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BROWN-SÉQUARD SYNDROME

A Case of Unusual *Ætiology*

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THAT hypernephromata frequently give rise to metastases in the central nervous system and its coverings is well known. The location of the secondary growths is most frequent in the bones of the skull and vertebral column and in the substance of the brain. Extension from the vertebral column or from the spinal meninges may ultimately give rise to symptoms of pressure on the spinal cord, but a case where the first and last symptoms of the tumour were due to invasion of the medulla spinalis and where at autopsy this was found to be the only lesion outside the primary tumour is sufficiently rare to warrant its being put on record.

Since Augstein (1921) has reviewed the subject there is little point in discussing the general question of the site of hypernephromatous secondaries. The literature abounds in accounts of metastases in bone, lung, liver, adrenal, and other tissues, but there are, so far as can be determined from a search of the literature, only two cases of spinal intramedullary metastasis. A case reported by Belz (1912) showed a mass in the lumbar cord extending into the upper sacral region. The author shows without doubt the intramedullary nature of the secondary, there being no involvement of the spinal coverings. Other features of the case were secondaries in the liver and a recurrent growth with involvement of the associated lymph nodes at the site of the original tumour six months after the removal of the primary mass. The appearance of the metastasis in the spinal cord is very similar to that in the present case. V. Pfunzen (1906) reported a case of metastatic growth from a hypernephroma where the secondary was completely intramedullary and was situated at the level of the second cervical segment; hæmorrhage had occurred into the

secondary and had given rise to a hæmatomyelia. No histological picture of the secondary is given. The primary growth had been removed one year before the onset of neurological symptoms.

Personal Case

W. S., a male 62 years of age, was admitted to the Western Infirmary of Glasgow on 29th October, 1936. His complaint was loss of power in the right leg of seven weeks' duration. He gave the following history. Seven weeks before admission he felt tingling and numbness in the toes of the right foot. Gradually these symptoms spread to involve the whole extremity. This extension was accompanied by a progressive loss of power in the affected leg. He felt that the leg had become useless and he volunteered the information that he had no knowledge of the posture of the limb. At the same time he became aware that stimuli which normally caused pain no longer did so when applied to the left leg. For some weeks before admission he had precipitancy of micturition, which, within the past week, had given place to incontinence of urine. On admission there was some pain in the region of the right scapular angle, which pain occasionally radiated to the front of the body.

Until seven weeks before admission to hospital his health was excellent and apart from the diseases of childhood he had never been ill. No facts of any present significance were recorded in the family history.

Clinical Examination.—October 30, 1936. The patient is very obese. He is uncomfortable as he lies in bed. The face is pale and slightly cyanotic. There is no complaint of pain, but some sense of discomfort is experienced in the right leg. The right leg lies immobile save for some involuntary twitching. Wasting is not in evidence. The nutrition of the skin over the sacrum is seriously impaired, but the surface is not actually broken.

Circulatory System.—The pulse rate was 94 per minute. The beats were regular in volume and in rhythm. The apex beat was impalpable. The transverse dimension of the area of cardiac dullness measured 4 in. to the left of the right border, which was situated $\frac{1}{4}$ in. to the right of the middle line. The systolic blood pressure was 160 mm. Hg., while the diastolic was 90.

Respiratory System.—No abnormality was detected.

Alimentary System.—The tongue was slightly furred. The abdominal wall was very obese; palpation revealed no local or general tenderness nor was any mass felt in the abdomen.

Urinary System.—The urine contained no pathological constituents.

Nervous System (see Diagram 1).—Examination revealed no abnormality in function of any of the cranial nerves. Sensory and motor functions and the deep reflexes in the upper extremities and shoulder girdle were normal. The abdominal and cremasteric reflexes were absent on both sides. The right patellar and Achilles' tendon reflexes were much more hyperactive than the brisk reflexes of the opposite limb. The plantar response was extensor in type on both sides. The right leg could not be moved voluntarily. Increased resistance to passive movement was not a marked feature, but clonus was elicited at the right knee and ankle. Sensory tests revealed a complete anæsthesia to pin-prick in the skin of the left inferior extremity. This area of anæsthesia was continuous, with a similar defect extending up to and involving the eighth dorsal segment. There was impairment without absolute loss of pain sensibility in the skin of the sixth and seventh dorsal segments of the left side. The area over which impairment of temperature sensibility was observed was practically co-extensive with that just described. Touch was appreciated well on both sides. Postural sensibility was intact in the left lower limb, but was entirely absent in the right. Vibration sensibility as tested by tuning fork was absent over the bony prominences of both inferior extremities except over the left internal malleolus, where vibration was appreciated only to a very slight extent.

