Wound botulism in the UK and Ireland

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There are three main, naturally occurring, epidemiological types of botulism: food-borne, intestinal colonization (infant botulism) and wound botulism. The neurological signs and symptoms are the same for all three epidemiological types and may include respiratory paralysis. Wound botulism is caused by growth of cells and release of toxin in vivo, is associated with traumatic wounds and abscesses and has been reported in drug users, such as those injecting heroin or sniffing cocaine.

Up to the end of 1999 there were no confirmed cases of wound botulism in the UK. Between the beginning of 2000 and the end of December 2002, there were 33 clinically diagnosed cases of wound botulism in the UK and Ireland. All cases had injected heroin into muscle or by ‘skin popping’. The clinical diagnosis was confirmed by laboratory tests in 20 of these cases. Eighteen cases were caused by type A toxin and two by type B toxin.

INTRODUCTION

Clostridium botulinum is an anaerobic organism and forms spores that are naturally present in soil, dust and aquatic sediments. Each strain of C. botulinum produces one (or rarely two) of seven different neurotoxins (botulinum toxin types A–G) (Lund & Peck, 2000). There are three naturally occurring epidemiological types of botulism: food-borne, which is caused by the ingestion of preformed toxin in food; intestinal colonization, which usually occurs in infants and results from ingestion of spores, followed by colonization of the gut and toxin production in vivo; and wound botulism, in which toxin is produced in vivo after localized tissue infection (Brett, 1999). The neurological signs and symptoms are the same for all three epidemiological types of botulism. The neurotoxin spreads systemically and binds to receptors in presynaptic membranes in the neuromuscular junction. This binding blocks the release of acetylcholine and results in the typical descending symmetrical flaccid paralysis of botulism. The neurotoxin binds very tightly to the presynaptic nerves ends of cholinergic nerves, so recovery is slow, occurring when new terminals have sprouted from the original nerve end-plate (Witcome et al., 1998).

Wound botulism was first described in the USA in 1951, after reporting began in 1950 (Davis et al., 1951), and occurs in traumatic wounds, in injecting drug users (IDUs), following sniffing cocaine and, rarely, in abscesses or following post-operative injury (Werner et al., 2000). Wound botulism in IDUs was first described in New York in 1982 (Weber et al., 1993). Cases in the USA comprise over 90% of known cases of wound botulism worldwide and those in California account for over 75% of these cases (Werner et al., 2000).

In the USA, there has been a change in the epidemiology of wound botulism: the number of cases has increased dramatically in the last 8 years and this has been caused entirely by a growth in the number of cases of IDUs (Werner et al., 2000).

There had been no reported cases of wound botulism in the UK and Ireland before 2000. This report describes clinically diagnosed and confirmed cases of wound botulism in the UK and Ireland since March 2000.

METHODS

Microbiology. The Food Safety Microbiology Laboratory (FSML) receives requests from clinicians for reference laboratory tests for the diagnosis and confirmation of suspect cases of botulism in the UK. Samples are also tested from Ireland. In suspect cases of wound botulism, serum samples are examined for the presence of botulinum toxins and tissue from wounds is cultured for the presence of C. botulinum organisms. Pus is cultured for organisms and, if the sample size is sufficient, examined for the presence of toxins. Nasal swabs are examined if the case reported the sniffing or snorting of drugs. Suspect material, drugs and injecting paraphernalia are also examined for organisms. Toxins were detected in clinical specimens using the standard bioassay and identified by neutralization with antibodies supplied by the Centers for Disease Prevention and Control, Atlanta, USA (Solomon & Lily, 1998). Organisms were isolated from tissue and pus samples by enrichment using a cooked meat medium with added starch and glucose, and the cell-free culture supernates were tested for the presence of neurotoxin. If neurotoxin was detected in the enrichment culture, C. botulinum organisms were purified and the toxin type confirmed (Solomon & Lily, 1998).
RESULTS AND DISCUSSION

Epidemiology

There were no confirmed cases of wound botulism in the UK and Ireland up to the end of December 1999 and fewer than one suspected case was referred for examination each year between 1990 and 1999. Since March 2000, there have been 33 cases of suspected or confirmed wound botulism. In 2000, six cases were examined and three were confirmed, in 2001, three of four cases were confirmed and, in 2002, 13 of 23 cases were confirmed (Table 1). All suspected and confirmed cases had signs and symptoms consistent with botulism and no other clinical diagnosis was made. All cases regularly injected heroin into muscle.

