

SARCOMA OF JEJUNUM

wise all normal structures have been replaced. The serosa is thickened by tumor, which is limited externally by fibrous tissue, mesothelium, and a few strands of fibrin. An adjacent portion of jejunum shows chronic inflammation of fourteen lymph nodes; one shows complete and another almost complete replacement by tumor like that in the jejunum, with the tumor breaking through the capsule. The other nodes show slight follicular hyperplasia, fibrosis, and chronic inflammatory infiltrate.

Reticulin preparations of the tumor show networks of fine fibrils between and apparently within the cytoplasm of tumor cells in some areas.

Pathologic diagnosis:

- (1) Reticulum cell sarcoma of jejunum with metastasis to mesenteric lymph nodes
- (2) Chronic jejunitis
- (3) Chronic hyperplastic lymphadenitis (mesenteric)

The postoperative course was uneventful, and the patient was discharged about two weeks after operation February 24, 1944. After discharge a series of x-ray treatments was applied to patient's abdomen and lumbar and sacral regions.

SUMMARY

A case of reticulum cell sarcoma (lymphosarcoma) of the jejunum has been reported, characterized by an insidious onset, and followed by symptoms of obstruction. A palpable mass was later found to involve the entire wall of the midportion of the jejunum and to cause marked stenosis.

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NARCOLEPSY AND CATAPLEXY

Report of Two Cases

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Narcolepsy is a term suggested by Gélinau¹ in 1880 to designate an uncontrollable desire for sleep resulting in a trancelike state of varying duration indistinguishable from normal sleep. This occurs under condi-

tions not ordinarily conducive to normal sleep. Cataplexy is a term suggested by Henneberg² to describe loss of muscle tone without loss of consciousness occurring in response to an emotional stimulus. This state is characterized by complete muscular atony, by absence of deep reflexes, and occasionally by a positive Babinski sign. The term cataplexy must be distinguished from the more common term catalepsy, which designates the state of muscular rigidity in the catatonic form of schizophrenia and in hysteria. The narcoleptic and the cataplectic syndromes may coexist in one person. Brock and Wiesel³ suggest that they are abnormalities of the two component parts of normal sleep: lack of mental awareness and sympathetic nervous relaxation.

Clinically, narcolepsy usually precedes cataplexy. A period of marked muscular weakness, such that the patient is unable to carry out voluntary motion, may occur either before or after the narcoleptic seizure. Both normal and narcoleptic sleeps in these patients may be accompanied by nightmares, sleepwalking, and sleeptalking.

The narcoleptic-cataplectic state appears in many respects to be merely an exaggeration of the normal reaction. Almost everyone has experienced sleepiness when listening to a dull, uninteresting lecture, and a sense of muscular weakness is exceedingly common under intense emotional strain.

CASE REPORTS

Case 1. A man, aged 27, was first seen at the Clinic on August 14, 1943. He complained of compulsive sleepy spells occurring daily during the previous year. These developed suddenly without obvious cause. A period of semiconsciousness was followed by sound sleep lasting from ten minutes to two hours. He was told that during semiconsciousness his pupils contracted. Upon waking he felt well but sometimes had forgotten what he was doing when the attack began. The attacks were never accompanied by convulsive movements.

He had insomnia frequently and sat up smoking a large part of the night. When asleep he was often wakened by a sensation of pulling or jerking in the abdomen. Persistent nocturnal erection occurred. The patient complained of severe recurrent headaches during the preceding year. These were referred to the occipital and suboccipital regions and were associated with a tight, full feeling in the head. He noted extreme muscular weakness under emotional stress.

For the preceding three years the patient had worked under great tension attempting to build up his own business. There was no history of head injury or brain inflammation. Except for measles, mumps, and chicken pox in childhood, he had been in good health.

General physical examination revealed a well developed, well nourished adult. Temperature was 98.8 F., pulse 84, blood pressure 130 systolic, 85 diastolic. The pupils were small and equal, and reacted to light and in accommodation. Optic disks were clearly outlined, and retinæ appeared normal. There was no thyroid enlargement. Chest appeared normal, heart was of moderate size, and heart sounds were regular and

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rhythmical. There was no enlargement of liver, kidneys, or spleen, and there were no abdominal masses. The rectal sphincter was spastic. Prostate was of normal size and firmness. Upon neurologic examination cranial nerves appeared intact, tendon reflexes normal, and there was no evidence of sensory or motor change. Examination of the nose and throat revealed some deviation of the nasal septum and some evidence of catarrhal rhinitis. Tonsils had been removed, tongue was normal, and teeth showed good dental repair.

