
Uncommon Neurologic Complications of Burns in Infants: A Parkinsonian Extrapyrarnidal Disorder and Massive Cerebral Infarction

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We report uncommon neurologic complications of moderate to severe burns in two infants aged 13 and 19 months, respectively. The first patient suffered a 25% total body surface area burn to her lower limbs; 3 days later she became mute and irritable, with increasing rigidity of limbs and trunk. Her face was expressionless and she also had mild dysphagea. Muscle-stretch reflexes were brisk and accompanied by episodes of coarse jitteriness. A diagnosis of an extrapyramidal parkinsonian disorder was made. The infant was treated with amantadine hydrochloride, and she recovered completely over a period of 2 months. The second infant was admitted with an extensive burn to his trunk and limbs; 3 days later he became comatose and had intractable seizures, which were more prominent over his right side. Dysphasia and right-sided hemiplegia became apparent; these conditions were and remained permanent, accompanied by focal seizures and a marked cognitive delay. Although relatively uncommon, central nervous system involvement may be a hazardous complication of burns in infants, who thus represent a high-risk group. (*J BURN CARE REHABIL* 1991;12:54-7)

Neurologic complications are currently uncommon in both children and adults with burns. Cogswell and Chu¹ did not report even a single case with neurologic complications in their study series of 580 children with severe burns. A generalized encephalopathy is the most common complication of burns in children, clinically represented by alterations of consciousness, hallucinations, and seizures.²⁻⁴ Transient focal deficits were also reported in three of 20 patients by Antoon and coworkers.² Young children may occasionally experience cerebral edema, intractable seizures, and hemiplegia after even minor burns.⁵

We report here on the cases of two infants with moderately severe burns who had unusual neurologic complications. An extrapyramidal parkinsonian syndrome was present in the first infant and massive cerebral infarction was present in the second.

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CASE REPORTS

Patient 1

A 13-month-old, previously healthy female infant was admitted with a second- to third-degree burn to her lower extremities and buttocks, approximately 20% to 25% total body surface area (TBSA). On admission she was fully alert and responsive, with a normal neurologic examination. Topical treatment with silver nitrate applications and baths was initiated. On the third day she began having recurrent episodes of high-grade fever. Repeated blood cultures remained sterile. On the fifth day she became mute and withdrawn. Eye contact with this patient could not be easily achieved, she showed no interest in her surroundings, and she intermittently uttered high-pitched cries. Her face became expressionless, with intact ocular movements. She maintained a severely flexed and rigid posture of limbs and neck (Figure 1). Recurrent vigorous episodes of coarse jitteriness involving mainly the right upper limb were noted both either spontaneously and in reaction to tactile stimuli. Examination revealed marked cogwheel rigidity of her limbs, trunk, and neck muscles. When pulled to a sitting position, she came forward like a log. Muscle-stretch reflexes were brisk, with extensor plantar response.

Laboratory data on admission disclosed a marked hyperglycemia with a glucose level of 540 mg/dl and a leukocytosis of 43,000/mm³, all of which gradually subsided. Her sodium level on admission was 125 mEq/L and was rapidly corrected over 4 hours to 135 mEq/L. Her sodium and glucose levels remained normal thereafter.

The marked hypomimia, severe truncal rigidity, cogwheel limb rigidity, brisk muscle-stretch reflexes, and withdrawn behavior suggested a parkinsonian extrapyramidal syndrome. An electroencephalogram showed mild generalized slowing. Magnetic resonance imaging of the head was unremarkable. No response was noted after a trial therapy with phenobarbital and clonazepam. Oral amantadine was then administered at a dose of 5 mg/kg; this treatment resulted in overt improvement over the course of a week. She became less irritable, more alert, and more interested in her surroundings. Her marked rigidity gradually subsided and disappeared over a period of 2 months. Her muscle-stretch reflexes became normal and her plantar response became flexor. A follow-up assessment 4 months after admission showed her to be normal in alertness, muscle tone, and muscle-stretch reflexes.

