the aetiopathology and the names given at times for this clinical entity, quick, safe, and successful treatment is still what really matters.

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Watery diarrhoea and ganglioneuroma

Sir,

We read with interest the case report by Swift, Bloom, and Harris (1975) of a 5-year-old girl with watery diarrhoea and a vasoactive intestinal peptide (VIP) secreting ganglioneuroma. In April 1975 we reported raised levels of VIP in a child with chronic watery diarrhoea and hypokalaemia (Udall *et al.*, 1975). The syndrome of intractable watery diarrhoea and hypokalaemia associated with pancreatic tumours was first described by Verner and Morrison (1958). Chronic watery diarrhoea and hypokalaemia has more recently been reported associated with pancreatic islet cell hyperplasia, neurogenous tumours, bronchogenic carcinomas, pheochromocytoma, and ganglioneuroma. The syndrome has now been associated with increased serum and/or tumour tissue levels of VIP (Bloom, Polak, and Pearse, 1973; Said and Faloona, 1975). There is evidence that the tumours may arise from cells that share a common derivation from the neural crest (Said and Faloona, 1975). Such cells (APUD) have a number of similar histochemical and ultrastructural characteristics (amine precursor uptake and decarboxylation) and also the capacity to secrete polypeptide hormones. There are many questions that remain unanswered since intractable watery diarrhoea and hypokalaemia in our patient and one reported by Pabst *et al.* (1969) was not associated with pancreatic hyperplasia or neoplasia. Serum VIP has also been shown recently to be increased in cholera (D. Nailand, personal communication). The significance of this peptide in diarrhoeal states awaits further investigation.

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