

Sri Lanka Poison Control: Toxicology Case Studies in Narrative

By Rais Vohra, MD

“The only way of discovering the limits of the possible is to venture a little way past them into the impossible.”
—Sir Arthur C Clarke



Sri Lanka. A tear-shaped island in the Indian Ocean, this is an ancient land with a long, incredible, and sometimes unfortunate history. Several decades of civil war have left the social infrastructure feeble and the general populace vulnerable to both physical and mental illness. Rebuilding efforts of the last few years were set back even further when the devastating 2004 tsunami struck and clashes between the military and rebels—the Liberation Tigers of Tamil Eelam (LTTE) or Tamil Tigers—intensified.

During completion of a fellowship in medical toxicology in San Diego, I traveled to Sri Lanka for a

rotation in that country’s only poison center. The burden of disease from poisoning in Sri Lanka, and throughout South Asia, is staggering—the number of severe poisonings and fatalities many times greater than in industrialized countries. I met many doctors who are involved almost daily in managing difficult poisoning and snakebite cases without access to many basic medical resources. The following passages are from my journal and e-mail notes.

Arrival

(April, 2006)

I landed in Sri Lanka about an hour north of Colombo, the capitol city—one airport serves the whole country. I stepped out of the airport and, before I knew it, I was in a taxi, speeding toward the town. My cab driver, unregistered in an unmarked car, darted into and out of the taxi lane, furtively looking for police. All officially registered taxis here are white minivans, so he was certainly nervous about picking up passengers.



We made small talk while he drove. I recalled some last-minute advice: distribute money in many places—and slipped a few bills into each sock and pocket. The driver is a Buddhist and speaks English quite well. He told me that as a “tsunami patient,” he had spent three days in a hospital at the time of the disaster. A shoulder injury—possibly a dislocation, not a fracture—occurred when his car rolled into the water. I asked, “Why were you in the hospital so long?” He said, “There were too many people and not enough doctors.” I let him know how shocking and heartbreaking it was to see the news about the tsunami. He seemed like a decent man—at the end of the hour-long trip he wrote down his cell phone number in case I needed another ride. He even invited me to go see his family’s rural home on the south coast. In the early morning hours we finally found my guesthouse in the Cinnamon Gardens district of Colombo. After nearly two days in flight I was ready for a real bed.

Later that morning, I awoke with no idea of the time. The birds there are LOUD. It was cloudy and the air was thick with moisture and the smells of incense and petrol. From the balcony, I spotted four large crows with mischievous eyes. A squirrel-like animal was making a high-pitched chik-chik noise. Deep,

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lush green trees and vines were everywhere outside of my bedroom window.

A local newspaper stated that the LTTE had set off a claymore mine on the northeast coast of the island, injuring soldiers and a British tourist couple. Tensions escalated, though the government was “committed” to continuing peace talks.

History

Sri Lanka (previously Ceylon), blessed with incredibly diverse natural beauty and year-round moderate climate, has welcomed visitors for many centuries. The Buddha visited three times; Sinbad the Sailor of the Arabian Nights visited twice, as chronicled in his 6th and 7th voyages; and Sir Arthur C Clarke, noted author and inventor, moved to “the Emerald Island” in 1956 and still lives there.



Environment

Colombo was still hot in April and the summer monsoon had officially begun; consequently the climate was both hot and humid, but I was having a wonderful time. The capitol city is a coastal metropolis, which lives, works, and plays according to the rhythms of the sea, and breathes a daily sigh of relief at dusk. Life is hard for almost everyone, but they tried to be helpful despite language barriers. Although



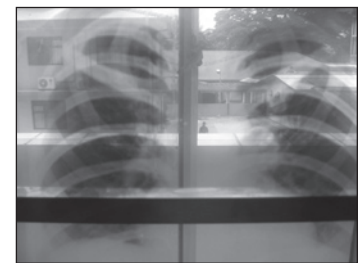
I had been warned about hustlers and thieves, everyone acted civil and followed the rules, except the rickshaw operators, who drove as if they were immortal. It was actually fun to haggle with them—I was forced to slowly learn the language that way—and I always offered to pay double if they would let me drive but no one accepted.

