Episode 43: Toxicology Mysteries - A Series of Poisoning Cases

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Transcript

I'm Rebecca. I'm Rob. This week we're going to be having a look at some very interesting and unique case studies that we have spotted over the last few weeks and thought, oh, that sounds interesting. Before we dive into the detail, I am going to let out a little secret, and this is probably not that surprising.

They all involve people eating things they shouldn't eat. And I wanted to share with you my toxicology origin story. I'm excited for this. And one of my earliest memories is actually of my brother eating a toilet duck, and I vaguely remember my mum finding him with a toilet duck in his mouth.

And the funny story is that my mum worked as a telephonist at the hospital. So she knew off the top of her head the poison's information number. So just dialed it and spoke to them, and they said, keep an eye on him. And he was fine.

Nothing bad happened. He was all all right. He turned out OK in the end. But yeah, I think that was probably my first indirect exposure to toxicology, essentially.

So why don't we jump into some of these cases? Before we jump into the cases today, if you want to stay up to date with our content, you can find us on Instagram at @the_tox_lab. We also have a LinkedIn page. We're quite active over there.

And for those of you who don't do social media, but still want to get in touch with episode suggestions or comments on our episodes, you can email us at thetoxlab@gmail.com. So this week, we're going to be looking at some rather interesting and unusual case studies that have caught our attention. Now we're going to try and keep you guessing as much as we can before we give the actual cause away. So Rebecca, dive into this first case.

So this first case involves a three-year-old female who presented to the emergency department with respiratory distress, cyanosis, vomiting, and abdominal pain. Her heart rate on admission was 143 beats per minute. Her respiration rate was 42 breaths per minute, and her O2 sats were 92%. A thoracic x-ray two hours after her admission to the emergency department showed signs of air trapping.

And as her respiratory symptoms progressed, she was admitted to the pediatric ICU. Here she required O2 and antibiotic therapy, and 24 hours after her admission, she was mechanically ventilated due to respiratory failure and decreased consciousness. She then developed a hemolytic anemia over the following 72 hours, and developed extensive subcutaneous emphysema in the chest and neck with a right-side grade 1 pneumothorax. Her symptoms did improve, and she was transferred to an inpatient unit and was discharged 14 days after her admission.

An x-ray and CT scan three months post-follow-up showed bronchiectasis in both lung bases. So a 14-day stay in hospital, quite unwell, and actually still poorly three months later. So what's going on with this patient? Give us a bit more information.

So prior to her admission to hospital, it transpired that a family member was using glitter to decorate a set of ornaments, and it was hypothesized that she'd been exposed through inhalation, dermal contact, oral ingestion, and ocular exposure. So this was a case study from Argentina, and the title of the paper was a case of glitter poisoning or words to that effect, so it immediately caught my eye.

I did have to do a bit of research, and there's actually a few words in Argentinian Spanish that vaguely translate to glitter in English, and it transpired in this case we're actually not talking about the plastic glitter that we stick on with PVA glue that we all know and love. This is actually essentially metal grindings or metal filings which are used for more adult craft projects. In this case, it was Christmas decorations, wasn't it? So actually it was inhalation of bronze grindings rather than traditional plastic glitter that we might think of.

So because of the glitter ingestion, they did take samples and measured both copper and zinc in serum, and they also measured copper in the urine as well, and they were all within normal limits. So what was actually going on in this case? We think probably this was likely inhalational exposure. Which it did also have abdominal pain and GI symptoms, so there might have been some oral ingestion as well.

There likely would have been, actually, wouldn't there, even if you inhale things up your nose, some of that does end up going down the throat, so it's possible quite a lot was ingested orally as well. So the problem in this case was probably the copper, wasn't it? Actual metallic copper in high doses can be quite irritating and toxic. You can get free radical production, which leads to irritation of membranes, and because it had been inhaled, the copper's right there in the lungs leading to these breathing problems, and actually, possibly long-term lung damage as well, is evidenced by the X-rays.

And it can cause systemic toxicity as well, can't it? That was why they measured the copper levels, although the copper levels weren't particularly exciting, so maybe there wasn't so much evidence of systemic copper toxicity there. More the lung disease. Zinc oxide inhalation can also cause a very characteristic acute respiratory illness, and zinc chloride as well, which is often found in smoke bombs, can cause quite a severe respiratory distress as well, so imagining both the copper and the zinc did play a role.

So this was quite an interesting case presentation, wasn't it? As I say, I think the headline caught my attention in a way that maybe wasn't intended, but there was actually a line in there, wasn't there, when they were discussing exposure to these metal powders in Argentina, that in parts of Argentina, glitter was banned in schools, and that made me slightly sad, because I imagined at first that this was shiny flakes of plastic stuck on with PVA glue, but actually, that was where I learned that it was actually metal shavings, which, to be fair, probably children shouldn't play with fine metal shavings.

