MENINGIOMAS WITHIN THE LATERAL VENTRICLE

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Meningiomas within the lateral ventricle of the brain, although rare, are interesting from both clinical and pathological points of view. Diagnosis without special investigation is difficult, because, although most of these tumours produce focal signs, such signs are indistinguishable from those due to more common tumours in the posterior part of the cerebral hemisphere. The benign nature and intraventricular position of the tumour are usually surprise findings. Another aspect of surgical importance is that although these tumours have characteristics which render their total removal easy, operation is often followed by severe and lasting disability, caused by inevitable damage to overlying brain in securing access to the growth. Their interest to pathologists lies in the problem of their origins and the inferences some workers have drawn from a study of their histology.

This paper will describe eight such meningiomas personally observed during the last five years, and will review, especially from the clinical aspect, cases previously recorded.

A note on terminology is necessary at the outset. The term "meningioma within the lateral ventricle" is used here to designate a meningioma either lying free within the ventricle except for its attachment to the choroid plexus, or lying partly in the ventricle and partly embedded in the cerebral hemisphere. Cushing and Eisenhardt (1938) differentiate between these types, calling the former "true plexus meningiomas" and the latter "lateral tumours of the velum, or sub-cortical meningiomas". However, they agree that at operation it is often difficult to assign a tumour to one of these categories and indeed that a classification into these two types may not be fundamentally correct.

Previously Recorded Cases

About 50 histologically proven examples of meningioma within the lateral ventricle have been described, 20 by Cushing and Eisenhardt (1938), 22 by Abbott and Courville (1942), and more

recently cases have been recorded by Rasmussen (1941), Knight (1949), Huber (1950), and Chavany, Bonduelle, and Guiot (1952).

The average age of the patients was 32 years, the youngest being $3\frac{1}{2}$ years (Gardner and Turner, 1938) and the eldest 65 years (Jefferson and Jackson, 1938). Females were affected more commonly than males in the ratio of 7 to 3.

Symptoms and Signs.—As with meningiomas elsewhere the duration of symptoms, which varied from a few months to eight years, bore no relation to the probable age of the tumour. The first symptom in the majority of cases was headache and as a rule there were no characteristics to distinguish it from headache due to intracranial tumour elsewhere, although occasionally a severe paroxysmal type was recorded (Campbell and Whitfield, 1940). Papilloedema was nearly always present.

Contralateral sensory or motor impairment was recorded in about 70% of cases and usually was not severe. Epileptic fits were rarely described and rarely had localizing value, although one of the patients described by Busch (1939) had had epileptic fits with a visual aura in the blind homonymous field for four years before headache began. Uncinate attacks were described by Love (1935). Homonymous hemianopia, involving mainly lower quadrants and sparing the macula, was described in 60% of cases, whereas visual hallucinations in the blind fields were rare, e.g., Fincher (1934). Dysphasia or dyslexia was only occasionally evident before operation.

Paraesthesiae, paroxysmal or continuous, in the trigeminal area on the same side as the tumour were occasionally noted, but objective evidence of sensory impairment was very rare; a case with depressed corneal sensation on the same side as the tumour was described by Woolsey and Klemme (1941).

Mental changes were often recorded, one of the

most striking examples being that of Gross (1939).

Signs suggestive of a cerebellar lesion, e.g., ataxia and nystagmus, were noted in about one quarter of the cases, the ataxia being always on the side opposite to the tumour.

Diagnosis.—By clinical examination alone the furthest step ever made was to locate a space-occupying lesion in the appropriate part of one cerebral hemisphere. Its position within the ventricle was never suggested at this stage.

Plain radiographs of the skull sometimes showed calcification within the tumour, e.g., Cohen (1941), but usually ventriculography followed by craniotomy was the method of diagnosis.

On the few occasions when carotid arteriography was used, it merely indicated a mass causing displacement of middle and anterior cerebral vessels. Ameli (1952) has described a case in which the tumour was shown by vertebral arteriography.

Position, Size, and Shape of Tumour.—In 64% of cases the tumours were on the left side, this being especially common in females. The tumour was usually confined to one lateral ventricle, expanding the trigone, having prolongations of variable size forwards into the body and inferior horn and frequently extending into parietal white matter to within 2 cm. of the cortex. The first case described by Busch (1939), which was approached through the frontal horn, showed a small extension of the tumour through the foramen of Monro into the third ventricle, and Busch later found the same feature in two other tumours approached through the parietal lobe.

The weights of the tumours varied from about 20 g. to 340 g. (Hoen quoted by Cushing).

Histology.—Abbott and Courville (1942) observed that all except one of the tumours recorded before 1942 had been called fibroblastic and this description applies to most of the tumours recorded since then. Cushing and Eisenhardt (1938), in differentiating between true plexus meningiomas and lateral tumours of the velum, describe the former as being more highly psammomatous than the latter.

Methods of Removal

In nearly all cases the tumour was approached through the parieto-occipital cortex and white matter, which were either incised or removed. Occasionally, e.g., Busch Case I (1939), the tumour was lying sufficiently far forwards in the ventricle for it to be approached through the frontal lobe.

Results of Operation.—Considered in terms of operative mortality and completeness of removal of the tumours, the results were excellent, but, as most of the tumours were in the left hemisphere, permanent disability of speech was often severe.

Immediate post-operative increase of speech disturbance and hemiparesis always occurred but the latter usually disappeared in the ensuing weeks. Epileptic fits were often recorded. A permanent homonymous field defect was noted when the approach was through the parietal lobe.

The Present Series

The series reported here consists of eight cases.

Case 1.—E. M., a man aged 33, a labourer, was admitted to the National Hospital (No. 10074) in May, 1948, under Dr. F. M. R. Walshe and transferred to Mr. Julian Taylor.

His main complaints were visual deterioration and headache for about two and a half years. The visual changes consisted of blurred central vision and the development of a right-sided field defect. Headache, which was mild and bifrontal, had occurred almost every morning for two years, remaining each time for about two hours and then gradually disappearing. He had had no facial paraesthesiae, no disturbance of speech, and no weakness of the limbs.