Hospital and Medical System

The overall medical system is as ambitious as it is under-resourced. The National Poisons Information Centre (NPIC) is based at the National Hospital of Sri Lanka (NHSL), the municipal hospital for the city and surrounding towns. It is the tertiary referral hospital for government-run hospitals on the island. Like the rest of the city, NHSL is overcrowded and disorganized, run almost entirely by interns and house officers. Somehow things get done with a logic and pace that I never totally decoded. The charts remain at the bedside; progress notes are handwritten on thin crepe paper; labs are recorded on slips the size of sticky notes tied onto the chart with yarn. All care and inpatient medications are free

in the municipal (government-run) hospitals; patients pay for outpatient prescriptions, labs, and x-rays.

Because I arrived just after a holiday for Buddhists (New Year) and Christians (Easter) the poison center was not fully staffed: one doctor and one research officer on duty with an incredibly polite assistant who made really good chai tea.



X-rays are read using windows as light-boxes.

Medical Practice

CT and MRI scanning is only done at private hospitals and is exceedingly uncommon. For ICU infection control measures, a sterile gown and “clean” flip-flops are required. The general medical and surgical floors are almost always full and crowded—in several wards I saw two patients sharing single beds. In a rural hospital, rubber gloves are

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washed and dried for reuse. Patients are given their records, compressed into a single laminated page, to take with them at the time of discharge.

At the NPIC, we received five calls over two consecutive days. Many poisoned patients reside on the wards, unknown to the NPIC. The ward interns call only when they have a question, not to report a case. There is no central registry in the emergency room or triage room to track all admitted patients. To track poison patients, I had to inquire from ward to ward. The doctors and staff were usually cooperative, though unsure as to my reason for inquiring. Most poisoning incidents admitted to the National Hospital are from ingestion; snake bites are more common in rural-area hospitals.

Case Studies

The following eight cases are studies of common poisonings at the National Hospital, though unique experiences for me.

Case 1: Dapsone

In my first week, the NPIC received a call about a woman age 19 years. Three months earlier she had been diagnosed with the dermal

variety of leprosy; she now had a few scars that looked like freckles. After an argument with her husband (women are shy about disclosing domestic problems) she ingested an overdose of her medications (exact dosage unknown). Relatives gave her a glass of coconut milk—a common home remedy—to induce emesis; it worked well. In the “Accident and Casualty Ward” (Emergency Department), she was given oral sodium bicarbonate to induce further emesis and finally activated charcoal, a common protocol.

The NPIC was called on the patient’s arrival to the floor, for advice about a medication called “MDT,” evidently an “antiepilepsy” drug. The poison center director explained that this was a congener for dapsone. We advised observing for delayed methemoglobinemia and hemolysis, but without a blood gas machine in that hospital, they couldn’t get cooximetry. Instead, they tested her for “methemoglobinuria” and for the telltale chocolate-colored blood when drawn. By the next morning the patient was asymptomatic, the CBC was within normal parameters, and the plan was for psychiatric review before discharge.



Rubber gloves being dried for reuse

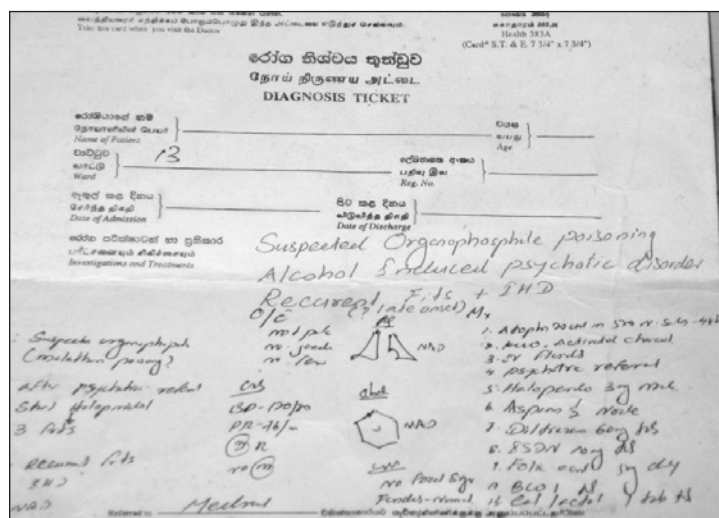
Case 2: Acetaminophen

After “fighting with her mother,” a woman age 19 years ingested 50 tablets of “Paracetol” three days before being admitted for dehydration. She presented with vomiting and drowsiness, and admitted to the overdose the next morning. Because drug levels are unavailable, acetaminophen overdose, which is becoming very common in Sri Lanka, is routinely managed symptomatically. Most patients can’t afford expensive liver function tests. Ironically, they are eligible to receive free IV n-acetylcysteine, although not all wards stock it. Treatment is started solely on the basis of history of ingesting greater than 150mg/kg. Physicians use a 24-hour continuous IV infusion protocol. This patient’s prothrombin time rose to 33 and transaminases to the 600s, and then normalized. Although abandoned decades ago in the west, many smaller hospitals still use methionine because NAC is considered too expensive. Methionine (2.5 grams q 4 hours x 4 doses) is an alternative sulfur-donating compound to help regenerate the glutathione molecules in hepatocytes.