Results of routine laboratory tests, including urinalysis, blood count, blood sugar, and blood Wassermann, were all within the normal range. Upon x-ray examination the skull was entirely normal. The pineal gland was densely calcified, but the sella turcica and the petrous pyramids were normal.

A diagnosis of narcolepsy and cataplexy was made. The patient was directed to take 10 mg. of benzedrine sulfate twice daily, after breakfast and after lunch. Since little improvement was noted after ten days, the dosage was increased to 30 mg. daily, to be taken before 2:00 p.m. Considerable improvement resulted from this medication, but the patient was also given $1\frac{1}{2}$ grains of dilantin twice daily because of undue stimulation from the benzedrine. He was advised to obtain more rest and to establish regular living habits. Medication combined with a more rational regimen produced definite improvement.

Case 2. A woman, aged 22, came to the Clinic on April 5, 1944 complaining of spells of sleepiness. These began eight years previously when she was in high school and continued during her college years when she frequently fell asleep in class. Almost from the onset she noted a lack of muscular tone when laughing or when under emotional stress. She believed that a high carbohydrate diet increased this weakness. Although she believed that the symptoms were subsiding, they interfered with her work as a dental assistant.

Apart from measles and whooping cough in childhood, her general health history was very good.

General physical examination revealed a well developed, well nourished adult. Temperature was 98.4 F., pulse 80, blood pressure 106 systolic, 76 diastolic. Pupillary reactions were normal. Optic disks were clearly outlined, and retinae were normal. Maxillary sinuses were transilluminated clearly, ear drums normal, and tongue normal. There were no dead teeth and only a small amount of dental repair. Tonsils were of moderate size. There was no thyroid enlargement. Breasts were normal. Chest was clear, the heart not enlarged, heart sounds regular and rhythmical. No enlargement of liver, kidneys, or spleen was noted, and there were no abdominal masses. Pelvic viscera appeared normal upon examination per rectum. Back was flexible and not tender. Neurologic examination showed the cranial nerves normal, the tendon reflexes normal, and no evidence of sensory or motor change. Results of routine laboratory tests, including urinalysis, blood count, blood sugar, and blood Wassermann and Kahn, were within the normal range.

A diagnosis of narcolepsy and cataplexy was made. The patient was directed to take 10 mg. of benzedrine sulphate and $\frac{3}{8}$ grain of ephedrine sulphate twice daily, after breakfast and after lunch. Considerable improvement resulted from this medication.

COMMENT

The pathologic basis of the narcoleptic-cataplectic syndrome is not established because postmortem studies are lacking. According to

the consensus, the syndrome is the result of an organic defect, possibly related to the sequelae of encephalitis or meningitis, or associated with neoplasm. The sleep-regulatory mechanism is believed to be in the posterior hypothalamic region, but some symptoms suggest involvement of the upper midbrain. Lack of consciousness in a narcoleptic attack indicates possible involvement of the cerebral cortex as well.

The term narcolepsy suggests a relation with epilepsy, but there is no evidence of common etiology although the attacks in the two conditions are somewhat similar. There is no record of a patient with narcolepsy developing epilepsy. According to Dynes,⁴ electroencephalographic studies reveal little difference between persons in normal sleep and those in a narcoleptic attack, in contradistinction to the abnormal electrical discharges from the brain of an epileptic patient. Furthermore, narcolepsy is best controlled by stimulating drugs such as ephedrine and benzedrine, whereas epilepsy is best controlled by sedatives.

The narcoleptic-cataplectic syndrome may persist for many years with periods of remission. Ephedrine and benzedrine may modify its course, but medication must be continued over long periods of time. Attention to the general health is of considerable importance. Apparently the condition does not shorten life.

SUMMARY

Two cases of the narcoleptic-cataplectic syndrome are reported. It is characteristic that these patients present no definite neurologic signs, although they present symptoms which are sometimes observed in patients with organic brain lesion. Treatment with stimulating drugs, chiefly benzedrine and ephedrine, does much to alleviate symptoms. The condition has no apparent relation to epilepsy, and the prognosis is generally good although the symptoms may persist through life.

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