On examination the abnormal signs were bilateral anosmia (probably due to chronic catarrh), corrected visual acuity of 6/24 right and 6/36 left and an incongruous right homonymous hemianopia, complete in the lower and partial in the upper quadrants with macular sparing. The nasal margins of both optic discs were blurred but assessment was difficult because of myopia. There was no trigeminal sensory loss, no facial weakness and no motor, reflex, or sensory abnormalities in the limbs or trunk.

Straight radiographs of the skull showed erosion of the dorsum sellae. An E.E.G. recorded low-voltage waves of about 2 per second from the left mid-temporal region.

At ventriculography through biparietal burr-holes, only the right ventricle was entered. On the left side at a depth of 3 cm. firm tumour was encountered, which biopsy showed to be a meningioma. The films, which were of little value because no air passed into the left ventricle, showed merely a considerable shift of the right and third ventricles to the right. The protein content of the right ventricular fluid was normal. Left carotid arteriography showed displacement of the anterior cerebral artery to the right, and, in the lateral view, elevation of the middle cerebral vessels. No vessels within the tumour were seen but the anterior choroidal artery was abnormally large and ended posteriorly in a small tangle of vessels.

Craniotomy was then performed under local anaesthesia and the left parieto-occipital area exposed.

Tumour was seen at a depth of 1 cm. after making a vertical cortical incision about 3 cm. long in the posterior parietal region. Attempts were made to remove its centre with the diathermy loop, but this proved difficult and it was eventually delivered intact.

The day after operation there was severe speech disturbance but no gross weakness of the face or limbs. One month later there was still considerable dysphasia and dyslexia and also slight weakness of the right foot and hand, but no postural sense loss or astereognosis. The right homonymous hemianopia was now complete in the upper and lower quadrants, the maculae being spared. Visual acuity had improved to 6/18 right and left.

Two years after operation, although he denied speech difficulty, there was obvious nominal dysphasia, and comprehension of written words was poor. The hemianopia was unchanged. He was considered fit to resume his previous work as a labourer although he had not then done so.

The main mass of tumour measured 7 by 5.5 by 5 cm. and weighed 134.5 g. Choroid plexus was adherent to its surface for a distance of 4 cm. Histologically it was a fibrous type of meningioma, composed mostly of bundles of spindle-shaped cells, which in some parts formed whorls. Areas of meningotheial cells were scanty and no psammoma bodies were seen (Fig. 1).

FIG. 1.—Case 1: Section of tumour. $\times 80$.

This case was unusual in that the only focal sign before operation was the homonymous field defect.

Case 2.—R. T., a man aged 41, a wood machinist, was admitted to the National Hospital (No. 20617) in December, 1949, under Dr. S. P. Meadows and transferred to Mr. Harvey Jackson.

His main complaints were headache and visual disturbance.

The headache began six months previously, was at first frontal, later occipito-frontal, occurred about twice a week and although usually only a dull ache, on about four occasions had been very severe. For the two or three weeks before admission he had been free of headache.

The visual disturbance was the complaint for which he sought advice because it interfered with his work. He described it as a "kink" or "zigzag" at the point of fixation, especially troublesome when looking at a line of print or a vertical line. There was no suggestion of hemianopia. In a similar way to the headache this symptom had also become less obvious for the two or three weeks before admission.

In addition to these main symptoms he complained also that during the attacks of vomiting accompanying severe headache there was a sensation that his surroundings were rotating, in no particular direction, and lasting for a few seconds only.

There was no history of mental change, of impaired visual acuity, or of sensory or motor symptoms in trunk or limbs. He was right handed.

On examination the abnormal signs were bilateral papilloedema greater on the left, irregular nystagmus on conjugate lateral gaze especially to the left, slight weakness of the right leg, slight ataxia in the finger-nose test on both sides, more on the left, slight bilateral ataxia in the heel-knee test, bilateral extensor plantar responses, slight unsteadiness of gait especially when turning rapidly, and postural sense impairment in the right hallux. There was no field defect, visual acuity was 6/12 on both sides, and there was no evidence of mental deterioration, dysphasia, or dyslexia.

Radiographs of the skull were normal.

An E.E.G. showed waves of 4 to 6 per second which, although bilateral, were more prominent on the left side, and also 1 to 2 per second waves which were almost entirely left sided, with a maximal focus in the anterior temporal region.

Ventriculography through biparietal burr-holes showed a shift of the lateral and third ventricles to the right with dilatation of the left occipital and temporal horns. The tumour was demarcated by crescentic shadows in the posterior part of the body of the left ventricle and anterior part of the occipital horn. Cerebrospinal fluid from the right ventricle contained 15 mg. of protein per 100 ml.; that from the left, 100 mg.

A burr-hole was then made above the upper temporal crest and about 3 cm. behind the coronal suture. Needling directly inwards encountered very firm resistance beyond which a cavity was entered and 10 ml. of cloudy-white fluid aspirated. Needling in other directions established that the mass was not very large. No biopsy was taken. (In retrospect it seems likely that the needle had penetrated through the tumour, although it was never decided whence the opalescent fluid had come.)

A large left fronto-temporal flap was later elevated and an attempt made to reach the tumour through a

vertical cortical incision in the frontal lobe, but without success.

Following this operation the patient had a right hemiplegia, aphasia, and a right homonymous hemianopia. Three weeks later the hemiplegia and aphasia had considerably improved and he was able to converse hesitantly. The field defect remained unchanged.

After a second ventriculography, a further needle biopsy was attempted. Although none of the tumour could be withdrawn, the impression was gained that it was movable.

A second craniotomy was then undertaken allowing access to the parieto-occipital region. A vertical cortical incision on the convexity was made and deepened until the ventricle was entered and the firm, nobbly tumour exposed, lying mainly in the expanded trigone with a short extension forwards into the body of the ventricle. Traction sutures were inserted and, as the mass was delivered, its main blood supply, from the choroid plexus on its anterior and medial aspects, was divided.

This procedure considerably increased the dysphasia and hemiparesis. Twelve days after operation he became drowsy and vomited repeatedly. Tapping of the left ventricle allowed 35 ml. of old blood to escape under pressure, and after another tap later in the day when 12 ml. more were removed, he gradually improved.

