serum cholesterol was 305 mg. per 100 ml.; serum albumin 5 g. and globulin 3.5 g. per 100 ml.; albumin/globulin ratio 1.43:1. Liver-function tests showed serum colloidal gold reaction 3+, gamma globulin (zinc sulphate) 8 units, thymol turbidity 4 units, and thymol flocculation +.

Pituitary snuff immediately controlled her polyuria and allowed her to sleep well at nights. After starting this treatment, however, she stated that her breathlessness was somewhat increased. Because of the hypochromic anaemia she was given iron, which resulted in some amelioration of the dyspnoea.

A year later her condition was unaltered and a further x-ray film of the chest showed no substantial change.

Comment

The diagnosis of diabetes insipidus cannot be in doubt, there being adequate evidence to exclude other causes of polydipsia and polyuria—that is, diabetes mellitus, renal disease, and hysterical polydipsia. The only other discoverable lesions were those in the lung, manifested by exertional dyspnoea and radiographic changes.

Spillane (1952) summarized fully the literature relating to this combination of lesions. Following him the diagnoses to be considered are sarcoidosis, tuberculosis, xanthomatosis, tuberous sclerosis, syphilis, honeycomb lungs, and bronchogenic carcinoma.

The first of Spillane's four cases was an undoubted example of sarcoidosis, and this diagnosis could not be excluded in his Case 4. The other two cases were considered clinically to be examples of xanthomatosis. Necropsy in Case 3 later showed, in the hypothalamus and the lungs, the changes of eosinophilic granuloma which shares a morphogenesis in common with Hand-Schüller-Christian and Letterer-Siwe diseases, all three being secondary xanthomatoses associated with progressive reticular hyperplasia. It is therefore interesting to note that one of Cates and Garrod's (1951) cases of diabetes insipidus occurred in a patient with xanthomatosis and honeycomb lungs who also developed an eosinophilic granuloma of bone.

Dennis and Rosahn (1951) reported the case of a man aged 30 with diabetes insipidus and cystic lungs. They concluded from the necropsy findings that it represented a granulomatous disease involving the reticulo-endothelial system.

In the present case sarcoidosis does not appear to be the most likely diagnosis. Spillane (1952) collected from the literature 18 cases of sarcoidosis associated with diabetes insipidus. In none were the lesions confined to the pituitary region and the lungs. Similarly his own Case 1, which was an example of sarcoidosis, had an initial characteristic uveo-parotitis with facial palsy. His Case 4, in which no certain diagnosis was reached, and in which sarcoidosis could not be excluded, showed lesions in these two situations only. In this respect it is similar to the present case in which the Mantoux test (positive to 1:1,000 O.T.) is rather against the diagnosis of sarcoidosis. In addition, the dyspnoea was more intense than might be expected in a case of sarcoidosis with similar radiographical findings.

If we adopt a histological definition of sarcoidosis, as does Scadding (1950), diagnosis must await confirmation by biopsy. In the present case no superficial glands were available for biopsy and, owing to her commitments at home, the patient was unwilling to accept admission to hospital for liver biopsy.

Although, therefore, it is not possible definitely to exclude sarcoidosis, a granulomatous or xanthomatous process seems by exclusion to be a more likely diagnosis.

It is possible that when lung changes and diabetes insipidus occur together as the only discoverable disease they constitute a syndrome the pathological basis of which is a granuloma of some as yet undetermined and possibly variable type. The two conditions may, however, occur as a part of more widespread pathological processes such as those indicated in the papers cited.

Summary

The case is described of a woman aged 57 who had diabetes insipidus associated with a combination of reticulation and miliary mottling throughout both lung fields. A granulomatous or xanthomatous process is thought to be the most likely diagnosis, though sarcoidosis cannot be definitely excluded.

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PULMONARY ACARIASIS IN SPAIN AN ILLUSTRATIVE CASE REPORT

BY

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Considerable interest has been created in many parts of the world by the association that has been postulated by workers in Ceylon between hypereosinophilic respiratory disorders and pulmonary acariasis. Carter Wedd, and D'Abrera (1944) were the first to detect mites (Acarina) in the sputum of asthmatic patients with high eosinophilia. This discovery was confirmed by Soysa and Jayawardena (1945), who also suspected that the syndrome might result after exposure to inhalation of airborne mites. Further investigations in Ceylon by Carter and D'Abrera (1946) and by Soysa (1949, 1952a) have verified the previous findings. The conclusions of the Ceylonese workers that pulmonary acariasis may be associated with respiratory symptoms and massive eosinophilic reaction have been supported by reports from England (Hall, 1946), the Netherlands West Indies (Van der Saar, 1946), East Africa (Wilson, 1947), etc.

