MEDICAL MEMORANDA

Inappropriate Secretion of Antidiuretic Hormone with Chronic Chest Infections

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British Medical Journal, 1974, 3, 785-786

Inappropriate secretion of antidiuretic hormone (ADH) occurs with several disorders of the lung, including carcinoma and tuberculosis. This is a report of a more uncommon association, with chronic inflammatory lung disease (*British Medical Journal*, 1972). The patient responded to treatment of the chronic chest infection with disappearance of the inappropriate ADH secretion.

Stormont and Waterhouse (1962) described the syndrome of inappropriate secretion of ADH in a patient with a pneumococcal pneumonia, and Utz *et al.* (1969) described it in a patient with cavitating pulmonary aspergillosis. Bryant (1972) has provided a third report in a patient with bronchitis and airways obstruction. All these diagnoses were based on clinical features only. Recently Rosenow *et al.* (1972) have described the syndrome of inappropriate secretion of ADH in three patients, two with viral pneumonia and one with a staphylococcal pneumonia. Plasma ADH concentration was raised in one of them. As the syndrome of inappropriate ADH secretion is well known in patients with carcinoma of the lung it is easy to forget that it can occur in other conditions, and we have been able to find reports of only six patients with chronic severe hyponatraemia associated with other lung infections. This report shows that it can also be treated successfully and may reverse when the underlying inflammatory lung disease improves.

Case Report

A 64-year-old man presented with a chronic chest infection which had been present for several months and which had failed to respond to conventional treatment with antibiotics. During this time he had a cough and lost half a stone (3.2 kg) in weight, had episodes when he produced green sputum, and had rigors with intermittent left pleuritic chest pain. Six days before admission the symptoms recurred and did not resolve when treated with tetracycline. He had lost his appetite and felt breathless on the slightest exertion. For 15 years his job had involved sharpening metal on a carborundum stone. He smoked 2-3 ounces (56-85 g) of tobacco rolled into cigarettes each week until four weeks before admission. He had a six-year history of chest infections in the winter, which responded rapidly to antibiotics and caused him no loss of work.

On admission he weighed only 6 st 11 lb (43 kg) and he was irritable and drowsy. He had several features of longstanding pulmonary disease: there was early finger clubbing, and he was

centrally cyanosed; the pulse was 136 beats/min and the jugular venous pressure was raised 4 cm; there were signs of fibrosis and collapse of the left lung with reduction in the size of the left chest and deviation of the trachea to the left; a coarse pleural rub was heard.

The chest x-ray picture showed collapse and consolidation of the left lung with cystic changes. Haemoglobin was 13.2 g/100 ml, W.B.C. 24,000/mm³, E.S.R. 12 mm/hr, blood urea 31 mg/100 ml, sodium 117 mEq/l., potassium 4.3 mEq/l., chloride 81 mEq/l., bicarbonate 27.4 mEq/l. The plasma cortisol was 22.3 mg/100 ml at midnight, and liver function test results were normal. The plasma osmolality at that time was 269 mOsm/l. and the urine osmolality 684 mOsm/l. ADH was detected in the urine at normal concentrations despite the very low plasma osmolality.

The initial clinical impression was that he might have a carcinoma of the lung with inappropriate secretion of ADH with an associated chest infection. A number of investigations showed, however, that this was not the case. Tomograms and a left bronchogram indicated a longstanding chest disorder, and bronchoscopy and open lung biopsy of the left lung showed only fibrous tissues. A lung scan showed no perfusion of the left lung, and the right lung contained emphysematous bullae with cysts. The chest infection on admission was found to be due to the pneumococcus and responded to penicillin. Tubercle bacilli were never found. He continued to produce large amounts of green sputum for several months and had several further courses of penicillin and tetracycline. The haemodilution due to water retention was treated by a restriction of fluid to 1 l./day. He soon became fully alert and gained 11 lb (5 kg) during the two-month period after water restriction was begun, and the serum electrolytes returned to normal. Six months after admission there was great improvement in the chest x-ray findings, and his exercise tolerance also increased so that he was able to walk half a mile (800 m) with no difficulty and could mow a lawn and go upstairs without stopping. A water load of two litres was handled normally. A blood sugar of 240 mg/100 ml was found at this stage, and he was given tolbutamide. Water restriction was therefore no longer necessary and he returned to work. He remained well one year after presentation.