Three weeks after operation there was no weakness of the limbs and only slight facial weakness. Deep reflexes were brisker on the right side and the right plantar response was extensor. The right half of the trunk and the right limbs showed tactile inattention without impairment of pain or touch sensation. Stereognosis could not be tested. He was dysphasic and unable to read, although he could understand simple spoken speech and was able to utter simple phrases. The field defect remained unchanged.

After five months of speech therapy he was able to read and speak simple sentences. About this time he began having fits, usually only right sided but sometimes generalized.

He was last heard of a year after operation. He was still having occasional fits, but had resumed his previous work as a joiner. Dysphasia was still obvious, the field defect unchanged, the right tendon reflexes were still the brisker and the right plantar response extensor.

The tumour, which measured 5 by 4 by 3.5 cm. and weighed 45.5 g., showed the same cellular structure as that of Case 1, except that islands of meningothelial cells were more numerous and occasional psammoma bodies were seen.

The outstanding feature of this case was the difficulty in deciding, by clinical examination alone, whether the tumour was above or below the tentorium. In fact it was the E.E.G. which gave the first real lead.

Another feature of interest was the onset of a complete homonymous hemianopia after an attempted transfrontal approach.

Case 3.—M. E., a woman aged 51, a housewife, was admitted to the National Hospital (No. 7812) in January, 1948, and again in January, 1950, under Sir Charles Symonds and transferred to Mr. Wylie McKissock.

Her main complaints were headache for one year and weakness of the right limbs for a few weeks.

The headache was always bifrontal, not more on one side than the other, present when wakening in the mornings and usually recurring in the evenings, was severe for only about 15 minutes at the beginning of each attack, and then gradually disappeared over the next few hours.

Weakness of the right hand had developed very gradually over the previous three weeks and of the right foot over a period of six weeks, the weakness slowly spreading to involve more proximal muscles. She was, however, still able to walk and to grip objects with the right hand.

There had been no visual disturbance, no fits, and no facial paraesthesiae. She was right handed.

On examination the abnormal signs were moderate nominal dysphasia, early swelling of both optic discs, visual inattention in the right half fields, slight right facial weakness, slight weakness of the right arm and leg with increased deep reflexes and impaired postural sense on this side.

There was no trigeminal sensory loss and visual acuity was 6/24 on both sides.

Lumbar cerebrospinal fluid pressure was 205 mm. and protein 55 mg. per 100 ml.

Routine radiographs of the skull showed erosion of the posterior clinoids.

An E.E.G. recorded slow waves of 1 to 4 per second from a wide area in the left parietal region.

Ventriculography through a right parietal burr-hole showed displacement of the lateral and third ventricles to the right and a filling defect in the region of the left trigone some 6.5 cm. in antero-posterior diameter. The left anterior horn was smaller than the right. The protein content of the right ventricular fluid was normal.

Biopsy showed the tumour to be a meningioma.

Craniotomy was subsequently performed and the left parietal cortex exposed. A vertical cortical incision about 3 cm. long was made in the posterior parietal region and deepened until soft vascular tumour was exposed, much of which was removed with the sucker. The wound was then closed.

This procedure produced a slight increase in the previously recorded signs. Dysphasia was a little more obvious and the right plantar response became extensor. The field defect, however, remained only relative.

Deep x-ray therapy was instituted and after this there was considerable improvement in the patient's general and mental state. Physical signs remained unchanged except that there was now a complete right homonymous hemianopia.

During the next six months her general condition continued to improve a little, but at the end of this period morning vomiting began and she was readmitted. During the first few days in hospital vomiting continued

and severe frontal headache appeared. Physical signs remained unchanged.

A second craniotomy, two years after the first, was then performed using the previous bone flap. A similar vertical cortical incision was made in the posterior parietal region and deepened until tumour was seen. After some separation, the dilated temporal horn was entered and choroid plexus identified. It was adherent to the tumour in the posterior part of the temporal horn and was divided. The posterior part of the tumour, apparently blocking completely the occipital horn, was removed, mostly by suction. There remained the main mass of tumour in the expanded trigone, with an extension forward into the posterior part of the body of the ventricle. Here again plexus was attached and was divided to enable delivery of this remaining mass.

The operation produced temporary increase of the dysphasia and limb weakness, but a month later the patient was able to converse hesitantly and there was only slight weakness of the right arm and leg. There was, however, grossly impaired postural sense in these limbs and a complete right homonymous hemianopia.

During the 18 months following the second operation there was gradual improvement, but the slight right-sided weakness, the gross cortical sensory loss, the field defect, and the now only slight disturbance of speech and reading remained.

The portion of tumour removed *en masse* at the second operation weighed 27.5 g. and measured 4.5 by 4 by 3 cm. Its histological structure was the same as the original biopsy, namely, masses of closely packed cells, most of which were rounded, but some were elongated, and with indistinct cell boundaries. In some areas streaming of the cells was apparent and elsewhere poorly formed whorls. There was a well-defined collagenous capsule but within the tumour only a little fibrous stroma and no psammoma bodies (Fig. 2).

FIG. 2.—Case 3: Section of tumour. $\times 100$.

The microscopic appearance of the tumour was the most noteworthy feature of this case. It was the only truly syncytial meningioma in the series.

Case 4.—J. M., a girl aged 15, a jewellery polisher, was admitted to the National Hospital (No. 22139) in February, 1950, under Dr. Macdonald Critchley and transferred to Mr. Harvey Jackson.

Her main complaints were headache and weakness of the left limbs. Headache began nine months previously, when, after being in the sunshine, she had a severe, thumping ache above the eyes. This disappeared after a few hours only to return the following day and nearly every subsequent day. Usually it wakened her in the mornings and lasted for about half an hour, and at the time of admission came on later in the day as well, again lasting for only a short time.

About four weeks before admission she began dropping objects from the left hand and she thought the strength of the limb as a whole was reduced. About the same time she began dragging the left foot, but this improved and when in hospital she thought the limb normal; indeed, she had cycled 60 miles only eight days before.

Also, for about four weeks, usually when washing her face, she had frequently experienced "a twitching feeling" on the right side from eye to chin, although not accompanied by actual movement. Her mother described one of these attacks as occurring at the time of a very severe headache and associated with transient severe weakness of the left hand. The patient was right handed.

