Remarks

ON

A CASE OF INTRACRANIAL TUMOUR

BY

A. F. TREDGOLD, M.D., F.R.C.P., F.R.S.Ed.

ASSOCIATE PHYSICIAN IN PSYCHOLOGICAL MEDICINE, UNIVERSITY COLLEGE
HOSPITAL, LONDON; PHYSICIAN IN NEUROLOGY, ROYAL
SURREY COUNTY HOSPITAL

WITH A PATHOLOGICAL ACCOUNT

BY

E. T. RUSTON, M.B., Ch.B.

ASSISTANT PATHOLOGIST, ROYAL SURREY COUNTY HOSPITAL

(With Special Plate)

It is thought that the following somewhat unusual case is of sufficient clinical and pathological interest to justify its publication. When the patient first came under observation the clinical signs and symptoms had been present for two years, and were chiefly referable to the nasopharynx, in which situation a hard swelling was present. There were, however, at this time some indications that the cranial cavity was also involved. When the patient was seen again three years later it was clear that the growth was causing marked pressure upon all the cranial nerves of one side, below and including the eighth. It is interesting to note that, although there was no actual involvement of the brain by the tumour, the man nevertheless presented marked and progressive dulling of perception, loss of memory, and general intellectual deterioration.

From the pathological point of view the chief interest lies in the nature of the growth, as to whether its origin was or was not in a "rest" of the notochord. It may be recalled that the chorda dorsalis is a specific embryonal tissue about which the spinal column develops. Portions of it commonly persist in the coccyx and in the junction of the sphenoid and occipital bones at the base of the skull. These portions occasionally give rise to small tumours, which in some instances increase considerably in size and assume malignant characteristics. The situation and naked-eye appearance of this particular growth, causing as it did marked absorption of the basi-sphenoid with extensions into the nasopharynx and posterior fossa of the skull, are highly suggestive of a chordoma. On the other hand, the histological appearance of stained sections is, on the whole, more closely akin to that of an endothelioma. While there is, perhaps, a certain amount of doubt as to the actual pathology, the probability is that it was a primary malignant growth of the nasopharynx which, after involving the basi-sphenoid, extended into the posterior cranial fossa.

CLINICAL ACCOUNT

A cowman, aged 59 years, was sent to the out-patient department of the Royal Surrey County Hospital on June 2nd, 1927. He had complained of pain in the left temporal region and left side of the throat, deafness in the left ear, epistaxis from the left nostril, occasional vomiting, and loss of weight for the previous two years, and he was gradually getting worse. On examination he was generally wasted, sallow, and looked ill. There was ptosis of the left eye, and a hard swelling on the left side of the posterior wall of the pharynx, but no other abnormality. Wassermann reaction of the blood was negative. He was seen by Dr. T. B. Jobson, surgeon in charge of the ear, nose, and throat department, and by myself, and we were of the opinion that the condition was one of malignant and inoperable growth of the nasopharynx. He was put upon local x-ray treatment, but after a short time discontinued attendance, and was not seen again for over three years.

On October 29th, 1930, I was asked by his medical attendant, Dr. Simpson of Farnham, to whom I am indebted for

further notes, to take him into hospital, as he had become much worse, and marked cerebral symptoms had now appeared. It was stated that, in addition to his former complaints, for the past three months or so his voice had been husky, there had been difficulty in swallowing, and he had become dull and depressed mentally. On one or more occasions he had threatened suicide. On admission into hospital his condition was as follows.

Mental Condition.—He was correctly orientated, but there was marked intellectual deterioration with slow cerebration, dull perception, and loss of memory. It was impossible to elicit from him any accurate account of his illness, or to get him to co-operate satisfactorily in several tests.

Cranial Nerves.—I: So far as could be ascertained, olfactory sensation was not abnormal. II: There appeared to be a general impairment of vision, but the exact degree of this could not be determined. The edges of the disks were ill defined, and both disks were pale on the nasal sides, but there was no definite papilloedema. III, IV, and VI: The left pupil was smaller than the right; both reacted to light. There was marked ptosis of the left eyelid. It is considered that these signs were due to pressure on the ascending branch of the cervical sympathetic. The eye movements were normal, and there was no strabismus or nystagmus. V and VII: The muscles of the face and jaws appeared to be feeble, but there was no definite paralysis. VIII: Hearing on the right side was normal; a considerable degree of deafness was present on the left side, and the left drum was thickened and perforated, with projecting granulations. IX and X: There was paralysis of the left half of the palate and of the left side of the larynx, with extremely husky voice and difficulty in swallowing. Involvement of the vagus was further indicated by a rather rapid and irregular pulse, varying between 80 and 120, without any rise of temperature. XI. There was paralysis and marked atrophy of the left sterno-mastoid and upper part of the left trapezius muscles. XII: The left half of the tongue was paralysed, atrophied, and marked by deep furrows; the tongue deviated to the left on protrusion.

