soon after being incurred responded well, while the prognosis was poor in the 16 cases which had remained untreated for periods extending from weeks to years.

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Diabetes Mellitus with Acute Polyneuritis

A true symmetrical polyneuritis is rare in diabetes mellitus (Hirson, Feinmann, and Wade, 1953). In the following case a symmetrical polyneuritis developed rapidly in a patient with recently stabilized diabetes.

CASE REPORT

A bus conductor aged 60 was discovered to have mild diabetes in September, 1954. The presenting symptoms were pruritus and a mild urethritis of about six weeks' duration. There was no relevant past or family history. Examination showed no abnormality except for obesity (weight 12 st. 7½ lb.—79.6 kg.) and slight hypertension (B.P. 180/100 mm. Hg). The knee- and ankle-jerks were present and vibration sense was normal in the legs. The diagnosis was confirmed by a glucose-tolerance test. (Capillary blood sugars:—fasting, 141 mg.; at half-hour intervals after 50 g. of glucose by mouth: 290 mg., 232 mg., 219 mg., 175 mg. per 100 ml.) Treatment was started with a reducing diet containing 120 g. of carbohydrate daily. His symptoms were relieved and his diabetes was easily controlled without insulin.

On December 2 he was admitted to Addenbrooke's Hospital under Dr. Leslie Cole, as an emergency, complaining of severe weakness of the legs, associated with pain and numbness, which had developed rapidly over the previous two days. There was no history of recent infection, sore throat, or alcoholism.

On examination he was found to have signs of a bilaterally symmetrical peripheral neuritis affecting the legs and to a less extent the arms. He was unable to walk. Voluntary movements in the legs were very weak in all muscle groups, particularly the dorsiflexors of the feet. Movements were severely ataxic. The knee- and ankle-jerks were absent. Plantar responses were flexor, but later became difficult to obtain, with occasional equivocal extensor responses. Vibration sense was absent at the knees and ankles. Position sense was grossly impaired in the toes. Sensation to light touch and pinprick was impaired over the feet and lower legs. The calf muscles were tender to deep pressure. Similar but less extensive signs were present in the arms. The abdominal reflexes were absent. The cranial nerves were normal. No other abnormal signs were found.

His mental state was good, and, although he later became depressed, he developed no signs of dementia. He had lost 10½ lb. (4.8 kg.) in weight. The cardiovascular system was normal (B.P. 160/90 mm. Hg). His diabetes appeared well controlled (fasting blood sugar 150 mg. per 100 ml.). His pulse rate was 80 a minute on admission. He later developed a persistent tachycardia (90–100 a minute). His temperature was 97.8° F. (36.6° C.), and he remained afebrile throughout his illness except for occasional spikes of fever during later episodes of intercurrent infection.

Lumbar puncture on December 8 was normal except for a raised protein (over 200 mg. per 100 ml.) in the cerebrospinal fluid with a practically normal cell count (4

lymphocytes per c.mm.); W.R. negative. Other investigations, including a full blood count, throat swab, blood W.R. and Kahn, examination of the urine for porphyrins, and x-ray pictures of the chest and lumbar spine, showed no significant abnormality.

His condition rapidly deteriorated. Paraesthesiae in the legs became intensely painful. Light pressure on the legs was enough to cause pain. He developed severe abdominal pain, constipation, and abdominal distension. Within one week of admission he had developed an almost complete flaccid paralysis of the limbs. He had a bilateral facial weakness of lower motor neurone type. He could not sit up or turn over in bed. He had retention of urine and needed catheterization.

During the second, third, and fourth weeks (December 9–30) his condition deteriorated still further and at times appeared hopeless. Respiration became embarrassed. Swallowing was difficult and nasal regurgitation of fluids occurred. He developed a right basal pneumonia and a urinary infection. Adequate relief of pain was difficult and required frequent injections of pethidine hydrochloride (50–100 mg.).

A gradual improvement was noticeable during the fifth week. By the sixth week he had regained a considerable amount of muscular power but still suffered a good deal of pain in the legs. Catheterization was no longer needed.

He was able to start walking with assistance in the seventh and eighth weeks (January 14–27, 1955) and was discharged home, to continue physiotherapy as an out-patient, on February 2, nine weeks after admission.

He had made an almost complete recovery six months later. He was able to walk easily and had returned to work. The knee- and ankle-jerks were absent.

Treatment was largely symptomatic and involved extremely careful nursing and physiotherapy. His diabetes was easily controlled with a small amount of insulin (I.Z.S. 20 units) and a diet containing about 120 g. of carbohydrate daily. Frequent courses of antibiotics were needed to control intercurrent infection. He was also given vitamin B₁₂ in large doses (500 µg. daily), parenteral aneurine hydrochloride (100 mg. daily), and nicotinamide (10 mg. daily) during most of the first six weeks of his illness.

COMMENT

Muscular paralysis is relatively rare in diabetes (Martin, 1953). It is usually asymmetrical and often involves one limb or muscle group only. A bilaterally symmetrical polyneuritis involving motor and sensory fibres, similar to that occurring in diphtheria, alcoholism, or infective polyneuritis, is extremely rare (Kraus, 1920), but occasional cases have been reported. Buzzard (1890) described a case of this type with a prolonged course. More severe cases with a relatively acute onset were described by Pitres and Marchand (1917) and Root (1922).

Particularly unusual features in the present case were the rapidity of onset and severity of the paralysis, the involvement of muscles of respiration and swallowing, the involvement of the bladder, and the bilateral facial weakness.

The polyneuritis in this case was not necessarily diabetic in origin, although no other cause was apparent. The clinical features were similar to those of infective polyneuritis in practically all respects except the absence of fever. This disease, or some unknown factor, may have been the cause of the symptoms.

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