On examination the abnormal signs were mild intellectual impairment, severe chronic papilloedema, complete left homonymous hemianopia with macular sparing, moderate weakness of the left arm, slight weakness of the left leg, brisker deep reflexes on the left with an equivocal left plantar response (the right being obviously flexor) gross impairment of postural sense, two-point discrimination and stereognosis in left hand and foot, with inattention on the left limbs to bilateral pain and touch stimuli.

Visual acuity was 6/6 on both sides, there was no facial weakness, and no trigeminal sensory loss.

Straight radiographs of the skull showed erosion of the posterior clinoids.

An E.E.G. recorded high-voltage waves of 1 to 3 per second arising from the right temporo-parietal region.

Ventriculography through biparietal burr-holes showed displacement of the ventricles to the left with dilatation of the right frontal and temporal horns. A mass, some 7.5 cm. in diameter, was outlined in the region of the right trigone. Cerebrospinal fluid from the left ventricle contained a normal amount of protein, that from the right about 45 mg. per 100 ml.

At operation, a curved cortical incision in the right superior temporal convolution and adjacent part of the lower parietal area was deepened until the ventricle was entered and the smooth-walled tumour exposed. After preliminary scalloping, and division of the main blood supply passing into its antero-lateral surface, the tumour was delivered intact.

Following this procedure there was no increase in the hemiparesis and three weeks later the weakness of the left arm and leg noted before operation had disappeared. There was, however, gross sensory impairment in the left limbs although less than before operation. The homonymous hemianopia was at first unchanged but subsequently improved slightly, hand movements being appreciated in the left lower quadrant.

She had her first fit five weeks after operation, and in the next two years had infrequent left-sided motor and sensory attacks, some with loss of consciousness. Nevertheless she was able to work.

The tumour weighed 160 g. and measured 8.3 by 6.5 by 5.5 cm. Histologically it was composed of bundles and whorls of spindle cells with numerous islands of more rounded cells. No psammoma bodies were seen.

This was the first case in the series in which trigeminal paraesthesiae were noted.

Case 5.—V. P., a woman aged 35, a housewife, was admitted to the Leeds General Infirmary in March, 1950, under Mr. W. R. Henderson.

Her main complaints were headache and blurring of vision for about two years. The headaches were always generalized, usually on waking, and had become more frequent and severe in the few weeks before admission.

In addition to the gradual deterioration of central vision beginning two years previously, she had had during this period attacks of diplopia each lasting for a few seconds and frequent visual blackouts, during which she was unable to see anything.

A few weeks before admission there had been an attack of unconsciousness, apparently without convulsion.

She had had no facial paraesthesiae and no speech disturbance. She had been unable to smell for about five years and had been deaf in the left ear for many years, following a childhood infection. She was right handed.

Eighteen months before, examination elsewhere had shown bilateral papilloedema, visual acuity of 6/36 in each eye, and evidence of a right lower quadrant homonymous hemianopia.

On examination at the time of admission the abnormal signs were bilateral anosmia, bilateral secondary optic atrophy more advanced in the right eye which was blind, and unsustained nystagmus on gaze to right. Vision in the left eye varied from time to time, because although she could apparently feed herself she denied better vision than perception of light. The visual field of the left eye, so far as could be tested, was full.

There was no trigeminal sensory loss, no facial weakness, no motor, reflex, or sensory changes in the limbs or trunk and no speech disturbance.

Lumbar C.S.F. protein was 43 mg. per 100 ml.

An E.E.G. recorded slow waves arising in the left temporo-occipital region.

Radiographs showed faint speckled calcification over an area some 3 cm. in diameter deep in the left mid-parietal region.

Left carotid arteriography showed stretching of the terminal middle cerebral vessels, as if from a subcortical

mass, but no vessels within the tumour. The posterior cerebral artery did not fill but it was noted that the anterior choroidal artery was larger than normal and ended in a tuft of vessels below and in front of the area of calcification.

Following this a burr-hole was made on the left side and what was apparently a dilated temporal horn tapped. During the next few days there was a remarkable and unexpected improvement of vision to J.1 left and J.18 right. Further estimation of visual fields was now possible, and no defect was found.

Ventriculography through biparietal burr-holes demonstrated a smooth filling defect about 3.5 cm. in diameter in the region of the left trigone, corresponding to the area of calcification. There was considerable shift of the lateral and third ventricles to the right with dilatation of the left temporal and posterior horns. The left anterior horn was smaller than the right. Unfortunately the ventricular fluids were not examined chemically.

Craniotomy was then undertaken allowing access to the left temporo-parietal region, and a cortical incision about 4 cm. long made in a downwards and forwards direction near the posterior end of the exposure. A vascular tumour was found and a biopsy taken, but no attempt was made to remove it at this stage.

This procedure produced an incomplete right homonymous hemianopia but, surprisingly, no motor or sensory loss in the limbs and no speech disturbance.

Vertebral arteriography later demonstrated several large posterior choroidal vessels ascending from the posterior cerebral artery and running on to the surface of the tumour, the size and shape of which was outlined both by encircling vessels and a haze of contrast medium. The normal pattern of the choroid plexus was not seen (Fig. 3).

FIG. 3.—Case 5: Vertebral arteriogram showing the outline of the tumour. Shows also the cortical incision (dotted).

At the second-stage craniotomy the original bone flap was elevated and the previous cortical incision enlarged in a downwards and backwards direction for 2 cm. from its lower extremity. The site of the incision is shown in Fig. 3. This extension necessitated division of a large

cortical vein running downwards and forwards. The tumour was considered too large to remove intact and about three-quarters of it was sucked away. As the tumour was separated from the wall of the ventricle fluid was released from the temporal horn and the choroid plexus coagulated just behind the foramen of Monro and again in the posterior part of the temporal horn. Between these points plexus was firmly adherent to tumour.

After three weeks she was walking normally and there was no weakness of the right arm or leg. There was, however, slight right facial weakness, gross postural sense impairment in the right fingers (but not in the toes), complete right homonymous hemianopia and severe dysphasia and dyslexia. Visual acuity remained improved.

She was last seen two years after operation when the signs were essentially the same as at the time of her original discharge. The homonymous field defect was complete, there was gross impairment of postural sense in the right hand, obvious dysphasia, although she was able to hold a reasonable conversation, and almost complete alexia.