Comment

The clinical features of inappropriate secretion of ADH are secondary to water intoxication, and the low serum sodium level is caused by haemodilution (Bartter and Schwartz, 1967). Patients become apathetic and irritable and after a stage of nausea and vomiting neurological symptoms predominate with convulsions and coma. The hyponatraemia is often profound, and the blood level of sodium is not altered by adding salt to the diet.

In these patients the serum osmolality is low, with a paradoxically concentrated urine. When the plasma osmolality is less than 283 mOsm/Kg, urinary ADH is normally undetectable, and the presence of ADH in the urine under these conditions is the diagnostic feature of inappropriate secretion of ADH (Miller and Moses, 1972). This was found to be so in our patient; thus there was no question about the diagnosis.

Finding a chronic inflammatory pulmonary lesion in this patient, in the absence of either a tumour or tuberculosis, suggests that the inappropriate secretion of ADH was caused in some way by the lung lesions. The source of the ADH here is not known, but it is possible that it comes from the lung. Alternatively the ADH could be of pituitary origin, with the defect in blood volume and the pressure in the left atrium (Rosenow *et al.*, 1972). The excess production was reversed after treatment for several weeks with antibiotics, so presumably the infected tissue was involved in the process.

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This finding indicates that it is important to treat patients with inappropriate ADH secretion with fluid restriction and antibiotics, as on occasions they will be found to have this type of treatable disorder.

We thank Dr. J. M. Holt under whose care the patient was admitted and Dr. Rhees, St. Bartholomew's Hospital, who carried out the urine ADH estimations.

Two Cases of Tuberculous Lymphoedema

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British Medical Journal, 1974, 3, 786

Tuberculous lymphoedema is rare because inguinal lymph nodes are not often affected. We report two cases of disseminated lymphatic tuberculosis, one of which presented with elephantiasis and the other with unilateral lymphoedema.

Case 1

A 35-year-old woman was admitted to hospital with a nine-month history of massive, non-pitting oedema of the feet and low grade pyrexia and complaining of pain and stiffness of the legs. On admission she had a fever of 99-100°F (37.2-37.8°C) and palpable cervical, supraclavicular, and inguinal lymph nodes, which were firm and matted. There was non-pitting oedema of the feet and ankles (fig. 1). Her total leucocyte count was 7,600/mm³ with neutrophils 65%, lymphocytes 28%, eosinophils 4%, and monocytes 3%. The E.S.R. was 100 mm in the first hour. The tuberculin test result was positive and x-ray examination of the chest showed tuberculous infiltration at the left apex. Lymph node biopsy showed chronic granulomatous inflammation with caseation necrosis compatible with tuberculosis. Direct smear and culture of the pus from the node was positive for *Mycobacterium tuberculosis*. The lymph nodes and pulmonary lesion regressed with antituberculous treatment but the lymphoedema still persisted after 10 months.

Case 2

A 24-year-old woman was admitted to hospital with a three-month history of pain and non-pitting oedema of the right leg, low grade pyrexia, weight loss, and malaise. On admission she had a fever of 100°F (37.8°C), the cervical, iliac, and superficial inguinal lymph nodes were palpable, and there was a discharging sinus in the neck. The right leg was swollen with non-pitting oedema (fig. 2). The liver was enlarged. Blood examination showed: W.B.C. 12,000/mm³ (neutrophils 80%, lymphocytes 18%, monocytes 2%), E.S.R. 85 mm/1 hr, serum bilirubin 0.9 mg/100 ml,

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FIG. 1—Case 1. Elephantiasis of the legs. FIG. 2—Case 2. Unilateral oedema.

serum alkaline phosphatase 3.1 Bassey-Lowry units, and SGOT 9 U/ml. Thymol turbidity was 11.5 units. X-ray examination of the chest showed bilateral tuberculous infiltration. The histology of the lymph nodes was compatible with the diagnosis of tuberculosis. Culture of the caseous material from the lymph nodes showed *M. tuberculosis*. Liver biopsy showed fatty changes with granulomas. After eight months of antituberculous therapy all the lesions improved except the lymphoedema.

Comment

Tuberculosis may present in various ways (Rab and Rahman, 1967). The two cases reported here show the mechanical effects of lymphatic obstruction by enlarged subinguinal and deep iliac tuberculous glands. Awareness of this manifestation of the disease is important in developing as well as in developed countries. In the former these cases would probably be diagnosed as filariasis while in the latter tuberculosis is now so uncommon that it might not be thought of.

We thank Professor N. A. Jaffery, professor of pathology, Jinnah Postgraduate Medical Centre, Karachi, and the photographic department of the hospital for their co-operation and help.

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