Case 3: Paraquat

In an emotional fit, a bulldozer operator and part-time farmer, age 36 years, ingested 50 cc of the green-colored herbicide Gramoxone

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Typical diagnosis ticket

(paraquat)—or so he related to the admitting team along with relating a recent history of green vomiting. He was given 300 cc of 30% fullers' earth, gastric lavage, and then activated charcoal. (Both fullers' earth and bentonite are talc-like binding powders that can inactivate paraquat in the stomach). He was being observed for respiratory failure.

He looked fine; he was actually walking around. The house staff doubted the history of ingestion—he had no oropharyngeal lesions, a telltale sign of the corrosive paraquat. The only finding in his mouth was orange-stained teeth from his chronic betel nut habit. Paraquat can cause sudden collapse and respiratory failure during the first ten days, so without objective measures, the staff felt compelled to observe him. They didn't obtain a chest radiograph given the absence of symptoms, and had no reagents for a urine test for paraquat. Oxygen saturation monitoring is available only in the ER and ICU, so he was treated with several doses of intravenous fluids and kept on a vegetarian diet “for renal protection.” Eventually he was discharged, asymptomatic. Our conclusion was that he was scaring his family with the threat of paraquat ingestion, or else he took far less than he said he did.

A true paraquat ingestion is tragic—patients develop oral burns and within days progress to

Adult Respiratory Distress Syndrome (ARDS) and respiratory fibrosis. The manufacturer of paraquat has developed a testing kit to help doctors make a rapid diagnosis. Adding sodium bicarbonate and sodium dithionite powder to urine can establish the diagnosis. In the presence of paraquat, urine turns

purple. I demonstrated this kit to myself by adding paraquat to “clean” urine and watched the color change to a deep turquoise. With diquat, a renal toxin used in herbicides in the US, the urine turns yellow-green with this test.

Case 4: Phosphoric Acid

A woman age 31 years with a past history of ingesting organophosphates ingested an unknown amount of phosphoric acid after an abusive attack by her alcoholic husband. Upon arrival at the hospital, she was treated with gastric lavage. Her initial labs showed: arterial blood gas: 7.23/pCO₂ 22/pO₂ 44/ bicarb 9, sodium 157, K 4.2, BUN 23, creatinine 0.5. No calcium or phosphate levels were sent. Urinalysis showed only 1-2 pus cells. An x-ray showed bilateral interstitial markings, and she was diagnosed with ARDS. However, I was not convinced she didn't vomit and aspirate acid, reflecting aspiration pneumonia. When I saw her five days later she was recovering and tolerating soft solids. Her medications included cefotaxime, ranitidine, metoclopramide, and steroids. Although she complained of throat pain, I saw no lesions. The plan was to endoscope her as an outpatient in a few weeks. Phosphoric acid is an interesting ingestion because, in addition to the corrosive local injuries, it can also cause systemic hypocalcemia, hyperphosphatemia, and acidosis.

Case 5: Cardiac Glycosides

A young woman ate a part of a fruit from her garden and then (ruefully minimizing her intent) said she mistakenly ate two seeds of a fruit from one of her trees. Her family made her swallow coconut milk at home and she vomited. She also developed headache and fainting later that night. In the hospital she

was treated with omeprazole, domperidone (an antiemetic) and fluids. Her heart rate drifted down over the first 12 hours of admission from the 70s into the 50s. Her electrolytes were normal per the house staff and no further treatment was rendered. NPIC was called because her pulse was still in the 60 to 70 range on day three and the house officer wanted to know the half life of this toxin.

I examined her partially consumed fruit, called Dia-Keneru or sea mango (*Cerebra manghans*). It contains cardiac glycosides similar to digoxin. She may have eaten parts of the peel although she insisted she picked out a couple of seeds and ate them. By the next afternoon, her heart rate was in the 100s and she was discharged. Patients in Sri Lanka must be monitored much longer than in a Western setting, where digibind can be given quickly to mitigate symptoms of toxicity.