The portion of tumour removed intact measured 6 by 4 by 3.5 cm. and weighed 45 g. Histological examination showed bundles and whorls of spindle-shaped cells with numerous psammoma bodies and only a few areas of meningothelial cells (Fig. 4).

FIG. 4.—Case 5: Section of tumour. $\times 80$.

Features of interest in this case were (1) the presentation as a case of visual failure due to secondary optic atrophy without focal signs, (2) the calcification seen on straight radiographs, (3) the improvement of vision after tapping the dilated temporal horn, and (4) the demonstration of the tumour by vertebral arteriography.

Case 6.—E. S., a woman aged 35, a housewife, was admitted to Leeds General Infirmary in January, 1951, under Mr. W. R. Henderson.

Her main complaints were headache and tingling on the left side of the face and in the right hand.

She had had headache, which had been called migraine, for at least 10 years, in attacks lasting a day or two, about once a month. It was always frontal, sometimes left, sometimes right, sided. It had disappeared during her first pregnancy 18 months before but had since returned, until at the time of admission it was of daily occurrence.

She had been seen twice, four years previously, because of this headache, but the only abnormal sign found was poor reaction of the right pupil to direct light. On the second of these occasions the headache was much improved and further investigation was not advised.

During the last three months she had had eight or nine attacks, each lasting a minute or so, of tingling on the left side of the face, beginning in the lower lip and spreading into the cheek and temple. They were not associated with headache.

For the same period a similar sensation had occasionally affected all the digits of the right hand. This likewise was independent of headache and also of the tingling in the face.

For a few weeks she had noticed blurring of vision. She was right handed.

On examination the abnormal signs were moderate bilateral papilloedema equal on the two sides, a relative right homonymous hemianopia, visual acuity of 6/12 left and 6/24 right, and brisker deep reflexes on the right side.

There was no trigeminal sensory loss, no abnormality of pupillary reflexes, no facial weakness or hemiparesis and no sensory loss on trunk or limbs.

Straight radiographs of the skull showed erosion of the posterior clinoids.

Lumbar C.S.F. pressure was not measured and the fluid contained 66 mg. of protein per 100 ml.

An E.E.G. was abnormal but without slow waves, showing only a less well defined alpha rhythm on the left.

Left carotid arteriography, at which unfortunately only lateral films were obtained, showed merely stretching of some terminal middle cerebral vessels as if from a deep parietal mass. No vessels within the tumour were seen, the posterior communicating artery was small and the posterior cerebral artery not seen. Definition in these films was poor and the anterior choroidal artery could not, with certainty, be identified.

Ventriculography through biparietal burr-holes showed displacement of the lateral and third ventricles to the right with dilatation of the left temporal and frontal horns. In the trigone region of the left ventricle there was a filling defect about 5 cm. in diameter (Figs. 5 and 6). Cerebrospinal fluid from the left ventricle contained 97 mg. of protein per 100 ml.

Vertebral arteriography showed numerous posterior choroidal arteries ascending from the posterior cerebral artery to form a mass of smaller vessels corresponding to the site of the tumour. The normal pattern of the choroid plexus was not seen.

FIG. 5.—Case 6: Antero-posterior ventriculogram showing the dilated temporal horn, air in the elevated trigone, displacement of the ventricles to the right, and dilatation of the anterior part of the body of the left ventricle.

FIG. 6.—Case 6: Lateral ventriculogram showing the tumour (dotted).

At craniotomy, exposing the left parieto-occipital area, the tumour was felt through several needle punctures to be firm and movable and lying about 2 cm. deep to the cortex. A cortical incision some 5 cm. long was made in a postero-anterior direction, beginning posteriorly just medial to the ventriculogram opening, about 3.5 cm. from the mid-line. After deepening the incision and examining the tumour it was considered necessary to enlarge the opening, and this was done, from its anterior extremity, in a downwards direction for 3 cm. After the choroid plexus, entering the antero-inferior aspect of the mass, had been divided, the tumour was removed intact with the aid of traction sutures. A few small vessels on the medial wall of the ventricle then required coagulation and these were the only vascular attachments of the tumour except for the plexus which had previously been divided.

Operation produced a right homonymous hemianopia and an almost complete right hemiplegia. By the tenth day there was no facial weakness and only slight weakness of the arm and leg. Speech slowly improved, but even 14 months after operation it was still impaired, although probably adequate for her needs. Reading, however, was very poor. At this time there was no weakness or postural sense loss in the limbs. The field defect remained complete.

Fits began two days after operation, were at first only right sided, but in the ensuing months were occasionally generalized with loss of consciousness. Suitable medication reduced their frequency to about once every two months.

The tumour weighed 78 g. and measured 6 by 5 by 5 cm. Histological examination showed it to be similar to that of Case 1.

Noteworthy features of this case were the long history of headache and the trigeminal paraesthesiae without objective sensory loss (compare Case 4).

Case 7.—G. S., a man aged 50, a motor mechanic, was admitted to Leeds General Infirmary under Dr. H. Garland in July, 1951, and transferred to Mr. W. R. Henderson's care.

The main features of the history were headache and weakness of the left limbs for a few months.

Headache had started three and a half months before, had become more frequent and severe, and at the time of admission was of daily occurrence. It was always right sided, beginning behind the eye and spreading to the sub-occiput, was present when he wakened in the mornings, disappeared after being up for an hour or so, and was often associated with vomiting.

The left hand had been weak for two and a half months but the weakness had progressed very little. He was still able to grip well but the hand was clumsy for fine movements. For one month he had noticed weakness of the left leg, although this had latterly improved.

During the investigation period in hospital mental confusion appeared and he agreed that this memory was rapidly becoming worse.

There was no history of visual impairment, of fits, or of facial paraesthesiae. He was right handed.

On examination the abnormal signs were complete left homonymous hemianopia, bilateral papilloedema greater on the right, moderate weakness, greater distally, in the left arm and leg and about equal in the two limbs, impaired postural and tactile sense on the left limbs and left side of the trunk, and an extensor plantar response on the left, the right being equivocal.

There was no facial weakness, no trigeminal sensory loss, and no impairment of visual acuity.

Straight radiographs of the skull showed no abnormality.

