Pyogenic ventriculitis secondary to ruptured brain abscess

Clinical Presentation

A 10-year-old girl was followed-up over 3 years for a cyanogen cardiopathy. She was admitted to the neurosurgical emergency department for signs of intracranial hypertension, which occurred 7 days earlier in the context of hyperthermia. The physical examination recorded a dyspneic febrile child at 39.5°C, lethargic having a Glasgow Coma Scale of 12. She also had marked nuchal rigidity and equal pupillary reflex. The neurological examination did not reveal any deficit. The cardiac auscultation registered a 3/6 systolic murmur at the left lower sternal border and apex. A CT scan with contrast demonstrated a left frontal collection with regular contour and wall enhancement; the collection was communicating with the right lateral ventricle. It also showed intraventricular fragments, with clear contrast enhancement associated to an enhancement of the ependymal wall (Figure 1). The biological examination showed inflammatory syndrome (sedimentation rate of 80 mm; C-reactive protein of 112ng/ml). Cardiac ultrasonography revealed interventricular communication.

Questions:

Question 1: What’s your diagnosis?

Question 2: What is the advised treatment?
Neurosciences Quiz

Answers

1. Ventriculitis secondary to ruptured brain abscess.

2. Surgical evacuation of the left frontal abscess associated to systemic antibiotics and instillation of gentamicin via an external ventricular catheter inserted in the right lateral ventricle.

Discussion

Recent improvement in imaging technologies, antibiotics, and surgical techniques have manifested healthcare; nevertheless, brain abscess remains a serious pathology in developed countries as the diagnosis and management are markedly delayed. Abscess rupture within the ventricle or subarachnoid spaces is an exceptional but critical complication because of its high mortality. Therefore, pyogenic ventriculitis constitutes a rare infection of the CNS characterized by the presence of suppurative fluid in the ventricles. Microorganisms mostly identified as being associated to pyogenic ventriculitis, are gram-negative bacteria, including bacteroides, Escherichia coli, and Klebsiella. These are followed by gram-positive microorganisms, such as the common Staphylococcus species. Clinical presentation is nonspecific and includes symptoms of acute intracranial hypertension associated with high fever and frequently disturbance of consciousness. Nuchal rigidity, cranial nerve paralysis, and focal neurological deficits; an extensor plantar response may be found on physical examination. Neuroimaging is the unique reliable tool for diagnosing this delicate condition. A CT scan is the exam of choice and convenient in the emergency context. The CT findings of pyogenic ventriculitis are hydrocephalus, pial enhancement, and debris in the ventricles. Subependymal enhancement on contrast enhanced CT is a key diagnostic finding for ventriculitis. An MRI is more sensitive and shows periventricular high signal on FLAIR images, ependymal enhancement and mostly pial or dura-arachnoids’ pathology. Irregular intraventricular debris seems to be the most specific abnormality. The treatment of this pathology consists of ventricular drainage, intraventricular instillation of antibiotics, and systemic anti-infection therapy. A high-dose of intravenous antibiotics must be administrated over a protracted period during the following weeks. In case of deterioration despite intravenous therapy, intraventricular administration via an Ommaya reservoir must be considered. Finally, pyogenic ventriculitis remains uncommon but constitutes a serious intracranial infection requiring early diagnosis and treatment. Although it is almost considered as universally fatal, some cures may be achieved with prompt treatment.

References