Lumbar Puncture.—This procedure showed a cerebrospinal fluid pressure of 150 mm. C.S.F. Compression of the jugular veins was attended by a rapid rise in pressure which fell quickly on release. Cells in the cerebrospinal fluid numbered 2 per c.mm. There was no increase in the protein content and the Wassermann reaction was negative. Shortly after the performance of the lumbar puncture the

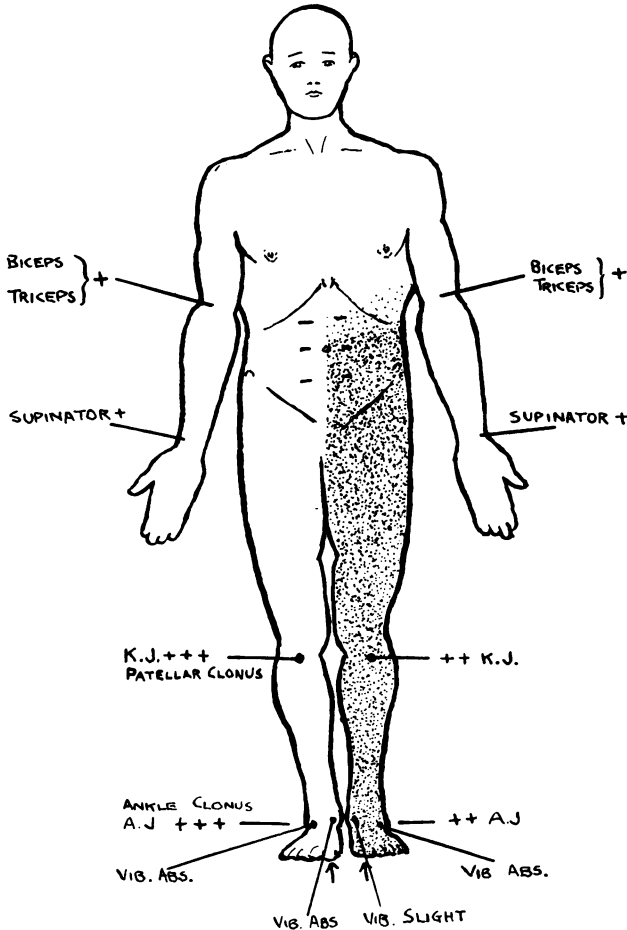


DIAGRAM 1.

SUMMARY OF THE PRINCIPAL FINDINGS. THE STIPPLED AREA REPRESENTS THE REGION OVER WHICH PAIN AND TEMPERATURE SENSIBILITY WERE LOST. PROPRIOCEPTIVE SENSIBILITY WAS LOST IN THE RIGHT LOWER LIMB BUT IS NOT INDICATED IN THE DIAGRAM

patient died and went to autopsy with the clinical diagnosis of a right-sided lesion in the mid-dorsal region of the spinal cord ; the nature of the lesion was not suggested.

The following is a record of the more important findings of an autopsy carried out on 2nd November, 1936, 48 hours after death.

The body is that of a male subject with considerable excess of fat both in the subcutaneous tissue and other fat depots. The *peritoneal cavity* contains 60 c.c. of blood-stained fluid.

Both *kidneys* are enlarged (right 800 gm., left 280 gm.). The entire upper pole of the right kidney is occupied by a hypernephroma, 9 cm. by 8 cm. in its maximum longitudinal and transverse measurements. Hæmorrhage, necrosis, and autolysis are marked features of the growth. It has no capsule and has replaced the kidney substance of the upper pole, without, however, invading the renal vessels or pelvis. The surviving tissue and the whole of the left kidney show no important change. The lower urinary tract and both suprarenals are normal. The *spleen* is enlarged (300 gm.) and soft; the *heart* is enlarged (480 gm.) and all its chambers are dilated. No secondary growths or other significant lesions are found in any of the thoracic or abdominal viscera.

Examination of the *central nervous system* shows only one lesion—a greyish-white, firm swelling in the right half of the spinal cord involving the eighth and ninth dorsal segments. This swelling produces only slight distortion of the normal shape of the spinal cord, since it is almost entirely intramedullary. The *dura mater* is not involved. The brain shows no flattening of the convolutions, but there is evidence of some degree of compression of the medulla, although without formation of an actual “pressure cone.” The vertebral column, skull, and meninges show no abnormality.

The *spinal cord*, after fixation in formalin and examination, segment by segment, shows only the lesion in the eighth and ninth dorsal segments as described above. Sections prepared for microscopic examination after paraffin embedding of the tissue demonstrate that the *renal tumour* (Figs. 1 and 2) is a typical hypernephroma. In the parts least affected by degeneration and autolysis the delicate vascular stroma supporting numerous large vacuolated epithelial cells is readily made out. In certain parts of the growth the epithelial cells are arranged in acini, while in others they are grouped in more solid irregular masses with many aberrant types of cell. Figs. 3 and 4 clearly indicate that the tumour in the spinal cord is of identical structure.

