of a vein. It is a rare condition and was first described in 1928 by Harris of Toronto.

It may be seen in either the internal or external jugular vein (more commonly the latter), and appears as an abnormal spindle shaped swelling which is due to a developmental abnormality of the vein wall itself.

Such dilatations have been referred to as 'venomas', thereby likening the process to cystic hygroma arising in the lymphatic system in the neck.

It was possible in this case to establish a diagnosis of venous cyst by physical signs in the first place, later confirmed by X-rays. The swelling was of soft consistency and cystic, but cystic hygroma or lymph cyst could be excluded however, as it did not transilluminate. Any arterial connection was excluded by the absence of pulsation and bruit.

Pataro, Crosbie and Martinez Conde (1961) state that the one characteristic present in their series of four cases, was the increase in size of the tumour when the patient made an effort (e.g. performing the Valsalva manœuvre) or to a lesser degree on lying down. They mention three types of swellings in the neck which show these features as follows:

- (1) A tumour of the superior mediastinum. This can be demonstrated by a chest X-ray.
- (2) A laryngeal diverticulum. This can be excluded by laryngoscopy and by normal X-ray appearances of the larynx and trachea.
- (3) The neck venous tumour.

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MYXOEDEMA FOLLOWED BY ADDISON'S DISEASE AND DIABETES MELLITUS

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BERNSTEIN (1948) described a patient with diabetes mellitus, Addison's disease and hypothyroidism, but there was no evidence of hypopituitarism found at post-mortem. This rare combination of diseases associated with apparently normal pituitary function has also been reported in cases by Howard, Mills and Mathews (1952), by Crispell and Parson (1952), and by Christy, Holub and Tomasi (1962), the last two patients also having hypogonadism. Beaven, Nelson, Renold and Thorn (1959) reported a case of a woman who had been treated for 'incomplete hypothyroidism' since childhood and who

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The diagnostic feature in the present case was the remarkable way in which a swelling could be made to appear, by pressure on the neck in the supra-clavicular fossa, with the patient erect, and the slow subsidence of the swelling on withdrawing the hand. This manœuvre could be constantly repeated and proved the swelling to be a cyst communicating freely with the venous system in the neck.

If this sign occurs, it would seem to establish the diagnosis of phlebectasia.

Summary

A case of jugular phlebectasia is described, and the few accounts in the literature of this anomaly are quoted. The diagnosis is discussed and the clinical signs that were present are described.

As well as Valsalva's manœuvre, simple pressure low down at the root of the neck followed by sudden withdrawal of the hand may be sufficient to demonstrate this condition. It can be confirmed by needle aspiration and injection of radio-opaque material followed by X-ray immediately prior to operation.

My thanks are due to Dr. D. F. Street for the X-rays and angiography and to Dr. D. B. Richards for the pathological report on the specimen.

I also wish to thank Dr. F. J. Davis for the photographs.

later developed Addison's disease and diabetes mellitus. Two other cases have also been traced in the English literature (Breslaw, Lashof and Klein, 1953; Beaven and others, 1959), but in each case with the hypothyroidism following treatment of thyrotoxicosis.

The patient described here had myxoedema for several years before developing Addison's disease and later diabetes mellitus.

Case Report

M.F., a housewife, aged 52, first attended the Out-patients' department in 1951, complaining of two recent attacks of vomiting, diarrhoea and abdominal pain for which no cause could be found, although a barium meal

showed a small hiatus hernia and duodenal diverticulum. She attended again in 1954 with a three months' history of weakness, lack of energy, and loss of weight. She said that ten years previously she had been told that she had thyroid deficiency and she described her condition at that time in the following words: 'My brain was all slowed up, my speech was all thickened; I used to start a sentence and not be able to finish it because I could not remember. My skin was very dry and flaky. My face was so puffed up I could not open my eyes. My hair fell out: I was almost bald. I was very constipated. I was always icy cold. When I took the thyroid tablets a miracle happened; all this cleared up and I lost ten years in the next three months'.

She took thyroid extract from that time until her doctor stopped her doing so three weeks before this attendance, in case her symptoms had been caused by thyroid overdosage. Her symptoms had worsened during these three weeks.