An E.E.G. recorded continuous low-voltage waves of 2 to 3 per second from the right hemisphere, the maximum disturbance being over the temporal lobe.

A right carotid arteriogram showed moderate displacement of the anterior cerebral artery to the left and,

in the lateral films, slight upwards displacement of middle cerebral vessels and splaying of terminal middle cerebral branches as if from a parietal mass. The posterior cerebral artery was normal. The anterior choroidal artery was larger than normal and ended posteriorly in a tuft of small vessels. No vessels within the tumour were seen.

A right posterior parietal burr-hole was then made and needling towards the trigone encountered from a depth of 2 cm. onwards abnormal resistance from which a biopsy was taken. On several occasions during the next few days he became comatose with a fixed and dilated right pupil but always spontaneously recovered after a few hours.

To obtain more information, vertebral arteriography was done and this showed numerous posterior choroidal vessels ascending from the posterior cerebral artery and apparently stretched over a mass. They did not, however, completely encircle it and no residual haze of contrast was visible. The normal pattern of the choroid plexus was not seen.

Craniotomy was then performed and in the right lower parietal region a horizontal cortical incision about 6 cm. long was made and extended in a curve upwards posteriorly. About a third of the tumour was removed with the diathermy loop and the main mass was then separated from the ventricular wall. A prolongation of the tumour into the temporal horn was seen to be receiving the main vascular supply from the plexus, and when this and a large vein lying posteriorly and passing medially into the choroidal fissure had been divided, the tumour was delivered without bleeding.

The immediate result of the operation was complete hemiplegia which rapidly improved. Three weeks later there was only slight weakness of the left hand and none of the leg, with only slightly impaired postural sense in fingers and toes. The field defect remained unchanged.

About four months after operation he had two generalized fits but had had no more in the ensuing three months. He returned to his previous work, and examination seven months after operation showed no weakness of the left hand and only slight impairment of postural sense in the left fingers.

The tumour weighed 138 g. and measured 8 by 7 by 5 cm. Histologically it was similar to those of Cases 1 and 6.

Of interest in this case were the attacks of coma with spontaneous recovery. These are discussed later.

Case 8.—E. H., a man aged 47, a costing clerk, was admitted to Leeds General Infirmary in October, 1952, under Mr. W. R. Henderson.

He was complaining of impaired memory for about a year, a left field defect for five months, and mild headache for three or four months.

Fifteen months previously he had had while at work an attack of unconsciousness without convulsion, preceded by a feeling of nausea. When he fell he injured the back of his head and had occipital headache for a few days but was then able to resume work. Since this episode he had often seen his doctor because of insomnia, produced,

he thought, by worrying about his work, at which he had become less efficient because of impaired memory.

He had first noticed the left field defect five months previously and he thought it had not become worse during this period.

Headache, which was never severe, had been present for three or four months. It was sub-occipital and above and behind the eyes, not more on one side than the other, usually occurred when wakening in the mornings, gradually disappearing after being up for an hour or so. It had not become more frequent or more severe, appeared only about twice a week, was never associated with vomiting, and never kept him away from work.

He had had no sensory or motor disturbances in limbs or trunk, no facial paraesthesiae and no fits. He was right handed.

On examination the abnormal signs were blurring of the nasal margins of both optic discs with distension of the retinal veins and a left homonymous field defect, absolute to a small object in the lower quadrants but only relative in the upper quadrants. There were no motor, reflex, or sensory changes in limbs or trunk and straight radiographs of the skull were normal.

Lumbar C.S.F. (examined elsewhere) and cisternal C.S.F. were of normal composition.

Cisternal encephalography produced only poor filling of the lateral and third ventricles which were displaced to the left. The larger anterior horn was on the right side and the temporal horns did not fill.

A right carotid arteriogram was then done, but unfortunately only poor films were produced showing displacement of the anterior cerebral artery to the left and splaying of the terminal branches of the middle cerebral artery.

Biopsy, through a burr-hole in the parietal region, showed the tumour to be a meningioma, although this was not suspected at the time because the tumour was very soft and easily aspirated. This operation produced slight impairment of postural sense in the left limbs but no weakness and no increased field defect.

The right carotid arteriogram was then repeated and the antero-posterior and lateral films showed an enlarged anterior choroidal artery ending postero-laterally in a small tuft of vessels (Fig. 7). In addition the lateral films

FIG. 7.—Case 8: Carotid arteriogram showing the enlarged anterior choroidal artery (a.ch.a.) ending posteriorly in a tangle of small vessels.

showed a few small abnormal tumour vessels over an area about 3 cm. in diameter in the region of the trigone.

Vertebral arteriography demonstrated numerous posterior choroidal vessels ascending from the posterior cerebral artery, but no definite outline of the tumour. The normal pattern of the choroid plexus was not seen.

This procedure was followed by craniotomy at which a triangular area of temporo-occipital cortex was excised and the smooth, reddish tumour exposed at a depth of 2 cm. The forward extent of the cortical excision was limited by a large superficial vein. When sufficient of the tumour surface was exposed, its centre was removed by diathermy loop and suction, a procedure which produced profuse bleeding. After the choroid plexus had been divided in the posterior part of the temporal horn and again in the body of the ventricle, the remaining shell of tumour was delivered. Choroid plexus was attached to its surface.

The operation produced no hemiparesis, but joint position sense was, for a few days, lost in the left fingers and toes and the homonymous hemianopia became complete with macular sparing. Postural sense gradually improved and two weeks later was only just detectable. The field defect remained complete.

The remaining shell of tumour measured about 5 by 3 by 2 cm., and with the pieces removed by diathermy weighed 42 g. This was probably about half the weight of the whole tumour, the rest having been sucked away. On the medial aspect of the most anterior part of the tumour in the body of the ventricle was a small nubbin about 0.5 cm. in diameter, which may have been a prolongation through the foramen of Monro. It was impossible to be certain of this at operation because of excessive bleeding.

Histologically the tumour was composed of bundles and whorls of spindle-shaped cells with numerous islands of meningothelial cells and psammoma bodies.

This case was similar to Case 1, in that the only focal sign before operation was the field defect.