In Sri Lanka, ingestion of yellow oleander (*Thevetia peruviana*) seeds is a very common method of self-poisoning, with fatalities reported with only four seeds, called “lucky nuts.” Poisoning is due to digoxin-like compounds, and characterized by vomiting, then bradycardia and other cardiac rhythm disturbances.

Much of the basic pharmacokinetic data for herbal and plant toxins has not been established. Because of the different varieties of plants, as well as unavailability of digibind due to cost, many physicians have extensive experience with the course and severity of cardiac glycoside poisonings. This is ironic, as many of the initial studies showing safety and efficacy of digibind were done in Sri Lanka with patients poisoned by yellow oleander. Researchers from the United Kingdom and Australia have continued to seek other, less costly treatments of yellow oleander poisoning here.

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Case 6: Snakebite

A house officer told me that a man with a cobra bite died the day before I arrived. He was bitten on the hand, went into renal failure, and transferred from the ward to the ICU, but expired anyway. He was a “snake catcher.”

Snakebites are a major cause of morbidity and mortality in Sri Lanka, particularly in rural and agricultural regions. After a bite, patients bring in the killed snakes to help physicians choose the type of antivenom to administer. The most common type of snakebite is from the hump-nosed viper (*Hypnale hypnale*), for which there is no antivenom. Symptoms of bite initially include mild local edema, necrosis, and blister formation; eventually, a quarter of patients develops renal insufficiency. Sometimes a Russell’s viper or a cobra is brought in as evidence of a recent encounter.

Case 7: Arecholine

The areca nut (Areca catechu), usually called betel nut because it is wrapped in leaves of the Piper betel, is found on street-corner carts all over South Asia. It is mixed with spices, fennel, candy pieces, wrapped, and painted with a chalky paste of calcium carbonate; the slightly basic chalk accelerates the release of plant alkaloids. The most potent compound in betel nut is arecoline, which resembles acetylcholine and works on nicotinic and muscarinic receptors.

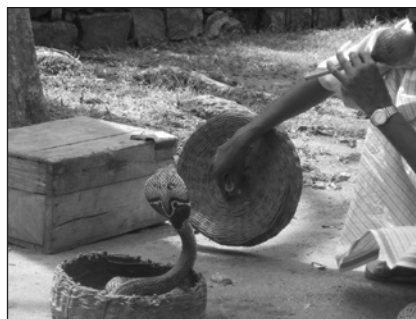
One night I ate several betel nuts and got mydriatic, flushed and slightly buzzed. Unfortunately, it was drizzling and after dark and I got a little lost, wandering into a Buddhist shrine/temple—there is one on almost every other street corner—which was empty except for a few stray cats. I met a man performing his nightly prayers who warned

about the drug addicts that haunt these temples at night. I fearfully walked back to my guesthouse.

Case 8: Cyanide

As they were trying to plant a claymore mine in the northeast part of the country, two Tamil Tigers, members of the LTTE, were caught by the military. Each of them immediately tried to swallow a capsule of cyanide. One of them was prevented from swallowing the capsule and he survived. The other became ill and was taken to a peripheral hospital where they specifically treated him for cyanide poisoning with hydroxycobalamin without much effect. Twenty-four hours later, he was transferred to the NHSL in Colombo. I was out of town, chasing snakebite cases in the small hospitals of the beautiful hilly countryside in the center of the island.

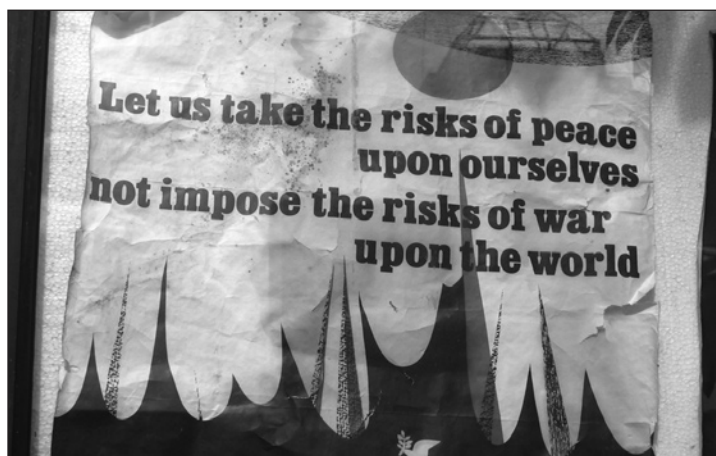
Meanwhile, a female suicide bomber, also a member of the LTTE, targeted a military hospital and base in the capitol. The woman detonated herself near an army jeep. She was standing at a speed bump to ensure that the vehicle would be moving slowly. Her target was an officer in the army, but she was successful in only sending him to the OR for a hemicolectomy, not to the morgue. He was a top-ranking army com-



Snake charmer

mander in Sri Lanka. The bomber had claimed to be pregnant, bypassing security by making an obstetric appointment at the army hospital.

