'XULQJ WKH ¿UVW WKUHH PRQWKV RI OLIH \*URXS % VWUHSWRFRFFXV RU \$JDOD associated with cerebrovascular accidents resulting from sepsis and infection of the central nervous system. This DUWLFOH SUHVHQWV WKH XQXVXDO FDVH RI DIHPDOH LQIDQW ZKR ZDV DIÀLFWHG

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hydrocephalus, abscesses, or empyemas. When suspected, it is generalizations in neurodevelopment at the time of their 18-month follow-recommended to exclude intracranial hypertension before performingp. In 18% of these patients, these alterations are moderate to severe a lumbar puncture [8].

e long-term evolution of patients with cerebral abscesses is unknown

Neuroimaging helps nd associated complications suspected from improved with rehabilitative therapy. Factors that have been related to the clinical signs. Early ndings from brain computed tomography death or severe deterioration are seizures, severe alteration of the state can be mild ventriculomegaly and increased subarachnoid spaces of consciousness, respiratory distress, bulging fontanelle, I is T1.2uc early cerebritis stage shows a poorly de ned subcortical hyperintense zone that can be observed on T2-weighted imaging [9,10]. Lesions appearing hyperintense on di usion-weighted imaging with apparent-di usion-coe cient (ADC) values of <0.9 are likely to be brain abscesses, whereas hypointense lesions on di usion-weighted imaging with ADC values > 2 are more likely to be non-abscess cystic lesions [9]. Contrast-enhanced T1-weighted studies demonstrate poorly delineated enhancing areas within the isointense to mildly hypointense edematous regions [9].

During the late cerebritis stage, the central necrotic area is hyperintense to brain tissue in the proton-density and T2-weighted sequences. e thick, somewhat irregularly marginated rim appears isointense to mildly hyperintense on spin-echo T1-weighted images and isointense to relatively hypointense on proton-density and T2-weighted scans. Rim enhances a er contrast administration, peripheral edema and satellite lesions can be observed [9].

During both the early and late capsule stages, the collagenous abscess capsule is visible prior to contrast as a comparatively thin-walled, isointense to slightly hyperintense ring that becomes hypointense on T2-weighted MRIs [10], with marked di usion restriction on di usion-weighted images (high DWI signal and low values in the ADC maps within the abscess). If a cerebral abscess ruptures into the ventricular system, purulent material within the ventricle appears similar to that within the central abscess cavity, with di usion restriction on DWI [9].

For treatment, use of broad-spectrum antibiotics should be established empirically. Antibiotics typically used are third or fourth generation cephalosporins (ce riaxone or cefepime), vancomycin, and metronidazole. Subsequent treatment should be selected according to the pathogen and antibiotic sensitivity. e isolated pathogen usually responds to penicillin or ampicillin. e duration of therapy is four to six weeks for abscesses treated surgically and six to eight weeks for patients who only receive medication. When the abscess measures less than 2.5 cm in diameter or when there are multiple small abscesses, treatment with medication without surgical intervention is recommended. Patients with brain abscesses less than 2.5 cm treated only with antibiotics should see improvement [10].

Systematic review and meta-analysis show that, long-term 32% of patients with a history of group B streptococcal meningitis had

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