Introduction:

Welcome to CUGH's bi-weekly clinical case-series, "Reasoning without Resources," by Prof. Gerald Paccione of the Albert Einstein College of Medicine. These teaching cases are based on Prof. Paccione's decades of teaching experience on the medical wards of Kisoro District Hospital in Uganda. They are designed for those practicing in low resource settings, Medicine and Family Medicine residents, and senior medical students interested in clinical global health. Each case is presented in two parts. First comes a case vignette (presenting symptoms, history, basic lab and physical exam findings) along with 6-10 discussion questions that direct clinical reasoning and/or highlight diagnostic issues. Two weeks later CUGH will post detailed instructors notes for the case along with a new case vignette. For a more detailed overview to this case-series and the teaching philosophy behind it, see Introduction to "Reasoning without Resources". Comments or question may be sent to Prof. Paccione at: gpaccion@montefiore.org

Note: If you would like to be notified when a new case is posted (along with instructor notes for the previous one), send your e-mail to Jillian Morgan at jmorgan@CUGH.org.

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CASE 38 – Stiff Stride

A 60 year old farmer was in his usual fully-functional state of health until about a week ago when he began to experience chest pain and "failure to breath" when climbing hills 1-2 times/day which forced him to stop for a few minutes before continuing. Two days later his two shoulders felt "burned" whenever he'd move, without notable abnormal movements or tightening he was aware of, and two days after that his jaw felt "tight" and he couldn't open his mouth all the way. A day later, the day prior to admission, he began to feel pain in his thighs and hips, and was unable to walk normally. He's had trouble sleeping for the past week.

He had no significant past medical history: no adult hospitalizations, other episodes of chest discomfort, history of hypertension, smoking or diabetes. He's generally stayed away from health facilities, and didn't remember taking medications or receiving injections in over 20-30 years. He hasn't used rodenticides nor ingested any unusual substances. He's had no fever, weight loss or cough, although this past week his throat felt sore without congestion or rhinorrhea. He's had no ear pain or discharge, headache, mouth or tooth pain when chewing. He has noted episodes of double vision for the past 2 days that reverted after minutes, and problems initiating a swallow. He recalls no significant trauma or wounds in the past year.

Physical Exam: Anxious appearing older male, sitting straight in bed, flanked by two male friends who walked him to the bed, one bracing each arm.

BP 126/90 (range first day, 115-128); HR 105 (range first day,90- 112); RR 16; T: 37.2 (range first day, 37.2-38.0)

Skin: no evidence of trauma, infection, burns or abrasions;
Head: held slightly extended; face: taut with wide smile, retracted eyes, wrinkled forehead, sweating profusely; can open mouth 2 cm, tongue protrudes with slight deviation to left, masseters tense, no pain while tapping teeth with throat-stick but reflex biting of stick with slow release; (-) Chvostek;
Mouth moist, no thrush;
Eyes: PERRLA, with dilated pupils; EOM full, without obvious strabismus or diplopia elicited; conjunctiva normal, no pallor nor icterus;
Fundi: benign, without arteriolar narrowing, A/V nicking, hemorrhages or papilledema;
ENT: ears normal, no evidence of otitis externa or media;
Neck: mild-moderate rigidity in all directions; no JVP/HJR; no nodes; thyroid normal;
Lungs: clear to auscultation and percussion
Heart: PMI normal, non-displaced; normal S1, S2; no murmurs or gallops;
Abdomen: bowel sounds slightly decreased; tense musculature, no hepato-splenomegaly to palpation or percussion; no tenderness or guarding; Rectal: no masses, stool brown, guaiac negative;
Extremities: no edema, pulses +2 diffusely; no cyanosis or clubbing; joints/hips normal, without heat, effusion or pain on movement;
Neurologic: Mental Status: normal orientation x 3; memory intact; anxious but lucid
Cranial Nerves: normal vision and eye movements as above: V sensation normal, and VII facial movement normal, with increased resting tone; hearing normal; testing "Gag reflex" instead elicits a strong bite on the throat stick; XI intact; XII: as above, tongue deviates to left;
Motor: diffuse muscle tone/rigidity all muscle groups, legs>arms; strength 5/5 diffusely; occasional twitching of muscle fibers evident over chest wall and thighs bilaterally;
Sensation: intact to vibration, position, pin prick; Reflexes: +2 diffusely except knees and ankles: +3-4 with myoclonus;
Cerebellar: difficult exam due to increased tone, grossly intact; no tremor outstretched hands;
Gait: stiff, extended posture, halting, robotic, slow.

