Prolonged Respiratory Paralysis in Wound Botulism

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Two patients had wound botulism with longer duration of respiratory paralysis than previously described. Each sustained extensive trauma to soft tissues and grossly contaminated wounds when thrown from a vehicle in a rural area. Progressive muscular weakness and respiratory distress occurred 8 and 13 days after injury, in the presence of infected wounds and clinical and laboratory findings characteristic of botulism. Spontaneous vital capacity and inspiratory effort served as bedside indicators of ventilatory function throughout 11 weeks of ventilatory support in each case and paralleled other clinical assessments of progress and recovery. Wound botulism may result in neuromuscular paralysis for a prolonged period. These cases illustrate the primary role of scrupulous nursing and respiratory care throughout such a period of ventilatory insufficiency.

Wound botulism is an infrequently reported cause of neuromuscular blockade. We describe two cases with longer durations of respiratory paralysis than previously reported. These cases emphasize the central role of respiratory support and other intensive care measures in management.

CASE REPORTS

Case 1

A 28-year-old man was thrown from his truck in May 1976 and suffered multiple trauma. Injury to soft tissues included a grossly contaminated wound in the left thigh. Initial therapy involved placement of bilateral chest tubes and endotracheal intubation, both no longer necessary after two days.

Thirteen days after the accident, the patient was transferred to Harborview Medical Center, Seattle, because of deteriorating respiratory function (Fig 1). En route, emergency intubation was required. Upon arrival at Harborview Medical Center, examination revealed respiratory distress, despite normal findings on thoracic examination and clear chest roentgenograms. Respiratory values were as follows: tidal volume (TV), 250 ml; vital capacity (VC), 450 ml; and inspiratory effort, 20 cm H2O. The patient received treatment with a volume-cycled ventilator.

On the 18th day, progressive weakness of bulbar muscles and the musculature of the arms was noted. The results of a test with edrophonium chloride (Tensilon) were equivocal, and two days of oral therapy with neostigmine resulted in no clinical improvement. Administration of gentamicin for treatment of an infection of the wound was discontinued, without change in muscular strength.

Complications during hospitalization included the following: right middle lobe pneumonia due to Pseudomonas, which responded to treatment; sinusitis related to a nasogastric tube; and significant emotional problems, which responded to therapy with doxepin and to more intensive personal attention. The TV and VC remained around 150 ml and 250 ml, respectively, until the middle of July (Fig 1). At this point, intermittent mandatory ventilation (IMV) was utilized, with all mechanical ventilation being discontinued after 77 days (90 days after injury). An electromyogram done before discharge demonstrated post-tetanic facilitation.

Case 2

A 30-year-old man was thrown from his truck in July 1976, sustaining injury to the soft tissue of his right calf and an open, severely contaminated injury of the right ankle. The admitting hospital administered tetanus toxoid and human tetanus immune globulin (Hyper-Tet) and debrided the injuries of the soft tissue. Blood cultures grew Clostridium perfringens and an organism tentatively identified as C bifermentans. Cultures of material from the wounds grew clostridial and coliform species. Therapy with penicillin and gentamicin was begun, but high fever and extensive muscular necrosis persisted, and an amputation below the knee was performed.

Eight days after injury, lethargy and confusion heralded respiratory acidosis, with an arterial pH of 7.22 and arterial carbon dioxide tension of 73 mm Hg. Mechanical ventilation was instituted. Neurologic examination demonstrated extraocular muscular palsy. The results of a test with edrophonium chloride (Tensilon) were normal, and the findings from lumbar puncture and myelograms of the posterior fossa were normal. Chloramphenicol was substituted for the aminoglycoside, without clinical change.

On the 14th day, the VC was 100 to 300 ml, and a tracheostomy was performed. Progressive weakness occurred, and a clinical diagnosis of wound botulism was made. The patient was given 40,000 units of bivalent antitoxin.