I didn’t even know about these developments until the next morning when I returned to the hospital. With both the army commander and one of the Tigers there, the hospital was a very tense place, and the city even more so. The terrorist who ingested cyanide was admitted to the intensive care unit, then intubated after losing consciousness, although he never had acidosis on blood gas. They didn’t check a lactate level, which can be used as a surrogate marker for severe cyanide toxicity. He was given thiosulfate (one dose of 25 mL from the antidote kit) but he remained hypotensive. He was even given a dose of cobalt EDTA at the recommendation of the toxicology attending physician. In the





1970s, studies conducted on sheep suggested that, like hydroxycobalamin, this compound can bind and inactivate cyanide, but the side effects are poorly tolerated and thus should only be used as a last resort. The thiosulfate was never redosed. He arrested and died early the morning after I returned.

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After these attacks, much of the city hastily converted into a military-occupied police state. To be honest, at this point I was truly scared. The government had positioned armed police and soldiers everywhere but they weren't much solace standing there, because anyone with a grim determination could still plow through their barricades with a car bomb. It was astonishing that hundreds of people still milled about in front of the hospital every morning, which I suppose is a testament to the average Sri Lankan's humanity and pacifism. Everyone was apprehensive, especially because they knew how much worse it can get when half the city is road-blocked and there are curfews, and people are picked up for interrogation every day. The military was starting to launch air

strikes against some LTTE camps in the northeast part of the island, but as with all air strikes, there would be collateral damage, which could provide further motivations for the rebels to continue their aggression.

Before I left Sri Lanka two fretful weeks later, I noted many aphorisms posted in public places in reference to civil war, signs that, in retrospect, have an added poignancy as the prospects for a significant ceasefire have grown even more dim.

Reflection

In his essay *The Myth of Sisyphus*, the Algerian-French author Albert Camus said, "There is but one truly serious philosophical problem, and that is suicide."¹ In the course of my rotation, I often wondered why there was such a high rate of suicide in Sri Lanka. When I asked people I met, they cited many factors: poverty, civil war, and easy accessibility of toxic substances in a poorly regulated agricultural economy. There also seems to be a cultural tendency towards self-harm that defies ethnic or class barriers. Other countries have these issues, and yet in Sri Lanka overdose is much more common.

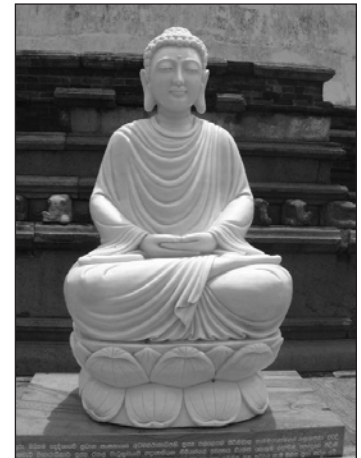
I was riding a bus to the hospital one day and saw graffiti on a fenced school courtyard along a sidewalk—"Life Sucks!" In the US, I wouldn't have given it a second thought, but here it unnerved me, particularly since it seemed to shout out the answer to the "philosophi-



Graffiti on school fence

cal problem" that Camus defined. The graffiti writer's despair, eagerly vented in an absurd and loud scrawl along a noisy street, is touching. It bespeaks the despair of a whole segment of society.

Perhaps I have been asking the wrong question. Perhaps instead of asking why there is such a relatively high rate of overdose in Sri Lanka, I should ask its converse: What is it that makes people under the same immensely stressful circumstances NOT resort to self-destructive behaviors? What can we as caregivers learn from their resilience, and how can that strength of resolve be shared with others in the community? What is the antidote to despair? ❖



Acknowledgments

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Reference

1. Camus A. *The myth of Sisyphus: and other essays*. Reissue edition. New York: Vintage; 1991. p 3.