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Non-typhoid *Salmonella* Meningitis Complicated by a Infarction of Basal Ganglia

A previously healthy 16-month-old Korean girl with symptoms of fever, vomiting, and generalized tonic seizure was diagnosed to have Group D non-typhoid *Salmonella* meningitis. The patient was treated with ceftriaxone (100 mg/kg/day) and amikin (22.5 mg/kg/day) initially and ciprofloxacin (30 mg/kg/day) was added later because of clinical deterioration and disseminated intravascular coagulation. Brain CT performed on the second day showed a well-demarcated low density lesion in the right lentiform nucleus and both caudate nuclei, without evidence of increased intracranial pressure. MRI performed on the 11th day confirmed CT scan findings as well as right subdural fluid collection, brain atrophy, and ventriculomegaly. She underwent subdural drainage and later ventriculo-peritoneal shunt operation. Despite receiving intensive treatment, she still has severe neurologic sequelae. Our case shows that infarctions of basal ganglia and thalami are not specific for tuberculous meningitis and that meningitis complicated by infarction is indicative of grave prognosis.

Key Words: *Meningitis, bacterial; Salmonella; Cerebral infarction*

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INTRODUCTION

Focal or widespread brain infarctions are not uncommon complications, which are demonstrated in one-third of children with bacterial meningitis complicated by seizure, hemiparesis or coma (1). On the other hand, the infarction area of medial striate and thalamoperforating arteries have been known as the "TB zone" specific for tuberculous meningitis (2).

We report a case with typical neuroimaging changes of infarctions of the left lentiform nucleus and both caudate nuclei complicated by *Salmonella* meningitis.

CASE REPORT

A previously healthy 16-month-old Korean girl presented to the emergency room with complaints of fever, vomiting, and generalized tonic seizure. On admission, her body weight was 11 kg, and her vital signs were as follows: temperature 38℃; heart rate 140/min; and respiratory rate, 56/min. On neurologic examination she was obtunded, and her pupils measured 3 mm equal in diameter and poorly reactive. There was no focal neurologic sign. A cerebrospinal fluid (CSF) study disclosed that milkish CSF contained 3,200 leukocytes/μL (lymphocyte 62%, neutrophil 38%), protein 169 mg/dL, and glucose 1 mg/dL. Antigen of the CSF by latex agglutination was negative for *Hemophilus influenzae*, *Neisserria meningitidis*, and *Streptococcus pneumoniae*. Gram stain of CSF showed numerous gram negative bacilli.

The patient was treated initially with ceftriaxone (100 mg/kg/day) and amikin (22.5 mg/kg/day). Additionally, dexamethasone and mannitol were administered. After admission, seizures consisting of focal clonic movements of the right hand and foot were developed frequently and were refractory to phenobarbital (two times loading) and diphenylhydantoin. Seizure were controlled after administration of intravenous depakine (30 mg/kg/day). On the second day, CSF culture grew *Salmonella* species, which was confirmed by API20F (Biomerieux Co, France) and VITEK (Biomerieux Co, U.S.A.), and susceptible to ceftriaxon and amikin. Later, group D non-typhoid *Salmonella* was identified by serological grouping. Blood, urine and stool culture were negative. Ciprofloxacin (30 mg/kg/day) was added on day 3 because of persistent fever and DIC. On the ninth day, CSF cleared. Amikin and ciprofloxacin were withdrawn after 14 days and ceftriaxon was continued for 6 weeks.

Brain CT performed on the second day showed well-

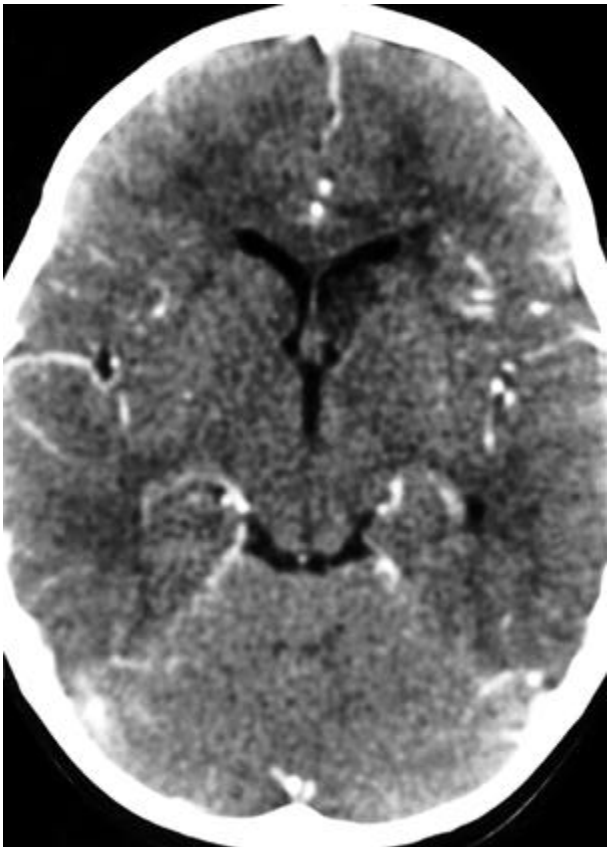

Fig. 1. Post-contrast axial CT scan reveals well-defined low density lesion on both head of caudate nuclei and left lentiform nucleus. In the left frontal lobe, periventricular white matter area, a subtle low-density lesion is shown.