Spinal Nerves.—There was general wasting of all the muscles of limbs and trunk, but no localized paralysis. Sensation appeared to be normal all over the body. Knee, ankle, plantar, and abdominal reflexes were normal. There was no Rombergism, but the gait was ataxic and reeling, with a definite tendency to fall backwards and to the left.

General Condition.—The cerebro-spinal fluid, removed by lumbar puncture, was under slight pressure, but showed no pathological change on examination. Both the fluid and the blood gave negative Wassermann reactions. The systolic blood pressure was 190, but otherwise the circulatory, respiratory, alimentary, and excretory systems were normal. X-ray examination of the skull showed destruction of the body of the sphenoid bone from the posterior clinoid process to the level of the temporal bone (see Plate, Figs. 1 and 2).

Course of Illness

From the time of his admission the patient steadily deteriorated. The mental dullness became more marked; he suffered from frequent and severe headache in the left temporal and parietal regions; he had several attacks of profuse epistaxis from the left nostril; he vomited frequently; his voice became much more husky; and swallowing became increasingly difficult. Seven weeks after admission he developed pneumonia, from which he died five days later. Until its onset his temperature and respiration had remained practically normal during the time he was in hospital.

Diagnosis

It was considered that the patient had a tumour situated in the posterior fossa, chiefly on the left side, but the nature of this was uncertain.

Post-mortem Examination

A post-mortem examination made by Dr. J. Mark, my house-physician, and by Dr. Ruston, assistant pathologist to the hospital, revealed a growth about the size of a large but flattened egg between the dura and the bone in the posterior fossa of the skull, in front and mostly to the left of the foramen magnum. The position of the growth was such as to cause pressure upon the 8th, 9th, 10th, 11th, and 12th nerves of the left side, but the encephalon itself was not involved.

Dr. Mark and Dr. Ruston were at great pains to remove the whole of the bone to which the growth was attached, and Dr. Ruston has been good enough to write the following account of it.

PATHOLOGICAL ACCOUNT

It was only possible to make a limited examination of the skull. Upon removal of the brain in the usual way, there was exposed a soft, white, extradural tumour, compressing considerably the left half of the cerebellum and the medulla. This tumour appeared to be arising from the basi-sphenoid, spreading more to the left of the middle line than to the right, and extending down to the foramen magnum, where its lower border was turned under. Irregular in shape, it measured $1\frac{1}{2}$ inches in its greatest length and $1\frac{1}{4}$ inches in its greatest breadth (less than 1 inch from the posterior clinoid processes). Its surface was lobulated, more so on the left side, where parts of it were raised half an inch above the neighbouring bone; its edge was similarly indented. No alteration in its consistency was made out, and nowhere was there any suggestion of bony or cartilaginous tissue. The dura was continuous over its surface, and no connexion was made out with any of the cranial nerves. A large vein issued from its surface about three-quarters of an inch from the lower pole; the lumen was free from growth.

The tumour was removed intact, together with the greater part of the basi-sphenoid, the anterior three-quarters of the foramen magnum, and the body of the first cervical vertebra. The pharyngeal aspect showed a small soft area of growth, which had penetrated right through the bone, but there was no other suggestion of pharyngeal origin, and a more complete examination was impossible. The specimen was decalcified and subsequently cut in the laboratory of University College, and then showed considerable absorption of the basi-sphenoid bone.

The naked-eye appearance of the tumour and its situation suggested the possibility of its being a chordoma; but the histological features were, on the whole, more like those of an endothelioma. Under the microscope there were masses of deeply staining endothelial cells within a fibrous stroma. These cell masses showed variations in the size and staining of the nuclei, in the packing together of the cells, and in the granularity of the cytoplasm. There were numerous large and multinucleate (giant) cells (3 to 6) with a paler cytoplasm. No cell nests or "epithelial pearls" were seen, and the fibrocellular stroma showed no inflammatory cells clustered together in defensive reaction.

MacCallum (*Textbook of Pathology*) gives a short note on chordomata, and speaks of their "lobules of groups and strands of large and small cells" which are rich in glycogen. The microscopical appearances of the present case closely resembled this description, and sections stained with iodine showed up the cell masses a deep brown. Ewing (*Neoplastic Diseases*), commenting on the difficulty of a positive histological diagnosis of chordomata, describes them as consisting of large, rounded, vacuolated, and hyperchromatic cells, and mentions a tumour described by Hassner measuring 5.5 by 6.5 cm., which produced symptoms over a period of four years. The clinical evidence in the present case points to a duration of over five years.

