Medical Memorandum

Cerebral Abscess Complicating Congenital Heart Disease

Cerebral abscess as a complication of congenital heart disease is rare. A few cases have been reported in the early literature, but the association of the two conditions was not firmly established till Ballet (1880) showed that in some cases of congenital heart disease death was due to rupture of a cerebral abscess. Abbott *et al.* (1923) collected six cases of the tetralogy of Fallot in which a cerebral abscess caused death, and Wechsler and Kaplan (1940) reported two similar cases. Up to 1940 only 14 cases of congenital heart disease with cerebral abscess had been reported, and in only three of these had the abscess been diagnosed during life. Hanna (1941), out of 160 cases of congenital heart disease coming to necropsy, found six complicated by cerebral abscess, in three suspected before death. Robbins (1945), from a study of 53 necropsies, reported three cases of cerebral abscess diagnosed during life; he also referred to 23 cases in the literature, in three of which the abscess was diagnosed during life.

Experiments have shown that a cerebral abscess can be produced only by implantation of organisms into an area of the brain previously injured or damaged by interference with its blood supply. The latter may be due to thrombosis of a cerebral artery in patients with cyanotic congenital heart disease, or to an embolus from a distant focus reaching the brain because of an atrial or ventricular septal defect (paradoxical embolism). Abscess of the brain is most commonly associated with the tetralogy of Fallot; 13 of the 53 cases reported by Robbins (1945) were of this type. That it occurs more frequently in the right hemisphere is believed to be due to the dextroposition of the aorta.

Modern treatment with antibiotics alone or in combination with surgery has given better results than surgery alone, and nowadays the prognosis is much more favourable. The first case to be treated successfully by surgical drainage and sulphonamides was reported by Smolik *et al.* (1946). Other cases in which success has followed treatment with penicillin, streptomycin, and surgical drainage and excision have also been reported (Neill, 1955). With the availability of these new methods of treatment physicians should be alive to the possibility that an abscess may be present whenever an intracranial complication is suspected in a patient with congenital heart disease. In such a case the sudden onset of symptoms and signs pointing to a focal lesion in the brain, together with fever, vomiting, leucocytosis, and papilloedema, should suggest this complication. Lumbar puncture may help to confirm the diagnosis, especially if the abscess has ruptured and caused a meningitis.

Below is described a case of cerebral abscess complicating the tetralogy of Fallot with a fatal outcome, to which inadequate treatment was doubtless a contributory factor.

CASE REPORT

A Sinhalese girl of 15 was brought to hospital for frontal headache of six weeks' duration with vomiting for one week. She had had generalized swelling of the body eight weeks earlier, which subsided completely. Two weeks later headache with weakness of the left side of the body and swelling of the right eye developed. Clubbing of the fingers and cyanotic attacks had been present from early childhood. There was no history of convulsions.

On admission the patient was rational but irritable and complained of a frontal headache. Her temperature was 99° F. (37.2° C.). Her fingers and toes were clubbed and cyanosed. The heart was enlarged, and the apex beat was in the fifth left intercostal space, 1 in. (2.5 cm.) lateral to the midclavicular line. There were no thrills. Both sounds were well heard in all areas. There was a rough systolic murmur maximal in the third left intercostal space,

near the sternum. Blood pressure was 100/60 mm. Hg. The lungs were clear and the liver and spleen not palpable. There was complete ptosis of the right upper lid with engorgement of the veins of the lid and bilateral papilloedema. The eye was not proptosed. The pupil was dilated and did not react to light. Eye movements were absent. There was a flaccid paralysis of the left half of the body without any sensory impairment. The deep reflexes could not be elicited on the left side, but the plantar reflex was extensor. Total leucocytes numbered 11,600 per c.mm., with 80% polymorphs and 18% lymphocytes. The haemoglobin level was 80% (Haldane). The urine contained no albumin or sugar, but there were pus cells numbering 4-5 per field. Radiography of the chest showed the *cœur-en-sabot* appearance of the heart and absence of the pulmonary artery shadow, though vascular markings were present in the lungs. The electrocardiogram showed right axis deviation. The skull was radiologically normal. The clinical diagnosis was tetralogy of Fallot with a cerebral lesion, probably an abscess.

Treatment with penicillin, 500,000 units six-hourly, was started immediately, but consent for surgery was not granted. The temperature settled slowly, though the general condition did not improve. The headache persisted and the patient became drowsy. Lumbar puncture was performed on the third day. The cerebrospinal fluid was under tension and turbid; protein content 100 mg. and chlorides 750 mg. per 100 ml.; polymorphs and lymphocytes present. The next day the drowsiness increased; there were crepitations in the lungs, and the patient gradually became comatose and died.

On post-mortem examination the heart was found to be enlarged, with hypertrophy of the right ventricle. There was a defect in the interventricular septum and dextroposition of the aorta. Several small openings were present in the interatrial septum. The pulmonary artery was narrow and thick-walled. The valves were normal. The lungs were congested, with patches of consolidation in the lower lobes. In the brain the meninges were congested over the right hemisphere, with some flattening of the convolutions and congestion of the superficial vessels. There was a large abscess involving the frontal and parietal lobes on the right side. The cortex was a thin shell. The abscess communicated with the right lateral ventricle, which contained a small quantity of light-green pus. Identification of the infecting organism by culture was not possible.

COMMENT

In the above case the abscess was in the right cerebral hemisphere and associated with the tetralogy of Fallot, the type of congenital heart disease with which cerebral abscess most commonly occurs. Such abscesses are usually caused by an infected embolus or thrombosis of a cerebral artery with secondary infection of the infarcted area. There is nothing in the present case to indicate how the abscess formed, and no evidence of a peripheral focus from which an embolus could arise. That the abscess had attained such dimensions suggests that it had developed over a fairly long period of time. Because it was so large treatment with penicillin was inadequate. Surgical drainage should have been tried, but this was impossible in the absence of the parents' consent.

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REFERENCES

- Abbott, M. E., Lewis, D. S., and Beattie, W. W. (1923). *Amer. J. med. Sci.*, 165, 636.
Ballet, G. (1880). *Arch. gén. Méd.*, 145, 659. Cited by Hanna.
Hanna, R. (1941). *Amer. J. Dis. Child.*, 62, 555.
Neill, C. L. (1955). *Mississippi Dr.*, 32, 226.
Robbins, S. L. (1945). *Arch. intern. Med.*, 75, 279.
Smolik, E. A., Blattner, R. J., and Heys, F. M. (1946). *J. Amer. med. Ass.*, 130, 145.
Wechsler, I. S., and Kaplan, A. (1940). *Arch. intern. Med.*, 66, 1282.