The age range of suspected and confirmed cases was 22–51 years with a mean of 29 years. There were no differences in age or gender between confirmed and unconfirmed cases. All cases made a recovery, although some patients discharged themselves before recovery was complete.

There were some temporal clusters of cases, for example cases 5 and 6, cases 12–14, cases 19 and 20, cases 21–23 and cases 25–28, but these clusters lived in different parts of the country. Cases 15, 17 and 18 were temporally and geographically related: two cases lived in Dublin and one was thought to have obtained their heroin in Dublin. However, it is possible that heroin was obtained from a part of the country distant from where a case lived, so detailed analysis of geographical clusters on the information available here is not possible.

Cases of wound botulism in injecting heroin users have been reported during the last 2 years in other countries in Europe. A cluster of cases in Switzerland was reported in December 1999 and January 2000 from which C. botulinum type A and type B organisms were isolated (Burnens, 2000) and a case in December 2000 occurred in Norway, in which the toxin type could not be determined (Jenseniuss et al., 2000). In the USA, there has been a marked increase in the number of cases of wound botulism since 1995 (Werner et al., 2000).

The reason for an increase in the number of cases in the UK and the USA is unclear. Heroin used in the USA is usually black tar heroin, which originates from countries south of the USA, whereas, in the UK, heroin is in powder form, usually originating from Asia. C. botulinum spores may be introduced into the heroin at any point from production to use, by the addition of cutting or bulking agents or by environmental contamination. Heroin is poorly soluble in water and is usually dissolved in a weak solution of citric acid, with gentle heat, before injection. Repeated injection of dilute acid into muscle will cause damage and scarring and reduce the blood flow. These changes will decrease the aerobicity of muscle and so make conditions more favourable for the growth of anaerobes. In addition, dissolving heroin in acid with heating will stimulate germination of C. botulinum spores and is likely to kill non-spore-forming bacteria that could compete with C. botulinum.

Laboratory tests to confirm a clinical diagnosis of botulism

Routine laboratory tests are not helpful in confirming a diagnosis of botulism and samples should be sent to a specialized reference laboratory such as the FSML. In cases of wound botulism, serum samples are examined for the presence of toxin and samples of tissue or pus for the presence of the organisms and for the presence of toxin if the sample is of sufficient size.

Results of laboratory tests for C. botulinum

Samples sufficient for examination were received from 32 patients (32 serum and 16 tissue samples). Botulinum neurotoxin was detected in serum samples from 16 of the patients. In one case, changes indicative of botulinum toxin were detected in the serum sample, but the sample was of insufficient size for confirmation and identification of the toxin. C. botulinum organisms were isolated from tissue biopsy samples from 12 of 16 patients. In 11 cases in which botulinum neurotoxin was detected in a serum sample, tissue or pus samples were also examined. C. botulinum organisms were isolated from samples from seven of these 11 cases and all isolates produced the same toxin type as that detected in serum. Botulinum toxin was not detected in serum samples from 16 cases. Tissue samples were examined in six of these cases: C. botulinum was isolated from samples from three cases (two type A and one type B). Of the 20 cases where the clinical diagnosis was confirmed by laboratory results, 18 were caused by toxin type A and two by toxin type B. Multiple serum or tissue samples were examined from two patients. In case 14, type A toxin was detected in the first but not the second serum sample. Unfortunately, the date of collection of these samples was not available. In case 11, C. botulinum type B was isolated from one pus sample from the left arm and from one out of three samples of pus or tissue from the right arm. One case (case 6) had received antibiotic treatment for several days before the tissue sample was collected, and organisms were not isolated. Used syringes were examined from this patient and C. botulinum type A and B organisms were isolated from the syringe contents.