1. What is the frame of this case (i.e. the key clinical features from the history and physical that the final diagnosis must be consistent with)?

- *60 year old man, previously healthy but no contact with health system*
- *1 week of progressive symptoms*
- *Chest tightness with "failure to breathe" first symptom, then shoulders "burning"*
- *Trismus, followed by difficulty walking*
- *Diffuse muscle rigidity/tone*
- *No headache, fever, change in mental status*
- *No recent wound*

2. a. What is the significance of the Physical Exam findings?
b. What exam observation noted here has been published as a highly accurate diagnostic maneuver for the disease this patient has?

a. The physical exam is diagnostic in this case: the unusually high specificity of the findings overrides diagnostically the otherwise (somewhat) atypical evolution of symptoms from the clinical history.

- *Trismus: inability to fully open the mouth, an early finding of tetanus, is caused by rigidity of the masseter muscles. Measurement of the distance between the front teeth with mouth maximally open can be used to follow disease course over time.*
- *Generalized muscle rigidity: defines a systemic process whose mediators disseminate via the circulation. Leg and neck extensors are notably involved more than arms.*
  The taut facial musculature produced in this patient another classic finding of tetanus, "risus sardonicus", "a sneering grin… thought of old to resemble the effect of a Sardinian ranunculus, which on being chewed contorted the face of the eater" (as quoted by Bleck, see below): wide mouth, raised eyelids with wrinkling of the forehead.
- *Sweating, tachycardia without fever (N.B. mild tachycardia 105), dilated pupils: all are manifestations of a hyper-sympathetic state, signs that the process affects the autonomic nervous system.*
- *Notable negatives on exam, relevant in the differential diagnosis include: no signs of local oral pathology, normal mental status without headache or meningismus (i.e. rigidity on neck flexion only); negative Chvostek; no fever;*
- *No cuts, burns, wounds: Usually a site of entry of the Clostridium bacillus is evident in cases of Tetanus, although not in this patient. Absence of any sign of external or possible internal source of infection is seen in 20% of adult patients with tetanus.*
- *No evidence of chronic otitis: chronic otitis has been reported as the underlying source of many cases of tetanus – which can be localized and chronic;*
- *Normal rectal, guaiac negative: GI sources of infection, such as malignancies, have been reported to cause tetanus.*

b. The "Spatula Test" has been described as a simple bedside test to diagnose tetanus (see below). "It consists of touching the oropharynx with a spatula (or stick) inserted through the teeth; normally this elicits a gag reflex and the patient tries to expel the spatula (negative test result). In contrast, patients with tetanus develop a reflex spasm of the masseters and bite the spatula (positive test result)."

Tested on 380 patients with tetanus, and 20 without tetanus but with tetanus-mimics, the test had a 94% sensitivity and 100% specificity. (N.B. Since tetanus is a clinical diagnosis without an independent gold standard, the study from 1995 requires replication/validation in a different cohort of patients, with more control subjects.)
The attempt to evaluate the gag reflex in this patient elicited a positive "spatula test" result.

3. a. What's the diagnosis in this case? How is the diagnosis made?