On examination she was euthyroid, with a serum cholesterol of 210 mg./100 ml. There were no physical abnormalities, except absent pubic and axillary hair and sparse eyebrows. Her weight was only five pounds less than three years before. Her blood pressure was 130/110 mm. Hg. The mini-chest X-ray was normal and routine urine tests were negative, but the ESR (Westergren) was 23 mm./hour.

After re-starting thyroid extract she soon began to feel better. However, a month later she attended Out-patients' again, having just had another attack of vomiting and collapse, similar to those she had in 1951. This time her skin was very brown and she had a little buccal pigmentation. Her blood pressure was only 85/60 mm. Hg, and her ESR had risen to 34 mm./hour. Addison's disease was suspected, and she was admitted for investigation. The possibility of Simmond's disease was considered, but was not supported by the menstrual history, which was as follows: her periods began at 16 years old, but were irregular. At the age of 32 she had a miscarriage, which necessitated a D and C., and resulted in severe blood loss. However, her periods began again and two years later she became pregnant. She delivered normally at nine months and although she had a retained placenta the blood loss was not severe. She breast fed her child for six months. Her periods began again and continued irregularly until she had a menopause at the age of 42.

In the family history the only significant fact was that a brother had died in a diabetic coma.

Investigations confirmed that she was suffering from Addison's disease. The serum sodium was 121.5 mEq./l., potassium 5.7 mEq./l., chloride 104 mEq./l. Her plasma 17-hydroxycorticosteroids were zero and did not rise significantly after an infusion of 2 units of ACTH per hour for six hours (Dr. R. Bayliss). There was marked impairment of ability to excrete a water load. Her 17-ketosteroid urine excretion was 1.8 mg./24 hours.

Other findings were: blood cholesterol 175 mg./100 ml., blood urea 28 mg./100 ml., Hb. 92%, routine urine tests negative. Chest, abdominal and skull X-rays were normal.

After treatment with cortisone 25 mg. daily, desoxycorticosterone 100 mg. pellet implantations, 2 gm. added salt and thyroid extract 60 mg./day, she improved dramatically, losing all her fatigue and regaining a stone in weight in five months. Her skin became less pigmented.

During the next four years she was kept in reasonable health on cortisone 37½ mg. per day and thyroid extract 120 mg. per day. In 1958, the thyroglobulin precipitin test was negative. A year later she was admitted to

hospital suffering from an Addisonian crisis, precipitated by a febrile illness.

In May 1960, she was admitted again to hospital complaining of one month's weakness, fainting, thirst and polyuria, and two days' vomiting. She was weak and dehydrated and collapsed on trying to stand. A diagnosis of combined Addison's disease and diabetes mellitus was confirmed. The serum sodium was 112.5 mEq./l., potassium 5.5 mEq./l. chloride 86 mEq./l. The blood sugar was 540 mg./100 ml.

The urine contained 2% sugar with ketone bodies. Other significant results were: serum iron 57 µg./100 ml., calcium 9.2 mg./100 ml., inorganic phosphate 3.5 mg./100 ml. The plasma proteins, serum electrophoresis and flocculation tests were normal.

She was treated with intravenous fluids and cortisone. As her condition improved satisfactorily and her insulin sensitivity was uncertain, this drug was withheld until there was adequate cortisone replacement. Two days later, as she had a persistent high blood sugar and ketosis, she was given soluble insulin. The dose had to be raised gradually to 32 units twice a day before there was satisfactory control. Fludrocortisone was given to aid salt retention. After changing from soluble to lente insulin she had a number of minor hypoglycaemic reactions and the dose had to be decreased gradually until four weeks later it was stopped. The diabetes mellitus was then controlled by a low carbohydrate diet. A week later, following some diarrhoea in the night, she relapsed and had to be treated with intravenous fluids, hydrocortisone and oxytetracycline. It was again necessary to give insulin and to continue doing so for a week. She slowly improved and was discharged on a maintenance regime of cortisone 37.5 mg. daily, fludrocortisone 0.1 mg. daily, thyroid extract 120 mg. daily, 3 gm. added salt daily, and a 120 gm. carbohydrate diet. Recently her husband has died and she has taken up a part-time domestic job, which is fully within her capabilities.