Patient 2

A 19-month-old previously healthy male infant was admitted with an extensive second- to third-degree scald of approximately 60% TBSA. His trunk and limbs were affected, sparing his head. On arrival he was irritable and hyperpyrexia but fully alert and responsive. Intravenous treatment with lactated Ringer's solution and topical applications of silver nitrate were initiated. Thirty-six hours after admission, he suddenly became comatose and experienced severe generalized seizures involving mainly the right side of his body. Acute apnea necessitated nasotracheal intubation and positive-pressure artificial ventilation. Intravenous treatment with high doses of phenytoin and phenobarbital initially failed to control his intractable seizures. Laboratory data from the beginning of this acute event revealed severe metabolic acidosis and severe hypoglycemia with glucose levels of 13 mg/dl, both of which were promptly corrected. Computerized tomography of the cerebrum demonstrated an extremely low density in the distribution of the left middle cerebral artery, a finding suggestive of massive cerebral infarction. Single-photon emission computerized tomography using technetium 99m-labeled hexamethyl propyleneamine oxine demonstrated decreased regional cerebral blood flow in the distribution of the left middle cerebral artery and over the right hemisphere.⁶



Figure 1. Extrapyramidal parkinsonian syndrome in a 2-month-old female infant with 25% TBSA thermal burn. Note the rigidly flexed position of limbs and the expressionless facies while the patient is awake.

His course of treatment was complicated by *Staphylococcus aureus* septicemia, for which he was vigorously treated with intravenous antibiotics and from which he eventually recovered. Mechanical ventilation was discontinued after 18 days. Marked right-sided hemiparesis remained, as did recurrent focal motor seizures over the right side of his body accompanied by dysphasia. Initially, eye contact was unattainable, and he uttered high-pitched, irritable cries. Over a 12-month follow-up period, he became more alert, more attentive, and more amenable to commands, although he still exhibited significant cognitive delay. He also exhibited very mild improvement in receptive and expressive speech skills. His seizures proved partially responsive to a combination of carbamazepine, phenobarbital, and clonazepam.

DISCUSSION

The most common neurologic complication seen in children with burns is a generalized disturbance of cerebral activity, termed *burn encephalopathy*. This

disturbance may range from irritability, personality changes, and hallucinations to seizures, coma, and even death. This complication occurred in 5% to 15% of cases of children with burns.²⁻⁵ A change in the severity and prevalence of burn encephalopathy has been reported over the last 20 years. Twenty years ago, more children suffered from intractable seizures and coma secondary to cerebral edema leading to herniation and death than do now.^{5,7-9} Mohnot and coworkers⁴ recently reported a 5% incidence of burn encephalopathy, with a very low mortality rate. This lower mortality was attributed to improved medical and surgical management, including appropriate fluid replacement and possibly the avoidance of neurotoxic compounds such as hexachlorophene for the topical disinfection of burned areas.¹⁰

The first infant in this report experienced an extrapyramidal parkinsonian neurologic disorder, from which she recovered completely. Five days after a thermal burn of approximately 25% TBSA, she became mute and withdrawn, had expressionless facies, and displayed increasing rigidity. Tactile and auditory stimuli induced recurrent episodes, mainly over her right upper limb, of coarse jitteriness with increasing tone. Treatment with amantadine hydrochloride seemed effective after the patient's disorder failed to respond to clonazepam or phenobarbital therapy. To the best of our knowledge, an extrapyramidal disorder as a complication of severe burns in an infant has not been reported previously. The underlying pathogenesis of such a disorder is still obscure and open to speculation. This infant had hyponatremia of 124 mEq/L on admission, which was rapidly corrected over a period of less than 4 hours. Central pontine myelinosis has been associated with rapid correction of hyponatremia.¹¹ This condition was found by McKee and coworkers¹² in autopsies of 10 out of 139 patients with severe burns; they attributed it to a hyperosmolar state secondary to rapid correction of hyponatremia. A striatal extrapontine syndrome was recently reported after rapid correction of hyponatremia.¹³ This patient's symptoms included mutism, bradykinesia, and rigidity, and he also showed improvement with antiparkinsonian drugs similar to that of the infant described here. One may speculate that in the infant described here a similar striatal extrapontine parkinsonian disorder may have resulted from rapid correction of hyponatremia; the disorder's rapid onset was similarly followed by marked improvement and full recovery.