*The diagnosis in this patient is **Tetanus**, a neurologic disease caused by a toxin elaborated by the anaerobic gram (+) bacillus Clostridium tetani which lives in soil and on surfaces throughout the world.*

The diagnosis of tetanus is clinical. Since the organism is ubiquitous and the disease is caused by a toxin elaborated in minute quantities and sequestered in nerves, cultures are non-specific and meaningless, and toxin assays are unavailable. (N.B. 250 grams of toxin are sufficient to kill the world's entire human population.)

The clinical presentation is relatively specific – diffuse muscle rigidity and spasms - presenting either simultaneously with or following "trismus", the masseter rigidity present in >85% on admission. Dysautonomia is frequently seen later in the first week.

Mortality, from antiquity up to the early 20th Century, was >80%, but since the turn of the Century and with prompt access to care, the availability of equine and now human anti-toxin serum has reduced mortality to ~10-20%. Death in the 3rd World is usually by asphyxia, inability to breath due to prolonged spasm of the pharynx/upper airway, or chest wall/diaphragm. In the developed world, with the resources to paralyze anesthetically and ventilate mechanically when necessary, death is more commonly due to hemodynamic instability caused by fluctuating dysautonomia: abrupt swings between severe hypertension and tachycardia and refractory shock and bradycardia.

Most importantly, tetanus is preventable through active immunization with tetanus toxoid: 3 vaccinations provides almost complete immunity for 5 years; the full recommended series of vaccines totals 5, with routine boosters every 10 years.

b. What is the epidemiology and pathophysiology of the disease?

In World War I, crude estimates placed the incidence of tetanus in 1 of every 500 casualties. Nowadays, about 1 million cases of tetanus are thought to occur annually in the world. The disparity in health resources and disease prevention between rich and poor countries has relegated modern tetanus to an affliction of the poor, with 80% of cases occurring in Africa and Asia. In the U.S., approximately 100-150 cases are estimated to occur annually, largely in people over 60 years old with waning immunity; in Africa, approximately 120,000 cases of neonatal tetanus occur in infants of mothers never immunized, accompanied by about an equal number of cases in children and most-often younger adults.

Spores of the ubiquitous bacterium C. tetani are introduced through often-minor breaks in the skin - punctures, lacerations, even insect bites; both indoor and outdoor activities are implicated, and farming and the normal play of barefoot children are occupational risks. Chronic wounds, septic abortions, chronic otitis and parenteral drug use are often found when external trauma isn't evident, although up to 20% of cases provide no identifiable source of infection.

The dormant spores of C. tetani germinate when introduced into a wound and proliferate if the redox potential of the tissue is low. Strains of C. tetani that contain a toxigenic plasmid produce the exotoxin "tetanospasmin" that causes all the manifestations of the disease. Tetanospasmin enters peripheral neurons through their pre-synaptic membrane, travels retrograde from the terminal to the cell body and can cross several synaptically connected neurons. It thus moves from the periphery to the spinal cord and ultimately to the brain. Although it's initially spread hematogenously, it must still enter the nervous system at (multiple) neuron terminals and ultimately reach the CNS via retrograde transport.
Tetanospasmin blocks neurotransmitter release from the pre-synaptic terminal of inhibitory interneurons in the spine and CNS. Since all motor action is a balance of orchestrated stimulation and inhibition of opposing muscle groups, without finely coordinated inhibition alpha motor neurons increase their firing rate, opposing muscles are not inhibited, and painful unregulated spasms occur. Spasms occur spontaneously and after diverse motor and sensory stimuli such as movement, touch, sound or light. Relaxation is impaired and diffuse rigidity ensues.
The amount of toxin produced in clinical tetanus is not enough to stimulate an immune response, and thus immunity doesn't develop after one episode. All survivors must be vaccinated.

c. What is the spectrum of clinical presentation?
What's atypical about this patient's presentation?

The clinical presentations of tetanus have been traditionally classified into 4 subtypes: generalized, localized, cephalic, and neonatal, classifications which simply reflect variations in the site of toxin action.