On the 31st day the patient was transferred to Harborview Medical Center, with the following respiratory values: TV, 150 ml; VC, 350 ml; and inspiratory effort, 8 cm H2O. The muscular weakness was documented. The findings from a sensory examination were normal. Hyperalimentation

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was begun, and sleeplessness and depression were treated with doxepin and resolved. The course of the patient's respiratory condition was complicated by bibasilar pneumonia, with Enterobacter sp and \(\beta\)-hemolytic streptococi on culture of sputum; the pneumonia abated with therapy with gentamicin and penicillin. Respiratory measurements were followed (Fig 2).

At eight weeks after injury, therapy with IMV was instituted. Mechanical ventilation was finally discontinued after 78 days (86 days after injury). By the time of discharge (171 days after the accident), bulbar muscular strength had returned to normal, although mild symmetrical weakness persisted in the extremities. Pulmonary function tests revealed a mild restrictive defect.

**Discussion**

Respiratory insufficiency requiring mechanical ventilatory assistance has been reported with both food-borne and wound botulism. Our cases demonstrate that this need may be protracted and emphasize the importance of careful supportive care.

The clinical findings in food-borne and wound botulism result from neuromuscular blockade. The acute onset of dysfunction of cranial nerves in a patient with dirty wounds suggests the diagnosis of

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**Figure 1.** Vital capacity (solid line) and maximal inspiratory effort (broken line) during course of respiratory insufficiency (case 1).

**Figure 2.** Vital capacity (solid line) and maximal inspiratory effort (broken line) (case 2).
wound botulism. Symmetrical descending motor paralysis and ventilatory insufficiency in association with normal mental status and normal results of sensory examination are typical features. Wound botulism may be associated with fever because of local infection. Nausea, vomiting, and ileus are not seen when a wound is the source of the toxin.4

The toxin, a polypeptide, induces a defect in coupling between presynaptic depolarization and release of acetylcholine. With a single supramaxinal stimulus to a peripheral nerve, the electromyogram shows decreased muscular action potential. Following tetanic stimulation, abnormal facilitation occurs. These defects return to normal with resolution of the paralysis. The velocities of conduction through the nerves are normal, as there are no toxin-induced sensory examination are typical features. Wound botulism may be associated with fever because of local infection. Nausea, vomiting, and ileus are not seen when a wound is the source of the toxin.4

The diagnosis of wound botulism rests on the typical clinical picture of botulism in the setting of a dirty wound. Laboratory confirmation comes from cultures of material from the wound, a positive mouse toxin neutralization test, normal cerebrovascular fluid, and compatible electromyographic findings. In the absence of such complete data, the diagnosis must be made presumptively.4

Treatment includes short-term management of the respiratory paralysis, the use of antibiotics and antitoxin, and local care of wounds. As in food-borne botulism, administration of antitoxin is recommended as soon as the diagnosis is suspected from clinical findings. Bivalent (AB) antitoxin should be sufficient, as type A has been the most frequently reported type of toxin in wound botulism.6 The role of antibiotics is less well defined, but it seems sensible that treatment with appropriate antibiotics (as determined by testing for sensitivity) might be an important adjunct to local débridement. Serum for determination of the toxin should be obtained before administering antitoxin, and anaerobic cultures of material from the wound should be obtained before administering antibiotics.

The final outcome depends largely on respiratory support and nursing care for muscular paralysis. Most patients with botulism do not have underlying pulmonary disease, so that short-term management consists of supporting a decreasing VC. The role of therapy with guanidine in the management of botulism is not clearly defined, but this agent should be considered.

Recovery from neuromuscular blockade is monitored best in terms of the return of respiratory function. The VC and inspiratory effort should be followed daily. It had been suggested that therapy with IMV may be helpful in preserving the coordination and strength of the wall of the chest;7 this method was used in our patients, and we believe that it was helpful in the psychologic aspects of weaning.

References