The maximum involvement of the cord is at the ninth dorsal segment where the entire right half of the spinal medulla is replaced by tumour tissue. The tenth dorsal segment is not involved and the upper limit of the growth is found in the eighth dorsal segment. Tumour is present in the sheath and substance of the posterior nerve root entering the eighth dorsal segment.

The clinical interest in this case lies in the fact that a syndrome closely resembling that described by Brown-Séquard has arisen from an intraspinal metastasis. It is generally agreed that the pure syndrome is but rarely seen and that its usual ætiology is traumatic. The picture here is not quite classical, since there is an extensor plantar response on the left side and sensibility to vibratory stimuli was absent or much diminished over the bony points of the left inferior extremity. Two clinical anomalies are worthy of note. Sensibility to vibration was lost in the limb in which postural sense was well maintained. Thus apparently there is a contradiction of the classical view that the same pathway in the cord subserves conduction of the two forms of stimuli; such a conclusion, however, implies equal vulnerability of the two systems. The other finding is that, in this case of cord neoplasm, the cerebrospinal fluid contained no excess of protein and cells.

We wish to express our thanks to Dr. John Gracie, in whose wards the patient was investigated, and to Mr. John Kirkpatrick of the Pathology Department for the photomicrographs.

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Fig. 1.—Tumour of kidney showing typical structure of hypernephroma. Mallory's connective tissue stain ; $\times 100$.

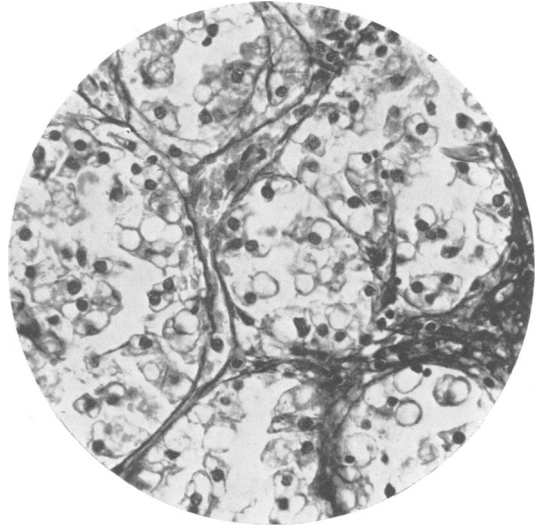


Fig. 2.—Higher magnification of Fig. 1 ; $\times 300$.

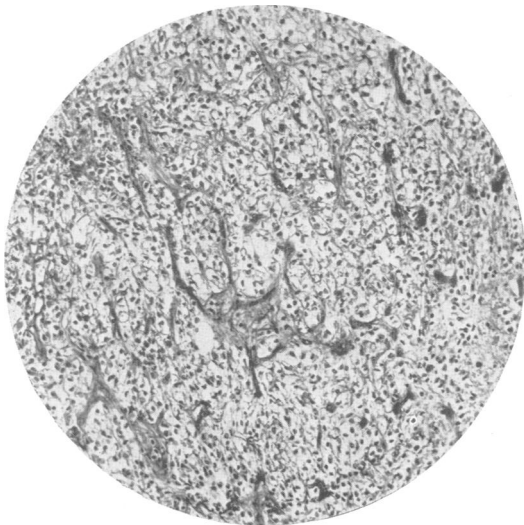


Fig. 3.—Tumour of spinal cord showing same general features as Fig. 1. Mallory's connective tissue stain ; $\times 100$.

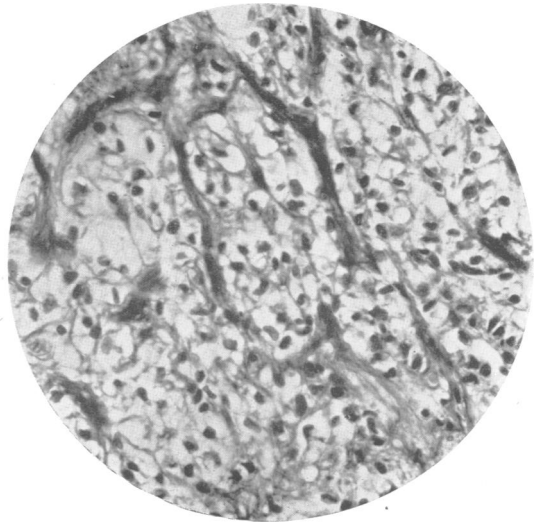


Fig. 4.—Higher magnification of Fig. 3 ; $\times 300$.