- *In most series, generalized tetanus is the presentation in over 80% of patients. Symptoms usually evolve over a few hours, initially with stiffness of the jaw (trismus) or neck, although back and shoulder stiffness may have been present for hours prior. In the generalized form, the beginning of symptoms marks the widespread dissemination of tetanus toxin throughout the nervous system, and symptoms predictably progress caudad over hours to a few days - from trismus and risus sardonicus - to rigidity/spasms of the chest wall and abdomen - and then to the legs, usually more than the arms.*
  Spasms of seconds to minutes in duration are a dominant problem in the first week of illness, lasting for up to 3 weeks; rigidity can persist for 4-8 weeks. Generalized spasms resemble decorticate posturing with opisthotonus of the trunk, extension of the legs, and flexion of the arms on the chest with clenched fists. In areas without well-equipped modern ICU's, death from ventilatory failure occurs during prolonged spasms of the glottis or chest wall/diaphragm, or via paralysis of the same structures through the toxin's effect on the neuro-muscular junction.
  Symptom severity often increases for up to 1-2 weeks after diagnosis, reflecting the time of transport of intra-neuronal toxin into the CNS. Recovery then begins which usually takes about 4 but up to 8 weeks. The recovery time reflects the time for the synthesis and transport of the new presynaptic components that are necessary for neurotransmitter release by affected inhibitory interneurons.

- *Localized tetanus presents with fixed rigidity of the muscles at or near the site of injury. It may be mild, persist for months, and resolve spontaneously, though if untreated many cases will progress to the generalized form. Partial immunity may be responsible for*

limiting hematogenous dissemination of toxin. DTRs may be brisk, but the rigid muscles painful and weak.

- *Cephalic tetanus is a relatively rare form of localized tetanus that occurs after head injuries or chronic otitis media with C. tetani (milder form). It presents with paresis of cranial nerves – thus facial paralysis most commonly, but dysphagia and ophthalmoplegia can be seen.*

- *Neonatal tetanus accounts for half of the cases of tetanus in Africa, and is one of the principle causes of neontatal mortality. It presents usually during the second week of life with weakness and inability to suck progressing to tetanic spasms, opisthotonis, a hypersympathetic state and death in 95%. It occurs in the offspring of non-immunized mothers and results from lack of aseptic technique in dividing the umbilical cord (a longer stump appears safer) and subsequent care in a clean environment.*

This patient's presentation was atypical in that *the first symptom was chest tightness and "failure to breathe" climbing hills (gesturing to his chest with a broad hand) which dissipated after a few minutes of rest. Although symptoms expressed across cultures and through translators often require liberal interpretation, his initial symptoms sound like new-onset typical angina – a rare disorder in rural Africa in a man without risk factors (other than a male of 60 years). When asked directly, he denied muscle tightening or obvious spasms of the neck or chest wall to help explain these angina-like symptoms.*
However, the specificity of the symptoms and findings that later evolved make the diagnosis of tetanus certain, and the timing of these initial angina-like symptoms - just 2 days prior to the "shoulder burning" and 4 days before the trismus, make it likely that they were part of the same tetanus syndrome and not 2 disorders. Could they have been spasms of his glottis, or esophagus?... or even a spasm of his diaphragm? Nevertheless, any of these would be atypical of tetanus as the initial manifestation of the disorder and not subsequent to trismus and/or neck spasms.

d. What is the relevant differential diagnosis of this disease? (see Bleck, summarized here)
The differential diagnosis of tetanus includes strychnine poisoning, meningitis, seizures, dystonia, tetany, stiff man syndrome, and "pseudo-tetanus".
The various disorders cause some symptom overlap with tetanus, but only strychnine poisoning, by competitively antagonizing glycine receptor-mediated inhibition in the spinal cord, mimics the disease.