The second infant described here suffered a massive cerebral infarction as a complication of an extensive burn. Burn encephalopathy is sometimes associated with transient focal deficits.^{2,3} Antoon and coworkers² reported four cases of children who had seizures and transient focal weakness late in the course of their disorder, three of whom fully recovered and one of whom died of *Pseudomonas* sepsis. The patient reported here exhibited intractable seizures and apparently massive hemispherical infarction, which appeared 36 hours after admission. He remained with complete left hemiplegia, intractable partial complex seizures, and marked developmental delay. Similarly, Emery and Campbell-Reid⁵ reported the case of a 5-month-old infant who suffered a massive cerebral infarction and died. A few factors may have played a role in the evolution of massive cerebral infarction. A hypercoagulable state during the period immediately after the burn and marked hypoglycemia, as detected here, may be associated with generalized encephalopathy and uncommonly even with focal motor deficits.¹⁴ This infant, in fact, has widespread damage to his brain, as manifested by his marked cognitive and motor delay. This is delineated by a ^{99m}Tc-labeled hexamethyl propylenamine oxine, single-photon emission computerized tomographic study of regional cerebral blood flow, which showed several areas of decreased regional cerebral blood flow including that region responsible for his severe motor deficit.

Our experience suggests that young infants with burns run a higher risk of neurologic complications than do older children. Vigorous treatment for these infants is necessary and should include slow correction of hyponatremia, close monitoring of metabolic derangements, and treatment of hypoglycemia or hypocalcemia, in addition to the usual prompt management of infection.

REFERENCES

1. Cogswell JJ, Chu AC. Severe burns in children, 1964-1974. *Arch Dis Child* 1976;51:67-72.
2. Antoon AY, Volpe JJ, Crawford JD. Burn encephalopathy in children. *Pediatrics* 1972;50:609-16.
3. McManus WF, Hunt JC, Pruitt A. Postburn convulsive disorders in children. *J Trauma* 1974;14:396-401.
4. Mohnot D, Snead OC, Benton JW. Burn encephalopathy in children. *Ann Neurol* 1982;12:42-7.
5. Emery JL, Campbell-Reid DA. Cerebral oedema and spastic hemiplegia following minor burns in young children. *Br J Surg* 1962;50:53-6.
6. Shahar E, Gilday DL, Hwang PA, Cohen EK, Lambert R. Pediatric cerebrovascular disease: alterations of a regional ce-

- rebral blood flow detected by Tc 99m HMPAO SPECT. Arch Neurol 1990;47:578-84.
7. Erb IH, Morgan EM, Farmer AW. The pathology of burns: pathologic picture as revealed at autopsy in series of 61 fatal cases treated at Hospital for Sick Children, Toronto, Canada. Ann Surg 1943;117:234-44.
 8. Morrison B. A study of burns and scalds in children. Arch Dis Child 1947;22:129-51.
 9. Walker J, Shenkin H. Studies on the toxemia syndrome after burns. II. Central nervous system changes as a cause of death. Ann Surg 1945;121:301-13.
 10. Chilcote R, Curley A, Loughlin HH, Jupin JA. Hexachlorophene storage in a burn patient associated with encephalopathy. Pediatrics 1977;59:457-9.
 11. Sterns RH, Riggs JE, Shochet SS. Osmotic demyelination syndrome following correction of hyponatremia. N Engl J Med 1986;314:1535-42.
 12. McKee AC, Winkelman MD, Banker B. Central pontine myelinosis in severely burned patients: relationship to serum hyperosmolality. Neurology 1988;38:1211-7.
 13. Dickoff DJ, Raps M, Yahr MD. Striatal syndrome following hyponatremia and its rapid correction: a manifestation of extrapontine myelinosis confirmed by magnetic resonance imaging. Arch Neurol 1988;45:112-4.
 14. Wallis WE, Donaldson I, Scott RS, Wilson J. Hypoglycemia masquerading as cerebrovascular disease (hypoglycemic hemiplegia). Ann Neurol 1985;18:510-2.