I think that was a solid piece of advice. So I do think it was a really nice little interesting study, shows that not all glitter is created equal, and that sometimes nuance is lost in translation. So in this next case, a two-year-old child was brought to the emergency department with abdominal pain and vomiting. A blood test on admission showed a severe hypokalemia, so low potassium, of 2.1 millimoles per liter, but after potassium supplementation, they recovered uneventfully.

The cause of this presentation? Ingestion of snake fireworks. So this was another headline grabber, and I had to Google what is a snake firework, so should we explain what a snake firework is? I think we have to.

They are fireworks that produce a, essentially, sausage-shaped trail of ash when you set light to them, and I was curious as to why somebody might eat these. They're actually very small, black pellets, so they're quite easy for children to pick up, and you put a light to them, they start burning, and then they

produce this snake shape of ash. It rises up out the pellet. You can find some nice videos of them on YouTube.

So this paper is a retrospective study of Oregon Poison Centre human exposure cases between 2009 and 2023 with substance codes for fireworks, and these were witnessed or suspected oral exposures. Out of these calls, 99 snake firework-related calls were found, with 64 calls meeting the inclusion criteria. All of these exposures were children, 70% of them were male, and 6 out of the 64 were symptomatic, 5 experienced vomiting, 2 experienced abdominal pain, and 1 experienced one episode of diarrhoea.

25 of these individuals were referred to a healthcare facility, with 5 being admitted for monitoring or depletion of potassium as a result of a hypokalemia. As with all poison control data, they categorised it based on the effect, so the majority of which had no effect or a minor effect, 4 cases were classed as moderate, and 1 child was classed as significant. So it wasn't just one-off ingestion of snake fireworks.

There was actually a significant number in a period of time, and as I said, it's because they do look like effectively just small black pellets, which could be mistaken for sweets, and so I think it's likely these were just mistaken identity put in the mouth by a child. So obviously eating fireworks is not generally a good thing to do, but the thing that is in these is the barium salts, isn't it? And this gives a green colour to the flame, so not only do you get this nice ash snake effect, but it gives a green flame, so it's the barium that's included, and that's what's causing the toxic presentation in a lot of these cases.

The low potassium is quite interesting, isn't it? Low potassium is a symptom of barium poisoning. So we're going to delve a little bit into some clinical biochemistry and explain why these patients had such low potassiums. In health, all of your cells have got these little proteins called sodium potassium ATPase, and this works to pump potassium into cells and pump sodium out of cells.

So cells contain a very high concentration of potassium relative to their environment, but there are also some ion channels, and these are called inward rectifier potassium channels, and they allow a bit of potassium to leak out, and that is what maintains your normal, healthy potassium balance in your blood. Barium basically blocks this channel, so the ATPases continue to pump potassium into the cells, but it can't leach back out, and so you end up with a low blood potassium.

Low blood potassium is bad because cells like nerves and muscles rely on a relatively consistent difference between intracellular and extracellular potassium and other charged ions in order to function. So normally a severe hypokalemia can cause some cardiac effects, but in this case, there is none of the patients experience any. And actually, despite some very low potassiums, there wasn't too many very severe presentations, was there? Was there one that was classified as severe?

Yeah, there was one that was classified as severe, and that was the one that had potassium of 2.1. And that is very low, bearing in mind it's normally between 3.5 and 5.3, so this really was quite low. Another little interesting quirk actually in this data was that some of the patients developed rebound high potassiums, didn't they? So the medical management of these patients would have been to give them some potassium to correct their low potassium.

But of course, the amount of potassium in their body hadn't gone down. It was just being trapped in the cells. So as soon as the barium was cleared and these channels reopened, the potassium could maintain its balance again. And so these patients' potassium is overshot a bit because they were being treated with potassium at the same time.

Full barium toxicity and magnesium sulfate can also be given, and this reacts with soluble barium salts to form insoluble barium sulfate, which then can't act on these receptors as well. So all in all, these patients all recovered, no long-term effects, moral of the story, don't eat fireworks. The other thought

I did have about this actually, I don't think it's clear from the paper, is whether they were eating the uncombusted firework or whether they were eating the trail of ash. That is, yeah, that's the point.

I'm not sure. No, I don't know what's more appealing to children to eat. Yeah, I'm not sure. In this next case series, 11 patients attended emergency departments of two different hospitals in Greater Toronto with symptoms such as oral parasthesia and GI symptoms, including nausea, vomiting, and abdominal pain.