- *Strychnine poisoning:*
  (Adapted from Smith, B. Strychnine Poisoning J Emerg Med, Vol. 8, pp. 321-325, 1990).
  Strychnine is currently used in rodenticides. In lower doses it has been used in human concoctions as analgesics, digestive aids, cold remedies, cathartics, tonics, vitamins, stimulants, and sedatives. It was banned for human consumption in the US in the 1980s. Strychnine competes with the inhibitory neurotransmitter glycine for receptors in the spinal cord, brain stem, and higher centers resulting in disinhibition - as in tetanus - although unlike tetanus it doesn't prevent release of glycine. Strychnine's stimulatory effects predominate in the spinal cord.
  Onset of symptoms usually begins within 15 to 30 minutes following ingestion. Initial symptoms include apprehension, a heightened sense of awareness, muscle spasms, hyper-reflexia and hypersensitivity to stimuli. In major intoxications this is followed by

overwhelming convulsions, typically lasting 30 seconds to 2 minutes, often precipitated by minimal external stimuli.
As in tetanus, opisthotonic posturing with the back arched, extremities hyperextended, and the jaw tightly clamped is characteristically seen. Facial muscle spasm may produce the sardonic smile (risus sardonicus). Abnormal eye movements persisting for 12 hours have been reported. Muscle relaxation usually occurs between convulsions. The patient generally maintains a clear sensorium during and after the convulsions. Most patients do not tolerate more than 5 convulsive episodes, and death commonly occurs within several hours after ingestion. Respiratory arrest secondary to spasm of the respiratory muscles is the usual fatal event. Prognosis for survival is good if the patient survives beyond 5 hours.

Strychnine poisoning is more acute than tetanus, with ingestion occurring within hours before symptom onset and the course of disease rapid. It doesn't cause trismus until late while trismus is an early manifestation of tetanus, and strychnine induces little abdominal rigidity between spasms.

- *Meningitis causes fever and nuchal rigidity, as can tetanus, but rigidity is seen only on neck flexion; is accompanied by headache and fever early, and often by a change in mental status and/or seizures, all unlike tetanus. Meningitis doesn't induce muscle spasms, the hallmark of tetanus.*
- *Seizures/epilepsy often elicits brief tonic spasms followed by rhythmic jerking, and loss consciousness. Tetanus is unaccompanied by rhythmic convulsions, and there is no change in consciousness.*
- *Dystonia induced by drugs that block dopamine in the CNS is rare in Africa, and should be suggested by history. Dystonia produces torticollis and oculogyric crises, not seen in tetanus, and doesn't induce tetanic reflex spasms. If in doubt, a brief trial of an anticholinergic agent like diphenhydramine relieves dystonia.*
- *Tetany induced by hypokalemia/alkalosis involves the extremities more than the axial muscles, producing signs like Chvostek's (facial twitch on tapping the facial nerve over the parotid) or Trousseau's (palmar spasm induced by palmar tap). Trismus is rare.*
- *Stiffman Syndrome is a rare autoimmune disease caused by auto-antibodies to GABAergic neurons, and has been likened to a chronic form of tetanus. However, its onset is insidious; cranial nerves minimally involved; trismus not seen; and rigidity is relieved during sleep.*
- *"Pseudo-tetanus" is a rare psychogenic disorder in which the patient's movements resemble tetanus. Usually the patient's posture is inconsistent and complex, and rigidity is lacking or obviously feigned. Secondary gain is usually apparent.*

4. a) Why are prognostic estimates for this disease particularly relevant in rural Africa?
b) What is the prognosis for this patient?

a) Estimating prognosis of a patient with Tetanus on admission in rural Africa is very relevant if referral and transport to a well-equipped ICU is possible. If impossible, prognosis/severity estimates may still be important in determining the allocation and timing of scarce resources and nursing attention.

There are a number of prognostic indexes for Tetanus dating back over 80 years. Not surprisingly for this clinically diagnosed and monitored disease, the (univariate) clinical

variables identified as predictive of survival are similar between indexes and haven't substantively changed in a century. Recent refinements are based on statistical definitions of "independence", with some features traditionally recognized as prognostically important not quite making the multivariable cut of independence once all features are considered.