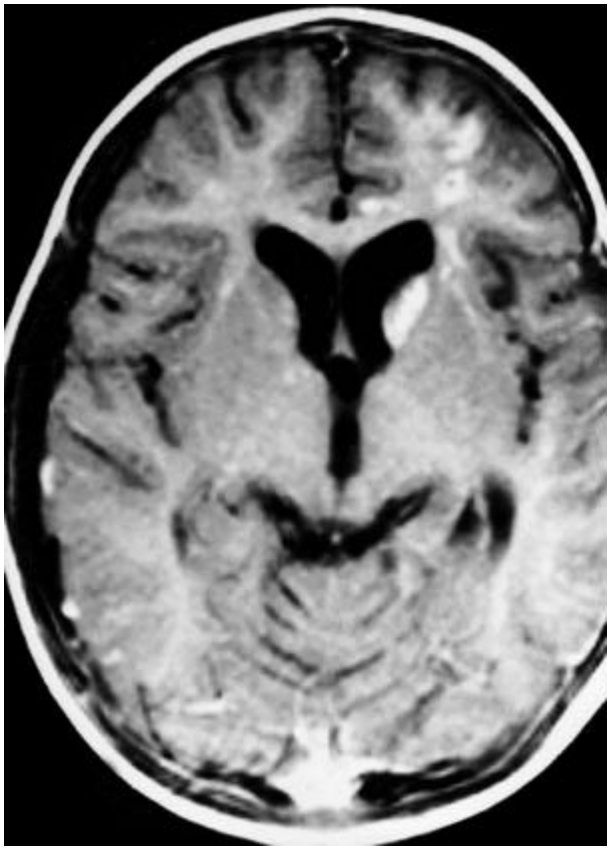

Fig. 2. Gd-enhanced T1 weighted axial image (11 days after CT scan) shows well-defined enhanced lesion on the left caudate nucleus head and left frontal lobe, which correspond to the low density in CT scan. A moderate amount of subdural effusion on the right temporoparietal area and moderate dilatation of the ventricular system are demonstrated.

demarcated low density lesions of the left lentiform nucleus and both caudate nuclei (Fig. 1). MRI was performed on the 11th day, revealing infarction of left lentiform nucleus and both caudate nuclei, right subdural fluid collection, and ventriculomegaly (Fig. 2). On the 30th day, follow-up MRI showed an increased amount of right subdural effusion and more dilated ventricular system. Evacuation of subdural effusion was performed to alleviate left hemiplegia. On the 60th day, follow-up CT revealed further increase in ventricular size. Ventriculo-peritoneal shunt was placed on the 65th day. These days, after 13 months, she cannot make eye-contact and her gross motor skills are limited to 3-4 month level.

DISCUSSION

This report describes a patient with typical neuroimaging changes of infarction of the right lentiform nucleus and both caudate nuclei, complicating *Salmonella* meningitis.

Salmonella meningitis is a rare clinical entity that occurs mainly during early infancy (3, 4). However, in many areas of the developing world, *Salmonella* species account for more than 50% of the gram-negative organisms isolated from cerebrospinal fluid (5). *Salmonella* etiology was established in 3.43% and 12.4% of bacterial meningitis in Ludhiana, India (6) and in Bangkok, Thailand (7), respectively. Most of them were children under two years of age. Response of *Salmomella* meningitis to conventional therapy is slow with frequent complications and recurrences and mortality rates of 60 to 80% are common (5, 8). Huang et al. (9) reported that 11 out of 15 patients with *Salmonella* meningitis had neuroimaging abnormalities.

When meningitis is associated with infarction, the prognosis is poor with hemiparesis, cognitive difficulties, and speech problems (10). Focal or widespread brain infarction as a sequellae of bacterial meningitis have been

demonstrated by computed tomography in one-third of children with meningitis complicated by seizure, hemiparesis or coma (1). Usually, the infarction area of medial striate and thalamoperforating arteries is specific for tuberculous meningitis (2). In tuberculous meningitis, the exudate infiltrates the walls of meningeal blood vessels and extends along small cortical vessels (tuberculous arteritis), resulting in occlusion and infarction (11). Postmortem examination reveals that basal ganglia and thalamus in the region of the lenticulostriate and thalamoperforating arteries are involved in 46% of tuberculous meningitis (12). Hsieh et al. (2) reported that among 14 patients with tuberculous meningitis complicated with infarction, 75% of infarctions occurred in the region of the lenticulostriate and thalamoperforating arteries. Schoeman et al. (13) reported that the basal ganglia infarction occurred unilaterally (21%) or bilaterally (10%) on admission and during treatment (22%) among 198 children with tuberculous meningitis. The main cause of permanent neurologic disability was basal ganglia infarction.

To our knowledge, there has been only one report of brain infarction following *Salmonella* meningitis (14). Infarctions in this case were presumed to be due to diffuse arterial vasculitis. Our case showed that infarctions of basal ganglia and thalami are not specific of tuberculous meningitis and infarction of the lentiform nucleus and thalami complicating meningitis is indicative of grave prognosis.

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