Laboratory tests confirmed the clinical diagnosis in around 60 % of cases. In all the cases reported here, a firm clinical diagnosis of botulism was made, and there was no other clinical diagnosis. There are several possible reasons for the failure to confirm the clinical diagnosis by laboratory evidence in all cases. Samples of wound or pus were not received from all patients, or were taken after antibiotic therapy had begun, so that organisms may not have been viable. Samples of tissue or pus were not of sufficient size for testing for the presence of toxin in addition to examination for the presence of organisms. Toxin was detected in situ in wounds in 32 % of 56 cases in the USA (Werner et al., 2000). Some serum samples were less than is needed for testing (5 ml), so the sensitivity of the test was reduced. The time of collection of serum samples may be important, because, once toxin reaches nerve endings, it binds irreversibly. The onset
of symptoms in some patients reported here appeared to be slow (7–14 days) while in others the progression was more rapid (1–3 days). This suggests that in some patients there was a slow release of toxin over a period of a week or two, with a gradual, cumulative effect. In this situation, it is possible that the amount of toxin present in serum at any one time was below the level of detection. The onset of symptoms in cases of wound botulism caused by injury was 4–18 days (Merson & Dowell, 1973) and 2–13 days (Werner et al., 2000).

Table 1. Suspected and confirmed cases of wound botulism in the UK and Ireland

<table>
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<th>Case</th>
<th>Sex, age</th>
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<th>C. botulinum organisms isolated from tissue or pus</th>
<th>Antitoxin administered</th>
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*Received antibiotic treatment before tissue sample collected.
†Changes indicative of botulinum toxin but insufficient sample for neutralization.
ND, Not detected; NE, not examined.
**C. botulinum** toxin types causing human disease

Human botulism is most frequently caused by toxin types A, B and E and more rarely by types F and C. Organisms producing types A, B and C are found in soil and dust while organisms producing type E are more frequently found in aquatic sediments. Only toxin types A and B have been shown to cause wound botulism to date. The proportion of cases caused by type A and B reported here is very similar to that in cases in the USA (Werner et al., 2000).

**The number of C. botulinum spores that causes human illness**

This is not known. It has been shown that subcutaneous injection of approx. 25 and 10 spores of type A produced fatal illness in guinea pigs and mice, respectively (Dezfualian & Bartlett, 1985). In at least three cases reported here, there were no obvious abscesses. The volume of **C. botulinum** culture needed to produce botulism may be very small (< 100 μl), so the site of infection can be very difficult to detect and not appear grossly infected, or, if a wound does appear infected, this may be due to the presence of bacteria other than **C. botulinum**.

**Antibodies to C. botulinum neurotoxins**

The amount of botulinum toxin that causes paralysis is small, and is less than the amount needed to induce an immune response. Patients who have contracted wound or food-borne botulism more than once have no detectable antibodies to botulinum neurotoxins in their serum (Weber et al., 1993; Beller & Middaugh, 1990; Schroeder & Tollosford, 1962). In contrast, in an adult with underlying Crohn’s disease and intestinal colonization with **C. botulinum** type A which lasted for at least 19 weeks, antibodies to toxin type A were detected in serum from week 19 to week 87, when testing ceased (Griffin et al., 1997).

**Disease in IDUs caused by other spore-forming organisms**

Spore-forming organisms other than **C. botulinum** can also produce illness in IDUs. The first cases of wound botulism in the UK coincided with an outbreak of severe illness in drug users in the spring and summer of 2000, which was caused by **Clostridium novyi** (McGuigan et al., 2002; Jones et al., 2002; Brazier et al., 2002; Noone et al., 2002). Five cases of **Clostridium histolyticum** infection with cellulitis or abscesses have been diagnosed in 2003 in the UK (HPA, 2003b). Cases of illness in IDUs caused by **Clostridium sordellii** have occurred in the USA (Kimura et al., 2001). Cases of clinical tetanus have been reported in drug users in the USA (Cherubin, 1970; Tunkel & Pradhan, 2002). There were two reported cases of tetanus in the UK between 1984 and 2000 known to be in IDUs (HPA, 2003a); however, since July 2003, there has been a sudden increase in the number of cases of tetanus (HPA, 2003a). **Bacillus** species also produce spores and there has been a recent case of injecional anthrax in a heroin ‘skin popper’ in Norway (Ringertz et al., 2000). A single case of illness in an IDU in the UK, caused by **Bacillus cereus**, was noteworthy because isolates of **B. cereus** from an aspirate from the case and from a sample of the patient’s own heroin were indistinguishable by amplified fragment length polymorphism (Dancer et al., 2002). Users of drugs other than heroin are also at risk of illness caused by clostridia; injection or snorting cocaine has resulted in cases of botulism (Kudrow et al., 1988).

