CASE NOTE

OCULAR MANIFESTATIONS IN A CASE OF HAEMORRHAGE INTO THE FOURTH VENTRICLE*

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PONTINE lesions due to intrapontine haemorrhage are rare. Such cases are always grave but they can usually be diagnosed in life by characteristic signs. The following case had a haemorrhage in the fourth ventricle which produced the signs of ponto-medullary involvement.

Case Report

A male patient, aged 25, came to the Eye Infirmary, Medical College, Calcutta, on August 17, 1951, with a history of sudden loss of vision, headache, and vertigo of about one month's duration. His vision improved within a few days of the attack, but nausea, vertigo, and occipital headache increased. He felt more sleepy than before and lost his taste sensation. During the last week he had found difficulty with speech, deglutition, and micturition. He was unable to close his right eye and felt weakness in the right side of his face. He also complained of impairment of hearing in his right ear. His past history revealed nothing of importance except an attack of fever about one year before.

Examination.—The patient looked ill and was not mentally alert. He answered questions with great difficulty.

Visual acuity was 4/60 in the right eye and 6/18 in the left. It may have been more but the patient was unable to co-operate.

The corneae were clear, and the pupils were small, equal, regular in outline, and reacted to light and convergence. The discs were pink and the veins full; otherwise the fundi were normal. The visual fields were normal as tested by the confrontation method. There was no central scotoma for white and colour.

The right palpebral fissure was wider than the left, and the right eye was slightly convergent.

Horizontal conjugate movements to right and left were absent, the vertical conjugate movements were normal, convergence was normal. Except the loss of outward movements, all other movements—upward, downward, and inward to convergence—were normal. There was no nystagmus.

The right corneal sensation was diminished and the right side of the face had an infranuclear type of facial paralysis. The hearing was defective in the right ear, and taste was lost in the anterior two-thirds of the tongue (posterior part not tested). Movements of soft palate and tongue appeared normal but speech was slurred. There was no defect of smell, and the other cranial nerves appeared normal.

There was weakness in the left hand. The patient could walk and his gait was not ataxic, but there was slight unsteadiness in his gait when his eyes were closed. The bladder was distended and had to be relieved by catheterization. No definite sensory

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disturbance in the body and limbs was detected. The abdominal and plantar reflexes were absent on both sides, but the knee jerk was present and was brisker on the left side.

On August 20, 1951, the patient was drowsy but not unconscious. The ocular movements were the same. Convergence was weak, and the pupils were constricted and unequal, the right being smaller than the left. Both pupils reacted to light. The fundi were the same.

Swallowing had become very difficult and speech was slow, slurring, and almost inaudible. There was some neck rigidity and Kernig's sign was positive. Abdominal and plantar reflexes were absent, but left ankle clonus was present.

Temperature 97.8°F., pulse rate 62, respiration rate 20, blood pressure 100/64.

Haemoglobin 75 per cent., total white blood count 9,000, polymorphs 70 per cent., lymphocytes 29 per cent., eosinophils 1 per cent. Blood sugar 166 mg./100 ml., urea 45 mg./100 ml., NPN 35 mg./100 ml., Kahn test negative. Sedimentation rate 12 mm./hr., bleeding time 3 min., coagulation time 4½ min., urine nothing abnormal.

Therapy.—Systemic penicillin was given, and as the pulse rate slowed down to 54 lumbar puncture was done. 14 ml. blood-stained cerebrospinal fluid were withdrawn under moderate pressure, cell count 600/ml., Wassermann reaction negative.

On August 21, 1951, the patient was drowsy but still conscious. Pulse rate improved to 78 after lumbar puncture. Temperature 98.8°F. Motor function as before. Painful sensations had diminished in the limbs and body, particularly on the left side.

The pupils were constricted and unequal, the left being now smaller than the right; reaction to light was sluggish, and convergence reaction impossible to judge. Vertical conjugate movements were present, lateral conjugate movements absent, no nystagmus. Fundi normal apart from pink discs and congested veins.

The next day the patient was semi-conscious. The pupils were constricted, the left almost to a pin-point, and both were inactive to light. The discs were intensely congested, with slight blurring of the upper nasal margin of the right disc and the upper margin of the left disc. Temperature 97.4°F, pulse rate 62, respiration rate 18.

