

## Medical Memoranda

### Norwegian Scabies (Crusted Scabies)

Norwegian scabies is an infrequently reported condition which usually affects institutionalized subnormal patients, especially male mongols (MacGuire and Kligman, 1960). Severely physically ill patients may also develop it (Morgan *et al.*, 1956; Dostrovsky *et al.*, 1956), and it has once been reported in a normal individual outside hospital (Martin Scott, 1957). It is a highly infectious condition caused by the common *Sarcoptes scabiei hominis* and not by the animal parasite as originally thought (Anderson, 1952). In contrast to ordinary scabies, up to 2,000,000 mites are probably present in the scales of any single patient (MacGuire and Kligman, 1960), and ward epidemics have been reported (Ingram, 1951; Wells, 1952). In view of the atypical and bizarre appearance of this type of infestation, and the unsuspected reservoir of infection that it may represent, it is thought worth while describing another case.

#### CASE HISTORY

A 76-year-old widow was transferred to a geriatric ward of Long Grove Hospital from another ward on April 11, 1961. She suffered from senile dementia with secondary depression. It happened that two days after her transfer "ordinary" scabies was discovered in the ward, and every one accordingly was examined carefully. The patient did not have scabies. Isolation and thorough disinfestation of the 11 affected patients was carried out, and after this a particularly careful watch was kept on the skins of all patients, and precautions against cross-infestation were tightened up. It should be noted that some scabies is endemic in the hospital.

On April 22 the patient developed redness and puffiness about the eyes, and weeping eczema of the neck, axillae, and antecubital fossae, which cleared with applications of 1% hydrocortisone ointment. In May she had a flare-up of this eczema, and in June she developed a generalized exfoliative dermatitis. She was seen by Dr. Ray Bettley, our consulting

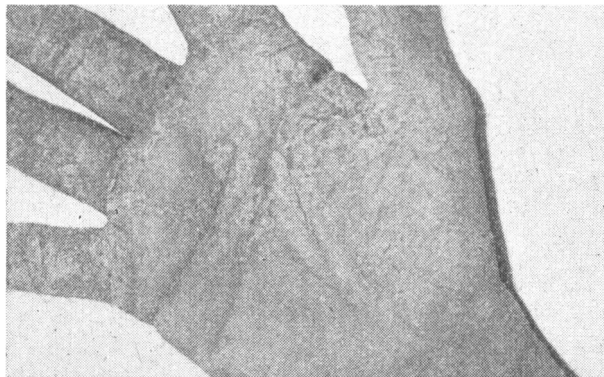


FIG. 1.—Showing crusting and fissuring of hand.



FIG. 2.—Showing scaling round the eyes.

dermatologist, who confirmed the diagnosis and at this point could find no evidence of infestation with scabies. Her skin responded slowly but well to oral prednisone in doses of up to 45 mg. a day, and although she periodically developed indolent boils her general condition improved to the extent that she was able to go for a month's holiday with her relatives.

She developed bronchopneumonia while on holiday, and after a period in a general hospital returned to her original ward on November 23, 1961. By February, 1962, there was again some exfoliation, and in March crusting and fissuring of her hands and feet, together with scaling round the eyes, appeared. These lesions were very irritant and painful, and are illustrated in Figs. 1 and 2. On April 14 she was again seen by Dr. Bettley, who diagnosed Norwegian scabies. This was confirmed by biopsy.

The patient was isolated and treated twice daily for three weeks with lotio crotamiton ("eurax" lotion). Dr. Bettley advised this, as he felt prolonged treatment was indicated and that benzyl benzoate was too irritant. The patient's skin cleared completely in two weeks, confirming the little-known efficacy of crotamiton as a sarcopticide, and refuting the theory that Norwegian scabies is predominantly a vitamin-A-deficiency disease and that success cannot be achieved by parasiticides alone (Hissard, 1956).

*Effect Upon the Other Ward Patients.*—Between April 13, 1961, and April 14, 1962, a total of 43 cases (exclusive of the original 11) of typical and atypical "ordinary" scabies occurred, several old women becoming infested twice, and a few thrice. This was a continual source of puzzlement and anxiety, since, as stated above, special precautions against cross-infestation were in force in this ward, and frequent inspections were made of all the patients. None of the nursing staff was affected, in contrast to previously reported epidemics (Wells, 1952). This may have been because, after treating infested patients, the nurses rubbed benzyl benzoate emulsion into their hands as a precaution. After the "Norwegian" case had been isolated there was a very sharp fall in the appearance of new cases, though the ward is not yet completely free of scabies.

#### COMMENT

It would seem likely from the history that the patient had unrecognized Norwegian scabies for some months, that an apparently classical exfoliative dermatitis was the first manifestation of this condition, and that it led to a ward epidemic.

Another suggestion is advanced, however. This is that the patient had "ordinary" exfoliative dermatitis, that scabies remained endemic in the ward (a thesis supported by the fact that sporadic cases have continued to occur since this patient was cured), and that she became infested later. Four of the six cases which I have found reported outside mental subnormality hospitals since 1950 had been receiving steroids for some time before developing the disease. Calnan (1950) emphasizes the absence of itching in this condition, and puts this forward as an aetiological factor. The above patient had intense prurigo throughout. It is therefore suggested that skin reaction to the mites may be lowered by steroid therapy, and that the syndrome of Norwegian scabies may result from this.