Since 1946 my attention has been engaged by the occurrence of mites in the sputum of certain patients with respiratory disorders associated with eosinophilic leucocytosis, and cases have been reported in Spain (Figueroa Taboada, 1946). I have been able, in my studies of this curious syndrome, to verify the ideas that have been so carefully and clearly presented by Soysa, and the results of my investigations are in complete agreement with his views concerning the three-stage clinical course discernible in many cases; the remarkable eosinophilic leucocytosis that is the most prominent and constant pathological feature of this syndrome; the variability of the pulmonary radiological appearances; the presence of mites in the sputum; the possibility of bronchial infestation by airborne mites; the ambient air importance of environment in relation to the acquisition of the disease; and the dramatic response to arsenical therapy, which is often associated with a Herxheimer reaction.

As an example of our findings in Madrid I cite a typical case history.

Case Report

A male clerk in a food store, aged 39, was investigated for asthma of seven months' duration. His personal and family history revealed no indication of any allergic antecedents. A blood count showed a total leucocyte count of

FIG. 1.—Eosinophilic reaction in a case of pulmonary acariasis. R=Routine treatment. S=Acetarsol treatment.

32,000 per c.mm., with 36% eosinophils. X-ray examination of the lungs showed fine linear striations superimposed upon the pathognomonic "ground-glass haze." Examination of the sputum by the special technique of Carter *et al.* revealed the presence of *Carpoglyphus* mites (Fig. 2).

FIG. 2.—*Carpoglyphus*.

Routine anti-spasmodic therapy for nine days produced no clinical response, while the eosinophils rose slightly. Acetarsol therapy was initiated thereafter, one tablet being administered twice daily for periods of five, three, three, and two days, separated by intervals of three, three, and two days, when the drug was suspended in order

to prevent cumulative effects. The patient developed status asthmaticus on the fourth and fifth days of arsenical treatment. Clinical control of the asthma was affected by the tenth day. The leucocytes gradually declined to 12,500, with eosinophils 11% on the twenty-second day, and to 10,000, with eosinophils 6%, on the thirtieth day after starting acetarsol therapy (Fig. 1). The patient has had no recurrence of asthma for fourteen months.

Comment

This case conforms in all its features to the typical cases recorded by Soysa and confirms his observations as regards the symptomatology, pathogeny, and therapy of this syndrome. I believe that pulmonary acariasis is a perfectly delimited pathological entity which should be separated from allied respiratory disorders.

Though generally regarded as a tropical disease, this syndrome may be much commoner than it may seem to be in temperate zones, such as Europe, where the causal Acarina are widely distributed—for instance, in stored food products. In England have been found the mites of the genera *Carpoglyphus*, *Glyciphagus*, *Cheyletus*, *Tarsonemus*,

and *Tyroglyphus* (Hughes, 1948). It seems possible, therefore, that careful examination of sputum in cases of eosinophilic respiratory disorders may disclose the presence of mites in countries that are not circumscribed within the tropical zone.

The laboratory procedure required for this purpose is long and laborious. Strict precautions have to be taken against accidental contamination of the specimens of sputum that have to be collected over a 24-hour period. The mass of material to be examined is so large that the work demands much patience and perseverance. The relatively small number of mites found in this quantity of sputum may appear insignificant, but it should be remembered that a few mites can cause disproportionate clinical disturbances in other acarine infestations, such as scabies. It may be argued that airborne mites may be prevalent in the respiratory passages without causing clinical or haematological manifestations. This objection has been met by the careful examinations made by Soysa (1949) of 44 control specimens of sputum taken from healthy persons and from patients suffering from non-eosinophilic respiratory diseases: not a single mite was found in any case, though 17 of these subjects were definitely exposed to the risk of inhaling airborne mites.

In a recent review of the aetiology and pathogeny of eosinophilic pneumonitis Soysa (1952b) discussed the various conditions that may present with somewhat similar clinical manifestations, and has suggested that, of the pathogenic agents that have been alleged to be responsible for eosinophilic pneumonitis, mites, metazoa, and fungi seem to come most under suspicion at the present time. He holds with the view that, despite the multiple aetiology and diverse pathological conditions that have been observed or suspected, the manifestations are linked by a common anaphylactoid pathogeny which is probably related to vascular allergy.

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