Discussion

It is difficult to explain why this patient had this unusual combination of illnesses. There seems no doubt that she had myxoedema. She had been treated already for many years when first seen, but a retrospective diagnosis based on her description of her response when thyroid extract was first given seems justified.

In 1954 the clinical picture and investigations unequivocally showed Addison's disease; the two attacks of vomiting and diarrhoea with abdominal pain in 1951 suggest that she might have had some adrenocortical insufficiency three years previously. The impaired thyroid and adrenal function suggested hypopituitarism, which was supported by absent pubic and axillary hair. On the other hand the menstrual history did not support this diagnosis and the presence of heavy pigmentation favoured normal pituitary function. The involvement of an autoimmune mechanism seemed possible, but no thyroglobulin antibodies were demonstrated.

During her last admission to hospital the main problem was the immediate treatment of a woman suffering from combined Addison's disease and diabetes mellitus. Gittler, Fajans and Conn (1959), have pointed out that the development of diabetes mellitus in a patient with previously well controlled Addison's disease is characterized by an apparent

exacerbation of the adrenal insufficiency with return of anorexia, lassitude, loss of weight, and low blood pressure. Although patients with Addison's disease, treated with cortisone, are not usually hypersensitive to insulin, in this present case, as the patient's condition improved satisfactorily, and her insulin sensitivity was uncertain, the latter drug was not used immediately.

It is interesting that her diabetes is controlled now without insulin, despite the fact that she needed

insulin on her last admission and also during her subsequent relapse.

Summary

A patient is described who initially had myxœdema and later developed Addison's disease followed by diabetes mellitus.

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DISGUISED PERNICIOUS ANAEMIA

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THE diagnosis of classical Addisonian pernicious anaemia is straightforward. It may prove difficult when blood values are normal, as may happen when the disease presents with nervous system manifestations. This case, which showed transitory leucoerythroblastic anaemia, demonstrates how difficult the diagnosis may be, even when the patient is anaemic.

Case History

A woman of 68 was admitted to the Victoria Infirmary on 16.2.61. She had been tired for three years, and had lost 2½ stones in weight in that time. One year previously, immediately after her husband's sudden death, she had pneumonia, and had since then complained of weakness, cough and breathlessness. She lived alone and felt depressed. For the past year she had eaten little except porridge, bread and butter and tea.

Although the patient was frail and slow of thought, she was nervous and hyperactive. There was marked sacral and leg oedema, and a sinus tachycardia of 116/min. There were signs of fluid at both lung bases, particularly the right. The liver was palpable 2 in. below the costal margin but the spleen could not be felt. No abnormality was detected in any other system. Her temperature did not exceed 99.2°F. There was no urinary abnormality.

An X-ray of the chest showed consolidation and slight collapse of the right base, with some free fluid at both

bases. Electrocardiographic complexes were of low voltage with flattening of the T waves. On admission hæmoglobin was 6.2 g/100 ml.; packed cell volume 21% MCHC 29.5%. The total nucleated cell count was 13,100 per c.mm., of which 7,900 were white cells. (Neutrophils 73%, lymphocytes 14, monocytes 5, neutrophil metamyelocytes 7, neutrophil myelocyte 1.) Most of the nucleated red cells were orthochromic normoblasts, but a few were polychromatic. The nuclei of some were normal, others lobulated and distorted. The periodic acid Schiff reaction was negative. There was moderate anisopoikilocytosis, marked polychromasia, and a number of hypochromic microcytes.

Pleural aspiration was performed and subsequent culture and animal inoculation were negative for *M. tuberculosis*. Treatment was commenced with crystalline penicillin, mersalyl and chlorothiazide, and ordinary ward diet was given.

Five days later nucleated red cells had almost disappeared. Total nucleated cells numbered 8,600 and were practically all white cells, which were fully mature. Hæmoglobin had risen to 7.6 g., and the reticulocyte count was 13.2%. Sternal marrow aspiration showed erythroid hyperplasia, and many megakaryocytes. Erythropoiesis was normoblastic and macronormoblastic, with predominant early and intermediate forms. A few very early forms had fine nuclear structure, and appeared to be transitional between normoblasts and megaloblasts. Myeloid granulopoiesis was active, and