Discussion

Diagnosis.—Of the purely clinical diagnosis of these tumours there is little to add to what has already been written by Cushing and Eisenhardt (1938). In their summary they say :

“ One may venture to ascribe a fairly characteristic syndrome to these lateral tumours of the velum, viz. : (1) pressure symptoms with headache tending to be ipsilateral ; (2) a contralateral homonymous hemianopia often bisecting the macula ; (3) a contralateral sensorimotor hemiparesis more marked in the sensory sphere, associated in a few cases with trigeminal numbness ; (4) symptoms suggesting cerebellar involvement in more than half of the cases ; and (5) almost invariably paralexia increased by operation when the tumour, as it commonly does, occupies the left hemisphere.”

Unfortunately analysis of clinical material and attempts at formulating a syndrome characteristic of these tumours are not of great help in diagnosis,

because their signs are the same as those produced by other tumours occupying the posterior part of the cerebral hemisphere. Many gliomas in this situation will fulfil Cushing's conditions (1), (2), (3), and (5). With regard to (4), consideration of all available evidence shows that signs suggesting a cerebellar lesion are present in only about one quarter of the cases and that these signs appear on the same side as the motor and sensory defects. It must be difficult in the presence of weakness and loss of postural sense to attribute ataxia to cerebellar dysfunction. Although it is probable, therefore, that these tumours cannot be diagnosed by clinical examination alone, they should be considered as possibilities when the syndrome outlined above presents itself.

Plain radiographs of the skull, electroencephalography, and analysis of the cerebrospinal fluid are as a rule of little diagnostic help. Occasionally, as in Case 5, the tumour is sufficiently densely calcified to show on radiographs, but such calcification need not necessarily indicate the pathological type of tumour or its intraventricular position. Usually the most that these radiographs show is evidence of increased intracranial pressure and lateral displacement of the pineal gland.

The E.E.G., which was done in seven cases of the present series, recorded slow waves from overlying brain in all cases except Case 6, where the only asymmetry was of the alpha rhythm. In only one instance however (Case 2) was it of real aid. In this case it provided the first definite indication of the supratentorial position of the tumour, localization of which was not possible from clinical signs alone.

Estimation of protein in lumbar or ventricular C.S.F. is likewise of little help. Of the two the latter is more useful, as it sometimes shows a high protein content of the fluid on the side of the tumour, contrasting with a normal amount from the opposite ventricle, e.g., Cases 2 and 6. This information, however, is not usually available until the ventriculograms have been seen.

In most of the cases previously recorded and in five out of six of the present series in which it was done, ventriculography has demonstrated the intraventricular position of the tumour and also its size. On only one occasion was it of no value (Case 1), no air passing into the affected ventricle from the opposite side. Usually the outline of the tumour is demarcated by air lying anterior to it in the body and temporal horn and posterior to it in the occipital horn. The temporal horn is dilated because of C.S.F. obstruction (Fig. 6) and frequently the occipital horn also is enlarged. The smaller anterior horn is sometimes on the same side as the

tumour, for example, Cases 3 and 5, sometimes on the opposite side, for example, Cases 4 and 6. There is usually considerable displacement of the lateral and third ventricles to the opposite side.

On the whole, therefore, ventriculography provides adequate information before craniotomy is undertaken. In recent years, however, carotid arteriography has often been preferred in investigating cases of suspected cerebral hemisphere tumour. In the present series carotid arteriography demonstrated many features of little specific diagnostic value, for example, displacement of the anterior cerebral artery to the opposite side and elevation and splaying of middle cerebral vessels, features which would be shown by any deep parietal mass. One abnormality, however, which may be characteristic of these tumours was shown in four cases (Cases 1, 5, 7, and 8), namely, an enlarged anterior choroidal artery, ending posteriorly in a small tuft or tangle of vessels near where the antero-inferior extremity of the tumour lay in the temporal horn (Fig. 7). In the other case (No. 6) in which this investigation was done, the poor quality of the films did not permit adequate visualization of the anterior choroidal artery. Only once, in Case 8, were vessels seen within the tumour.

Vertebral arteriography, which was done in four cases, showed in the lateral views one constant feature, namely, absence of the normal pattern of the choroid plexus. Even the opposite plexus was not seen, presumably because most of the contrast medium was taken up by vessels entering the tumour. In addition, the posterior choroidal vessels were abnormally numerous (Cases 5, 6, and 8), or stretched (Case 7). In one instance (Case 5), the tumour was outlined both by encircling vessels and a residual haze. The antero-posterior films, as would be expected, were of little help, because in this view the choroidal arteries are foreshortened.

Visual Fields.—In all except two cases of this series (Cases 2 and 5), there was some field defect before operation, varying from visual inattention to complete homonymous hemianopia. That this is due to a lesion of the optic radiation, especially of its upper fibres passing lateral to the trigone, is suggested by the involvement of the lower rather than the upper quadrants.

In all cases after operation hemianopia was complete and remained so, except in Case 4 in which there was subsequent slight improvement. This increase in the hemianopia following operation is presumably due to damage to the radiation; by an incision through it, by interfering with its blood supply, or by bruising during tumour removal. That

this explanation is not the whole story, however, is suggested by Case 2, in which there were full fields before the first (frontal) craniotomy and a complete homonymous hemianopia afterwards.

Facial Paraesthesiae.—In two instances (Cases 4 and 6) sensory attacks in the face on the same side as the tumour were recorded. In Case 4 it was described as a “twitching sensation” not associated with visible movement, and in Case 6 as “tingling”. In neither case was there objective sensory impairment.

Effects of C.S.F. Obstruction.—These tumours are so placed that the normal flow of C.S.F. from the temporal horn is impeded and, since they are to some extent movable, it is reasonable to assume that sometimes complete obstruction will occur. Only rarely in the present series did intense paroxysmal headache feature in the history, but in Case 7 there were transient attacks of coma associated with a fixed and dilated pupil, which may have been due to intermittent blockage of the temporal horn.

Gross Features of the Tumours

Site.—Cushing and Eisenhardt (1938), as already mentioned, differentiate between true plexus meningiomas lying wholly within the ventricle and conforming to its shape, and lateral meningiomas of the velum which tend to be globular and lie partly within the ventricle and partly embedded in the cerebral hemisphere. All the tumours in the present series belong to this latter group. It seems probable, however, that such a differentiation is artificial, for whether or not a tumour lies wholly within the ventricle must, to a great degree, depend on its size. As the tumour enlarges the ventricle will distend, but ultimately the ependyma will rupture and the tumour will then be in contact with the white matter.