Professor M. J. Stewart of Leeds (*Journ. Path. and Bact.*, January, 1922) describes the chordomata as possessing an alveolar structure within a fibrous stroma, and stresses the importance of demonstrating mucin production in the multinucleate and vacuolated syncytial cells; he says, "the fully developed physaliphorous cell of Virchow is one ballooned with mucin." This condition I was not able to demonstrate satisfactorily in the present case, although with polychrome methylene blue the giant cells do take on a faint pink, and with orange G the granularity is more evident in the cytoplasm, and in places appears to stream out from the cells. It may be remarked, however, that in his article Stewart mentions a case described by Eitel in which mucin was absent and the cells were rich in glycogen. While, therefore, there are many features of this case which are suggestive of its

being a notochordal tumour, the signs are not conclusive, and on the whole the evidence is in favour of its being an extending pharyngeal carcinoma. It well illustrates the difficulties of making a diagnosis between these two conditions.

I am indebted to Dr. Tredgold for the opportunity to examine this case, and at his request have written this note.

MENORRHAGIAS NOT DUE TO UTERINE DISEASE:

CAUSATION, CLINICAL FEATURES, AND TREATMENT*

BY

JAMES YOUNG, D.S.O., M.D., F.R.C.S.Ed.

GYNAECOLOGIST, ROYAL INFIRMARY; PHYSICIAN, ROYAL MATERNITY AND SIMPSON MEMORIAL HOSPITAL, EDINBURGH

(With Special Plate)

Cases of irregular and excessive uterine haemorrhage are divided into two main classes:—(1) Those in which the uterus is the seat of tumours (fibromyoma, mucous polypi and malignant disease) or retained fragments of placenta, and (2) those in which such a gross uterine pathology is absent and where instead there is nothing more than changes in the endometrium, usually of a proliferative nature, with commonly, in addition, some general and uniform overgrowth of the fibro-muscular wall of the uterus.

It is with the second of these two groups that this discussion is concerned. It embraces the majority of cases of bleeding uteri, and it includes the bulk of those cases which in the past have been described by such terms as chronic endometritis, chronic metritis, fibrosis uteri, and subinvolution.

ETIOLOGY

It is now some twenty years since Schroeder,¹ Beckwith Whitehouse,² the present writer³ and others directed attention to evidence suggesting that many such cases owe their origin to disturbed ovarian function. In a paper published in 1913 and entitled "Uterine haemorrhage of ovarian origin" I gave reasons for the view that "where there is found on examination nothing abnormal except some uterine enlargement, and where the mucosa is found to exhibit the appearances of so-called 'glandular endometritis,' an ovarian source should be suspected."

It was Schroeder who, from the chaos of clinical and pathological data, first succeeded in building up a condition with some semblance to a separate entity which he has termed "metropathia haemorrhagica." Its main symptom is *metrorrhagia or irregular continued bleeding*, and, though occurring at any age from the menarche onwards, it is found characteristically between 40 and 50. Its pathology consists of a diffuse, often polypoidal proliferation of the endometrium with a hyperplasia and cystic dilatation of the glands, marked congestion of the vessels, free extravasation into the stroma and extensive endometrial necrosis. In a considerable number the muscular wall is increased as the result of a myohyperplasia. The ovaries exhibit characteristic changes in the shape of a cystic degeneration of the follicles and a failure of formation of corpora lutea and, according to Schroeder, the uterine changes and the consequent bleeding arise from this ovarian pathology. The views of Schroeder are supported in their general lines by Wilfred Shaw,⁴ Graves,⁵ and others. Shaw, for example, in 200 consecutive hospital cases of "dysfunctional" irregular bleeding found

* Read in opening a discussion in the Section of Obstetrics and Gynaecology at the Annual Meeting of the British Medical Association, Eastbourne, 1931.

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FIG. 1.—Radiograph of skull showing at (A) extensive destruction of basi-sphenoid bone.

FIG. 2.—Radiograph of basi-sphenoid and adjacent bones. A. Basi-sphenoid showing destruction of bone by tumour. B. Semicircular canals. C. Odontoid process of axis.

FIG. 3.—Section of tumour $\times 17.5$. A. Nasopharyngeal tumour. B. Mucoperiosteum of nasopharynx. C. Basi-sphenoid bone. D. Periosteum. E. Intracranial tumour.

FIG. 4.—Section through intracranial portion of tumour.