**Clinical diagnosis of botulism**

Botulism is often low on the list of differential diagnoses at presentation of bulbar palsies. Diseases that can be confused with botulism and have been considered amongst the differential diagnoses in UK cases of wound or food-borne botulism are shown in Table 2, together with some of the distinguishing features compared with botulism.

Early symptoms of food-borne or intestinal colonization botulism may be gastrointestinal (nausea, vomiting and diarrhoea followed by constipation), but these do not occur in wound botulism. In contrast, neurological symptoms are the same irrespective of the route of entry of botulinum neurotoxin. The classical picture is of descending symmetrical flaccid paralysis, with no fever. Early symptoms include cranial nerve palsies – blurred vision, double vision (diplopia), difficulty in focusing, drooping eyelids (ptosis), facial weakness, sluggishly reacting or enlarged pupils, difficulty in swallowing (dysphagia), difficulty in speaking (dysphonia) and slurred speech (dysarthria). Weakness in the neck and arms, loss of the gag reflex, weakness in lower limbs and respiratory paralysis may follow. There may be autonomic signs with dry mouth, fixed or dilated pupils and gastrointestinal, urinary and cardiovascular dysfunction. Altered sensory awareness and fever are not associated with botulism; however, a concurrent infection may be present, particularly in cases of wound botulism. Deep tendon reflexes may decrease over time in some cases.

**Clinical presentation of cases**

The order of presentation of symptoms in some cases reported here did not follow the classical picture. A marked respiratory compromise and respiratory arrest occurred in at least three patients before the typical oculobulbar weakness and descending flaccid paralysis developed fully. Ptsosis and facial weakness were absent in at least one case on presentation, despite weakness in the neck and shoulders. Fever, thought to be due to respiratory infection, was present in at least two cases on presentation. Abscesses were not detected in at least three patients. Descriptions of the clinical presentations of some of the confirmed cases have been published: case 3 (Anonymous, 2000; Athwal et al., 2001; Mulleage et al., 2001), case 4 (Mulleage et al., 2001), case 5 (Hood et al., 2000) and case 22 (Merrison et al., 2002). A brief summary of these and other, non-confirmed, cases follows.

Case 1. A 22-year-old male presented with a 3 day history of bulbar palsies, head weakness and lateral gaze palsy, followed by an inability to move his shoulders and some indication of
descending paralysis. He was later ventilated and antitoxin was administered. Botulinum toxin was not detected in a serum sample collected on admission, 2 days after onset of head weakness. The patient made a full recovery.

Case 2. A 43-year-old woman presented with an abscess, which resolved following treatment. Approximately 3 weeks later, she had a short history of breathing problems, followed by facial weakness and then proximal arm weakness. There was no change in eye movements and the patient was not ventilated. Botulinum toxin was not detected in a serum sample collected at the onset of facial weakness.

Case 3. A 34-year-old woman was admitted for treatment of deep abscesses in the buttocks that healed well. Seven months later, she was admitted, complaining of shortness of breath. She had a 2 day history of variable ptosis and diplopia, was pyrexial and had signs of left lower lobe pneumonia, external ophthalmoplegia, sluggish pupillary responses and severe proximal limb and respiratory muscle weakness. Tendon reflexes were present throughout. Sensory perception was normal. Antitoxin and intravenous benzylpenicillin were administered. She was ventilated for 5 weeks and remained in intensive care for a further 2 weeks. Bulbar dysfunction resolved after a further 5 weeks. Botulinum toxin type A was detected in a serum sample collected on admission, 2 days after onset of pyrexia (38°C) and a painful groin swelling.

