Recurrent cerebral abscess in tetralogy of Fallot

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Tetralogy of Fallot (TF) classically consists of the combination of right ventricular outflow obstruction (pulmonary stenosis), ventricular septal defect (VSD), overriding aorta, and right ventricular hypertrophy. The degree of pulmonary stenosis and VSD determine the variety of clinical manifestations.

This type of congenital heart disease accounts for about 10% of all congenital cardiac deformities and is the most common cyanotic lesion after the first year of life. Cerebral abscess is a serious complication in TF and is usually seen after the age of 2 years.

Report of the case

A 13-year-old boy was admitted to the Pediatric Ward of Wahidin Sudirohusodo Hospital, Makassar due to unconsciousness and generalized seizure. There was no fever. The child complained of headache and vomiting since two days before admission. Poor intake of fluid and food was found. Cyanosis had been noted since he was 7 months of age. There were activity intolerance and squatting.

The physical examination revealed a severely ill, well-nourished, and unconscious child, GCS 11 (E3M5V3). Blood pressure and pulse were not measurable; body temperature was 37.4°C. Central cyanosis was noted. The pupils were isochoric; the light reflex was normal. Meningeal signs were not found. Cardiac examination revealed an apical impulse on the left fifth intercostal space at the left anterior axillary line. The first heart sound was normal and the second sound was single. Ejection systolic murmur grade III–IV/VI was heard on the fourth intercostal space of the left sternal border, which was transmitted to the left axilla and left and right sternal borders. Liver and spleen were not palpable. Clubbing of the fingers was noted. Femoral artery was not palpable.

Blood investigations revealed a hemoglobin level of 19.1 g/dl and erythrocyte count of 6,081,000/µl, WBC was 11,500/µl with 80% segments, 13.5% lymphocytes, and 6.5% monocytes. Platelet count was 23,000/µl and hematocrit was 63%. Feces and urine studies showed normal results.

We performed lumbar puncture. The cerebrospinal fluid (CSF) was clear with CSF glucose/blood glucose ratio of 0.45, protein level of 45 mg/dl, and cell count of 3/µl.

The chest X-ray showed normal heart size and configuration, cardio-thoracic index was 0.5. The electrocardiogram revealed sinus rhythm with a QRS complex of 80/min, right axis deviation, and right ventricular hypertrophy.

Echocardiography showed a ventricular septal defect, overriding of the aorta, pulmonary stenosis, and right ventricular hypertrophy. Computed tomography (CT) scan of the head showed a cerebral mass.

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in the left frontal lobe (Figure 1). The patient was diagnosed as having tetralogy of Fallot and suspected cerebral abscess.

Surgery was performed and about 50 ml of purulent abscess materials were removed. Bacteriologic analysis of the materials showed no growth in the culture. The histopathological examination noted a wide necrotic inflammation and hyperemic areas in the central nervous system tissue and inflammation infiltrates consisted of polymorphonuclear and mononuclear leukocytes, especially lymphocytes and plasma cells. The patient was therefore diagnosed as having tetralogy of Fallot and cerebral abscess.

The treatment consisted of intravenous fluid drips to overcome the hypovolemic shock and intravenous cefepime 750 mg, twice a day for 7 days to cover the possible cause of the abscess. The patient was finally discharged in a good condition after 30 days of hospitalization.

Three months later the patient was hospitalized again due to headache and vomiting. CT scan of the head again showed a cystic mass in the left frontal lobe suggesting cerebral abscess (Figure 2). An abscess excision was performed and Acinetobacter calcoaceticus was found in the culture. He received ceftriaxone 1 gram every 8 hours and was discharged in good condition after 14 days of hospitalization.

Heart catheterization showed severe infundibular stenosis. Total correction of TF was performed. One year after, the patient presented without any complaint.

Discussion

The diagnosis of TF in this patient was confirmed by echocardiogram which showed a VSD, right ventricular hypertrophy, overriding aorta, and pulmonary stenosis. On catheterization, a severe infundibular stenosis was found.

Two important defects of TF—a large ventricular septal defect (VSD) and infundibular pulmonary stenosis—determine the severity of the right-to-left shunt.2,4 The degree of right-to-left shunt is responsible for the hypoxia of cerebral tissue which could lead to cerebral infarction3 which is considered as a precursor of cerebral abscess. Headache, vomiting, convulsion, and unconsciousness might be the signs of cerebral abscess. The head CT scan of the patient showed a cystic mass in the left frontal lobe (Figure 1), which was confirmed as an abscess by surgery.

Polycythemia superimposed by dehydration is responsible for the hypoxic area of the cerebral tissue.3 Infected emboli from the systemic venous reservoir is carried across the ventricular defect to the hypoxic area, contributing for the abscess formation.3,5,6 The source of infection may come from teeth, ear, nose and throat, upper respiratory tract, or skin infections.6

The treatment of cerebral abscess is surgical intervention. This procedure is needed not only to remove the purulent materials, but also to confirm the diagnosis. The organism which is found
from the abscess is usually $\alpha$-Streptococcus besides Staphylococcus aureus, Haemophilus influenzae, Pseudomonas aeruginosa, Gram-negative and Gram-positive rods.\textsuperscript{1,3,6} In this case, surgical treatment was performed but no growth of bacteria had been found in the culture. The histopathological examination confirmed the presence of cerebral abscess. The patient was given cefepime, a bactericidal agent that has a broad spectrum activity against a wide range of gram-positive and gram-negative bacteria, including most strains resistant to aminoglycosides.\textsuperscript{7}

Three months later, the patient had recurrent abscess in the same place which was proven by the head CT scan. Abscess excision was again performed, and Acinetobacter calcoaceticus was found in the culture. The patient was given ceftriaxone, and was then discharged in a good condition after 14 days of hospitalization.

Complete correction of heart defect was done and after one year follow-up, no signs of sequelae or any complaint were seen.

References