Two indexes will be outlined here: one, the Dakar Index, simplified and summarized by Bleck, and the Tetanus Severity Index.

Dakar Index (as per Bleck)

Score 1 point for each on admission: a) Incubation period <7days; b) period of onset <48 hours; c) high-risk portal of entry (i.e.burns, umbilical stumps, surgery, compound fractures, septic abortions, IM injections); d) generalized spasms/rigidity; e) core temp >40C (104F); f) tachycardia (HR>120 adults, >150 neonates);

Severity and Prognosis:

Score	*Severity*	*Mortality (with treatment)*
0-1	*mild*	*<10%*
2-3	*moderate*	*10-20%*
4	*severe*	*20-40%*
5-6	*very severe*	*>50%*

Exceptions: Cephalic tetanus is always scored as severe or very severe
Neonatal tetanus is always scored as very severe

Tetanus Severity Score (TSS):

The TSS was developed in Vietnam on retrospective patient data from 1993-2002, and validated in the same institution on prospective data in 2003; published in 2006). Multi-variable statistics were used to identify the clinical features that independently predicted survival, and a score derived from adding scores from the various categories.

A score of >8 was used to predict mortality – with sensitivities of 77% and 65%, and specificities of 82% and 91% in the derivation and validation cohorts respectively.

Age: <70, 0; 71-80, 5; >80, 10;
Time from first symptom to admission (days): </= 2, 0; 3-5, -5; >/=5, -6;
Difficulty breathing on admission: No, 0; Yes 4.
Co-existing medical conditions (as per ASA physical status scale): fit and well, 0; minor illness or injury, 3; moderately severe illness, 5; Severe illness not immediately life threatening, 7; Immediately life threatening, 9;
Entry site: internal, open fractures, or injection: 7; other, including unknown, 0;
Highest SBP recorded during first day in hospital: <130, 0; 131-140, 2; >140, 4;
Highest HR recorded during first day in hospital: <100, 0; 101-110, 1; 111-120, 2; >120, 4;
Lowest HR recorded during first day in hospital: <110, 0; >110, 2;
Highest temp recorded during first day in hospital: <38.5, 0; 38.6-39, 4; 39.1-40, 6; >40, 8;

In the study introducing the TSS (Thwaites, et al.), it was compared to the Dakar index. The Dakar, using a cutoff of 3, had sensitivities of 13 and 25% and specificities of 98 and 96% in the derivation and validation cohorts respectively; and, in the validation cohort, an "Area Under the ROC Curve" (the "AUROC") - a measure of discriminative power and overall accuracy - for predicting mortality of .80 compared to the TSSM .89. The latter difference wasn't statistically significant.

[N.B. The differences between the derivation and validation cohorts are probably due to differences in how the data for such scores are obtained: the derivation scores are really the best mathematical fit for the retrospective data being assessed. The validation cohort provides a better test, but ideally when the data are collected in a fully independent cohort - from another time and place - with the study fully blinded to investigators, data collectors and subjects . Thus, the lack of statistical significance between the TSS and the Dakar indexes in predicting mortality in this less-than-optimal study biased towards the TSS is notable.]

Our patient's severity/prognosis would be scored in the following ways:

Dakar: incubation period, unknown; onset > 48 hours; non-high risk portal of entry; +generalized spasm/rigidity; core temp <40; no tachycardia: TOTAL SCORE: +1: MILD

TSS: points for: time from first symptom to admission (in our patient, 4-7 days) -5 or -6. Highest HR first day, +1; TOTAL: -4.

i.e. Under both scoring systems, our patient would have a good prognosis (with appropriate basic therapy).