Case 4. A 27-year-old male had a history of abscesses. He was admitted with pyrexia (38°C) and a painful groin swelling. He received antibiotic treatment for 1 week. Ultrasonography of the groin revealed no evidence of abscess. Three days later, he presented with progressive dysphasia, dysphagia and general flaccid limb weakness, particularly in the proximal muscles. Twelve hours later, he suffered a respiratory arrest and required ventilation. After resuscitation, he developed bilateral ptosis, dilated pupils which reacted sluggishly to light, diplopia and depressed reflexes. Electromyography results were suggestive of botulism. Antitoxin and intravenous antibiotics were administered. He was ventilated for 8 weeks. Botulinum toxin type A was detected in a serum sample.

Case 5. This patient, a 27-year-old man, had two groin abscesses for 7 days. He was admitted to hospital with a 2 day history of diplopia. The abscesses were drained. Pain and fever were absent. Over the next 2 days, the diplopia worsened and he developed increasing difficulty in swallowing, slurred speech and ptosis. Descending symmetrical weakness and respiratory paralysis developed over the next 12 h. He was treated with antitoxin and benzylpenicillin and was ventilated for 16 days. Botulinum toxin type A was isolated from a tissue biopsy.

Case 6. A 33-year-old man was admitted to hospital with multiple leg abscesses but self-discharged before adequate treatment was completed. Three weeks later, he developed a swollen leg. A few days later, he reported double vision and difficulty in focusing and, the following day, difficulty in swallowing and loss of head control. He was admitted to hospital and the next day he had a respiratory arrest and was
ventilated. Botulinum toxin was not detected in serum collected after he had been ventilated for 1–2 days. Organisms were not isolated from tissue collected after several days of antibiotic treatment. The contents of eight containers of used syringes were examined and Clostridium botulinum type A and type B spores were isolated.

Treatment of wound botulism

Early treatment with antitoxin is essential and antitoxin should be administered after a definite clinical diagnosis of botulism has been made. Antibiotic therapy and eradication of abscesses are also important in patient management and in avoiding a relapse after the antitoxin has cleared the body. It is important that debridement is performed after antitoxin treatment, so that any toxin released during the procedure is neutralized. Prompt treatment, with antitoxin given on day 1 of symptoms in cases of food-borne botulism, resulted in patients being ventilated for fewer days and in a shorter hospital stay (Werner et al., 2000). Wounds associated with botulism may not always be easy to locate and may not appear infected.

Reducing the number of cases of wound botulism

Clearly the single most effective way to reduce infection in IDUs is to reduce their injection of drugs into muscle or by ‘skin popping’. Botulism is unlikely to be caused by sniffing or snorting drugs and is highly unlikely following injection into a vein. Failing that, a reduction in the use of shared needles and the reuse of injection materials, together with improvements in the use of sterile technique when injecting, should reduce infections. However, Passaro et al. (1998) reported no reduction in risk of botulism due to cleaning the skin or paraphernalia, which suggests that the main source of Clostridium botulinum is the heroin or substances added to it, and not the skin or the environment of the user.

Conclusions

Wound botulism is now the most common cause of botulism in the UK and the number of cases is increasing dramatically. Confirmation of the clinical diagnosis requires laboratory tests that are specialized; in the UK, the Health Protection Agency FSML provides the reference service for these tests. Wound botulism must be considered in drug users who present with the typical symmetrical descending flaccid paralysis of botulism, i.e. cranial nerve palsies, skeletal muscle weakness and respiratory insufficiency, where there is a history of injecting drugs, particularly into muscle or by ‘skin popping’ and when epidemiological investigation suggests that food-borne botulism is unlikely. The diagnosis must also be considered in cases that present with only some of the signs typical of botulism, such as slurred speech, difficulty in swallowing and loss of head control. The order of presentation of symptoms may not follow the classical picture, with weakness in the neck or a marked respiratory compromise and respiratory arrest before the typical ocular-bulbar weakness and descending symmetrical flaccid paralysis are fully developed. Sinuses or abscesses may be deep-seated and difficult to find, and wounds may not appear to be clinically infected. The number of cases in the UK appears to be following the trend in the USA of increasing dramatically. This increase has implications not just for the health of drug users, but also for costs incurred in caring for these patients and use of intensive care facilities for long periods of time.

REFERENCES