Therapy.—A second lumbar puncture was done when the pulse rate was 40, and 10 ml. blood-stained cerebrospinal fluid under slight pressure were withdrawn. Lumbar puncture did not improve the condition and the patient died on August 23, 1951, 6 days after admission.

Post-Mortem Findings.—There was a small blood clot in the subarachnoid space of the cisterna cerebello-medullaris in the posterior fossa, and another blocking the right cerebello-pontine angle and extending to the roots of the right seventh and eighth nerves (Fig. 1). The superficial surfaces and base of the brain, the optic nerves, the chiasma, and the optic tracts appeared normal. On sagittal section of the brain, dividing the brainstem, cerebellum, and cerebral hemispheres into two halves, a large blood clot (2" × 1") was found to fill the cavity of the fourth ventricle which was itself very much distended (Fig. 2). The clot extended as a wedge upwards into the lower part of the cerebral aqueduct, while the upper part of the aqueduct was completely closed by the oedematous brain substance, which prevented entrance of any blood into the third ventricle. The ventricular clot extended downwards into the central canal of the medulla, and laterally into the lateral recesses of the fourth ventricle. On the right side the clot protruded into the right cerebello-pontine angle and extended to the roots of the right seventh and eighth nerves. Posteriorly the blood clot was firmly apposed to the ventral surface of the cerebellum, while anteriorly it severely compressed the floor of the fourth ventricle, forming an angulation at the ponto-medullary junction. The cut surfaces of the brainstem showed the blood clot penetrating for a depth of 3 or 4 mm. into the brain substance at the place of angulation. The lateral ventricles, the third ventricle, and the cut surfaces of the cerebrum and cerebellum appeared normal. No

FIG. 1.—Base of brain. Arrow indicates blood clot at right cerebello-pontine angle.

FIG. 2.—Cerebral hemispheres, cerebellum, and brainstem in sagittal section. Cavity of fourth ventricle completely filled by blood clot, which has compressed floor of fourth ventricle and plugged lower part of cerebral aqueduct, the upper part being closed by oedematous brain tissue.

diseased vessel or aneurysm was seen macroscopically in any cerebral vessel at the base or on the surfaces of the brain.

Discussion

The clinical findings of the case showed that there was in the early stage of the disease paralysis of the fifth, sixth, seventh, and eighth nerves of the right side, and conjugate horizontal gaze paralysis of both sides. Later there was involvement of the ninth and tenth nerves, the sympathetic and pyramidal fibres, and finally of the sensory fibres and cerebellum. Autopsy findings explain all the clinical signs. Pressure on the floor of the fourth ventricle by the blood

clot affected the cranial nerve nuclei or their fibres. Further pressure can involve the medial longitudinal bundle, supra-nuclear nuclei, or their path for horizontal conjugate movements, sympathetic, pyramidal, and sensory fibres. The right seventh and eighth nerves can also be affected at their roots by a blood clot at the cerebello-pontine angle where the pyramidal fibres may also be involved.

No explanation could be found for the absence of high temperature which is

said to be a very characteristic sign in pontine haemorrhage. Probably this was due to the failure of blood to enter the third ventricle and

damage the hypothalamic thermal centre as the cerebral aqueduct was blocked by the oedematous brain tissue. The cause of sudden loss of vision is also difficult to explain. Perhaps it was due to reflex vasospasm of the blood vessels supplying the visual pathways—an attempt by nature to stop the haemorrhage? Another notable feature was the absence of nystagmus, though the pathological process had in all probability involved the vestibular nuclei and the medial longitudinal fasciculi. The final difficulty is to explain the cause and source of haemorrhage in the absence of any diseased vessel or cerebral aneurysm. Intrapontine haemorrhage from the median pontine branches of the basilar artery may in rare cases burst into the cavity of the fourth ventricle, but the cut surface of the pons did not show much destruction of the pontine tissue except at the dorsal surface of the pons. Though primary ventricular haemorrhage is rare, the haemorrhage in the present case may have come from the bursting of a congenital aneurysm in connection with a minute vessel on the floor of the fourth ventricle or from the choroidal plexus of the fourth ventricle, but the immediate cause of rupture was not understood as the blood pressure was low.

Summary

A case of haemorrhage in the fourth ventricle with autopsy findings is described. The clinical picture was that of a pontine lesion, except that the patient had no rise of temperature. The sudden loss of vision at the onset of the disease was not clearly understood, but a possible mechanism is suggested.