5. What is the treatment for this disease?

Mortality that was once >80% for untreated tetanus, can be reduced to 20-40% by administration of anti-toxin and basic hospital management described below, and to 10% or less in modern ICUs.
ICU care is usually impossible in the areas of the 3rd World where tetanus is prevalent. An article by Oladiran, et. al (see below) from Nigeria sums up the situation in most district hospitals in rural Africa: " ... we share a common set of problems: a lack of supplies and functioning equipment, an unreliable supply of electrical power, and a lack of clean water. Intensive care takes on a different meaning in such a context, where it may mean nothing more than a conscientious nurse who stays awake all night taking care of a tetanus patient by kerosene lantern light in an over-crowded ward with 30 other patients."

Treatment relies on
a) reduction of active toxin that's not already intra-neuronal: human tetanus immunoglobulin (HTIG), if available, is preferred to equine antiserum. Both bind circulating unbound toxin, which is key in preventing progression. However progression for a few days should still be expected as the already-intra-neuronal toxin makes its way retrograde to the CNS.
HTIG, 500 IU should be given once IM; it has a half-life of 23 days;
Equine antiserum, often the only anti-toxin available in rural Africa, has a 20% incidence of anaphylaxis, thus a test dose should be first administered. Full treatment with antiserum involves 10,000 to 1 million units IM. The recent literature has shown that smaller doses may be equally effective, thus 10 - 20,000 units can be given.
Intrathecal administration has been shown more efficacious than IM, and should be considered if feasible.

b) eliminating clostridium bacteria with antibiotics: metronidazole is preferred to penicillin which in high doses may itself inhibit GABA transmission. Metronidazole 500mg IV q. 6 hours for 7-10 days.

c) control of spasms both medically and environmentally
In district hospitals, control of spasms pharmacologically usually involves IV Diazepam, frequently at (high) doses of 300mg IV per day. Combining IV with oral as soon as possible limits the amount of propylene glycol administered. Diazepam should be continued as needed up to weeks, and tapered over 2-3 weeks to prevent withdrawal. Lorazepam and baclophen are alternatives.
Reducing sensory stimuli which trigger spasm can be achieved by placing the patient in a dark, quiet environment. Often in district hospitals, this is not possible. Placing the patient in a side room may lessen stimuli, but also minimizes monitoring and nursing care.

d) management of "autonomic storms": hypertensive/tachycardic emergencies are best treated with labetalol (0.25-1mg/min) or morphine sulphate (0.5-1.0 mg/kg/hour). Clonidine and calcium channel agents are also used. Propranolol alone may be dangerous as it leaves alpha receptors unopposed and in hyper-sympathetic states a hypertensive crisis can be precipitated. Hypotension requires fluids and norepinephrine; bradycardia, atropine and/or isoproterenol;

e) vaccination with tetanus toxoid should be given for long-term immunity, in a site distant from HTIG or antiserum. The dose of toxin that causes disease is insufficient for immunity against future bouts of tetanus.

f) if necessary, transfer to a well-equipped health. If spasms aren't controlled at a dose of muscle relaxant that decreases respiratory drive, neuromuscular blockade with vecuronium and mechanical ventilation is indicated.

Our patient was placed in the first bed on the open ward for the first few days to ensure adequate nursing, and then on day 4 moved to a side room to minimize spasms, with family instructed in his care. He declined the 5 hour trip to the referral hospital in Mbarara. Since we were unsure about how much better the available resources would actually be in Mbarara, and his history was one of insidious evolution and the severity indexes suggested mild disease, he was managed in Kisoro. He received the last dose of HTIG the hospital had, and treatment with diazepam and metronidazole.
His spasms initially got worse, but were controlled finally with diazepam. By day 5 of hospitalization his family wanted to take him home, remarking on his improvement. They couldn't understand the need for continued in-hospital monitoring for at least 2 weeks. When it seemed as if they might elope in the middle of the night, we reduced his diazepam dose, the spasms returned, and they agreed to stay the course.
His BP varied, but never became difficult to control.
He left the hospital after 18 days, walking with residual but less rigidity (as expected for 1-4 more weeks) but no spasms, on tapering diazepam.
His entire family received tetanus immunization, and were scheduled to return to complete the primary immunization schedule of 3 doses.

Suggested Readings:

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