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## Sheehan's Syndrome in Association with Diabetes Insipidus

Sheehan's syndrome and diabetes insipidus are both well-known conditions, but the combination of the two is unusual and is of considerable physiological interest. Only 17 cases of this association have been reported in the literature. These have been reviewed by Evans (1960). A further case is recorded here, not only because of the rarity of the association but also because the failure to recognize the hypopituitarism during the first two years after the onset illustrates a common cause of diagnostic error.

### CASE REPORT

The patient, aged 30, was admitted to the obstetric department of the hospital for delivery at the end of a normal pregnancy. She was very obese, and weighed 103 kg. (16 st. 3 lb.). After the delivery there was atonic post-partum haemorrhage, which was so severe that she became unconscious and hysterectomy had to be performed to arrest the bleeding. Next day she drank 16 litres of water. She was forbidden to continue drinking such large quantities, but during the following night she was extremely thirsty and kept screaming that she would die unless she could get water to drink. Her condition distressed the other patients in the ward so much that they took her jugs of water to drink throughout the whole night. The acute phase of polydipsia gradually subsided, but from this time onwards she had gross polyuria and a correspondingly high fluid intake. Lactation failed to occur. She was extremely weak and could not even sit up in bed to wash herself. Her blood-pressure was 70/50. She was kept in hospital for the next five months, being quite unfit to be discharged home. During this time her weight fell to about 88.9 kg. (14 st.), her axillary and pubic hair fell out completely, and there was some loss of head hair.

The correct diagnosis was not made at this stage because the patient was so obese. Many physicians still believe the old concept of Simmonds's cachexia—that is, that the characteristic feature of hypopituitarism is emaciation without demonstrable cause—though Sheehan has given clear evidence since 1937 that significant loss of weight is an extremely rare sequel to post-partum necrosis of the pituitary.

After the patient was discharged from hospital she continued in the same very poor state of health, and was so weak that she could take only a few steps with assistance. She spent most of the next 18 months in bed. The systolic B.P. varied between 70 and 90. The intake of water and the output of urine were about 6 to 8 litres daily.

At the age of 32 she was readmitted to hospital for investigation. The most prominent clinical features were somnolence, a dry skin, and pallor. Laboratory investigations showed a slightly raised serum cholesterol level and a low basal metabolic rate. These findings led to an initial diagnosis of myxoedema, though the serum cholesterol level is characteristically high in genuine myxoedema and is often normal in cases of hypothyroidism of pituitary origin. However, there was also evidence of adrenal insufficiency: great physical weakness, hypotension at 95/70, and a low excretion of adrenal steroids. Further laboratory investigations gave the following results: glucose-tolerance test 70, 114, 119, and 75 mg./100 ml.; blood-sugar curve after protein-loading showed a decrease characteristic of adrenal insufficiency (Góth *et al.*, 1952), 88, 72, and 92 mg./100 ml.;

serum cholesterol, 260 mg./100 ml.; sedimentation rate, 20–27 mm.; blood count normal; urinary excretion of 17-ketosteroids, 2.3 mg./day; urinary excretion of 17-ketogenic steroids, 4.5 mg./day; B.M.R., minus 16%; urine specific gravity, 1000–1005; radiological measurement of the sella showed a normal area, 75 sq. mm.

The clinical condition and the laboratory findings finally led to the correct diagnosis—that is, that the patient was suffering from Sheehan's syndrome in association with diabetes insipidus. This diagnosis was confirmed by the excellent effects produced by substitution therapy. Treatment was begun with the daily administration of 50 mg. of cortisone, 75 mg. of thyroid extract, and 50 mg. of methyl androstenediol. This treatment produced a dramatic improvement. There was a rapid disappearance of the asthenia and somnolence, and she had a marked sense of well-being so that she was soon able to undertake her ordinary work again. Her blood-pressure rose to 140/80.

In the six years since this therapy was instituted she has remained very well and is able to work normally. The diabetes insipidus is basically less severe, but varies according to the treatment being given. The output of urine is 2 to 3 litres a day when all therapy is stopped experimentally, and rises to 5 to 7 litres a day when she is taking cortisone but no antidiuretic. The polyuria is well controlled by the injection of "pitressin" tannate twice a week. On the day of the injection the urinary output falls to less than 1 litre a day, and for the next two days it is 2 litres a day.

### DISCUSSION

It is very difficult to explain how, in rare cases, diabetes insipidus can occur after severe post-partum necrosis of the anterior pituitary. The posterior lobe is seldom involved, and there is no necrosis in the hypothalamus, which is the probable source of the posterior pituitary hormones.

In experimental studies on animals the presence of the anterior lobe seems to be necessary for the development and continuance of diabetes insipidus. Thus removal of the posterior lobe alone causes diabetes insipidus. On the other hand, removal of the anterior and posterior lobes together does not do so; there is an initial polyuria and polydipsia, but this disappears in about 24 to 36 hours, when the effects of the loss of the anterior lobe become manifest.

When women have surgical hypophysectomy for carcinoma of the breast, about 50% of them develop mild diabetes insipidus (Lipsett and Pearson, 1957). This continues while cortisone therapy is being given, but disappears if the cortisone is omitted (Ikkos *et al.*, 1955; Baron *et al.*, 1958). The mechanism of this phenomenon is not yet fully explained, but Dingman *et al.* (1958) suggest that cortisol may have an inhibitory effect on the secretion of antidiuretic hormone.

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The British Council's *Annual Report 1961–1962* shows that of the 7,522 overseas visitors in Britain assisted by the Council 1,030 were studying medicine. Of the others 2,198 studied education, 1,717 science and technology, 1,535 social science, 864 arts and humanities, and 178 miscellaneous subjects.