A good opportunity for studying one of these tumours *in situ* was afforded by the specimen shown in Fig. 8. Little is known of the history of this woman aged 68, who died suddenly at home, except that for two years she had had fits and dizziness and impaired vision in the left eye (possibly a left homonymous defect). The tumour, which weighed 42 g. and measured 5 by 4 by 3 cm., occupied the trigone of the right lateral ventricle and extended backwards into occipital white matter. Histologically it was composed of bundles of spindle-shaped cells with numerous islands of meningotheial cells. It had two main vascular pedicles, both formed of choroid plexus, one passing forwards and downwards into the temporal horn and one medially towards the junction of the body and the trigone.

FIG. 8.—A meningioma in the trigone of the right lateral ventricle, extending posteriorly into occipital white matter. The tumour has been rotated backwards to show the choroid plexus passing forwards into the temporal horn and into the body of the ventricle. Inset is a diagram for orientation.

In this latter pedicle, which continued along the body of the ventricle towards Monro's foramen, a large choroidal vein was conspicuous. Between the attachments of the pedicles, choroid plexus was firmly adherent to the tumour but did not encircle it. The tumour was freely movable except for its attachment to the plexus and to a few small veins running from its postero-lateral surface directly into the brain. The occipital lobe white matter into which the tumour had burrowed showed, on histological examination, a gliosed surface layer but no ependyma.

Although belonging apparently to the group of "lateral tumours of the velum", it is reasonable to suppose that this tumour, at an earlier stage of development, lay wholly within the ventricle and then extended backwards to embed itself in brain.

Blood Supply.—The normal choroid plexus obtains most of its blood from the posterior choroidal arteries ascending into the velum from the posterior cerebral artery. The terminal branch of the anterior choroidal artery entering the plexus is very small, (Alexander, 1942), and, whereas it is single, the posterior choroidal vessels form a group.

Meningiomas arising from the plexus (or velum), in a similar manner to meningiomas elsewhere, produce enlargement of feeding vessels and this is shown in arteriograms as enlarged anterior choroidal arteries or as undue vascularity in the territory of the posterior choroidal vessels.

Microscopic Features of the Tumours.—Abbott and Courville (1942) pointed out that the 50 or so intraventricular meningiomas recorded up to 1942 had, with only one possible exception, been described as fibrous, fibroblastic, or psammomatous. From this and other observations they deduced that there are two main histological types of meningioma: a fibroblastic type, which they suggest originates in the fibrous stroma of the arachnoid, and a meningothelial or syncytial type arising from the "covering" or "cap" cells which form the outer layer of the arachnoid. The present series of cases shows, however, that meningiomas within the lateral ventricle differ from each other in histological structure as do meningiomas elsewhere. Sometimes fibrous cells predominate (e.g., Case 5), sometimes meningothelial cells (e.g., Case 3), but usually both types are seen in varying proportions.

Methods and Results of Treatment.—The treatment of these meningiomas must consist of their removal by operation. There is no reason to suppose that any type of decompression would be useful and radiotherapy would be ineffective in overcoming the obstruction of the temporal horn.

It is, as a rule, technically easy to remove these tumours completely, because their blood vessels are concentrated in two main pedicles and separation from white matter or ependyma is straightforward. They are easier tumours to remove than, for example, many parasagittal meningiomas.

Why then do the results of operation often compare unfavourably with those of meningiomas elsewhere? One reason, no doubt, is that the surgeon is confronted not only by the unexpected

but also, because of their rarity, by something with which he is unfamiliar. The main reasons, however, are that these tumours are more common on the left side and incision of the overlying brain causes dysphasia.

In most of the published cases and in all of the present series, the tumours were approached by the most direct route. Incisions were made in various parts of the parietal or posterior temporal lobes and deepened until the surface of the tumour was exposed. The separation of the tumour and coagulation of its feeding vessels, as a rule, presented no difficulty.

The usual immediate effects of operation are hemiplegia, gross impairment of discriminative sensations, complete homonymous hemianopia and, if the tumour is in the dominant hemisphere, severe speech disturbance. The hemiplegia rapidly recovers, the sensory loss more slowly and sometimes incompletely, and the hemianopia remains permanently. The ultimate defect, therefore, with a right-sided tumour is a left homonymous hemianopia and perhaps some postural sense loss as in Cases 4, 7, and 8. In all these cases the residual disability was minimal and the patients had resumed their previous employment. The most successful of this group was the patient described as Case 8, in whom the forward extent of the cortical excision was limited by a large superficial vein, a circumstance which although making the operation more difficult no doubt contributed to his rapid neurological recovery. When the approach is through the parietal area a field defect is inevitable, but from Cases 4, 7, and 8 it appears that to cause the least disturbance of discriminative sensation, the cortical incision should be made as far posteriorly and as low as possible.

Following the removal of left-sided tumours, however, there is the added disability of speech and reading, a defect accentuated by the rarity of dysphasia before operation. In all five cases (Cases 1, 2, 3, 5, and 6) some dysphasia will probably be permanent, and in two (Cases 5 and 6) difficulty in reading amounting to almost complete alexia occurred. Despite these defects the labourer (Case 1) and the joiner (Case 2) had resumed their employment and the housewives (Cases 3, 5, and 6) were managing fairly well. Some degree of dysphasia and dyslexia when approaching the tumour through the left posterior parietal region is probably unavoidable

but the disability is lessened by planning the cortical incision on the exposed brain as far back as possible and with the least interference to blood vessels.

In this series of eight, fits occurred in four cases, beginning after operation at intervals varying from a few days to five months. In all cases, however, the fits were reasonably well controlled with medication.

Summary

The available literature on meningiomas arising in the lateral ventricle from the choroid plexus or velum has been reviewed. About 50 of these tumours had previously been described.

Eight additional cases have been described and also a specimen showing one of these tumours *in situ*.

The various diagnostic procedures have been evaluated and certain features of carotid and vertebral arteriograms described.

Operative results have been discussed and measures described to minimize post-operative disability, which is usually severe, especially with the more common left-sided tumours.

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