And this all occurred shortly after eating food at a local restaurant. The patients were aged between 20 and 90 years old and all presented over an eight-hour period, as symptoms developed between five minutes and 60 minutes after eating. After the initial GI symptoms that they presented with, most patients developed dysrhythmias, including ventricular bigemini, bidirectional ventricular tachycardia, and complete heart block. And the spectrum of illness varied widely from just having the oral parasthesia and requiring no intervention to these complex dysrhythmias requiring multiple medications.

So lots of people all getting unwell at the same time, all eating at the same restaurant. And it didn't sound like food poisoning. No. The poison centre was contacted and they were recommending supportive care.

Public health services attended and closed the restaurant and identified one chicken dish that was ordered by all the patients who presented. So this particular chicken dish contained a unique spice, known as sand ginger, and this had been restocked from their supplier earlier in the day. Both the spice and blood samples from patients were tested and aconite was detected. So sand ginger and aconite, two very different things, aren't they?

Sand ginger is a type of ginger found in Indonesia, China, Taiwan, and it's used in certain cooking recipes. Aconite, a very poisonous plant, also known as monk's hood, wolf's bane, devil's helmet is another. And I've also seen queen of poisons written down as well. And it contains a chemical called aconitine, which is an alkaloid, and it's incredibly poisonous, actually.

It's a very, very poisonous plant. The actual pure compound, aconitine, as little as two milligrams might be fatal, and that's about one gram of plant matter, so relatively small amounts. And aconite poisoning has got a long, rich history, actually. It's described in Greek mythology where the goddess Hecate is said to have invented the plant.

It's apparently mentioned in Shakespeare play, Henry IV. It's definitely featured in a lot of murder mystery type shows as well. I'm pretty sure it's been on Midsomer Murders and things like that. If it hasn't, it'd be a great plot line.

So it's incredibly poisonous plant. The plants themselves are really quite nice, nice purple flowers, and people do sometimes grow them in their garden because they are really pretty. It also has a use in traditional Chinese medicine for analgesia and also is reported to have anti-inflammatory properties. So how did this then end up in this chicken dish in Canada?

What has happened here? So it turns out that the distributor packages both sand ginger and aconite in China for obviously very different purposes, and there had been a mix up along the way, and so aconite had been packaged as sand ginger and shipped to this restaurant. So we think it was a mix up at the factory. Yeah.

Some of them only had mild tongue symptoms, tingling, parasezia of the tongue, which is a commonly described symptom of early aconite poisoning, I'm told. But some of them did get quite unwell, didn't they? Quite severe cardiac arrhythmias and even complete heart block, wasn't there at one point in

one patient. And I believe all the patients made a full recovery as well.

So we were talking earlier, weren't we, about potassium channels and how barium affected potassium channels. When it's aconite, it's actually all about sodium channels. And these channels in your nerves and your heart, they're basically gates. They open and allow sodium in to allow depolarization of the cell.

And then normally they snap shut again to allow the cell to repolarize. And aconitine binds to those channels and forces them to stay open. So in nerves, such as in your mouth, you get the tingling and the numbness, but in the heart, it's a lot more dangerous because you get chaotic firing or no firing at all. And so people's heart rhythm is disrupted and ultimately their heart can just stop.

Now this case was quite well-publicized, wasn't it? It certainly made the news and unsurprising because it was a mass poisoning event. I think they did pull other packets off the shelves following this. I'm not sure if there was any other mislabeled products identified, whether this was simply a one-off.

So if you want a bit more of a deep dive into this particular case and want to hear from the toxicologists who worked on this and the guys at the Poison Center, I'd thoroughly recommend going and checking out the Poison Lab podcast who recently covered this. Yeah, do go and check them out. They did a really thorough job of covering this case and really go into the detail quite nicely. So this final case involves a 60-year-old male with no prior psychiatric or medical history who presented to the emergency department concerned that his neighbour was poisoning him.

He stated that he wasn't taking any meds or supplements and his vital signs and physical exam were all normal. Lab tests showed he had a raised chloride-negative anion gap, a low phosphate, and his bicarbonate was elevated. So a negative anion gap is quite weird, isn't it? An anion gap is a calculated parameter.

You take your sodium and your potassium, you subtract your chloride and your bicarb, and what you're left with is what we call the anion gap, the difference in positive ions and negative ions. And normally, there is a non-negative number which accounts for ions like phosphate and things like that. In this case, the negatively charged parameters, there were more of them than there were positive, and that's unusual. So on admission, the patient shared that he maintained multiple dietary restrictions and even distilled his own water.

Despite the fact that he was very thirsty, he was really paranoid about the water that he was being offered at the hospital. So during the first 24 hours after his admission, he developed increasing levels of paranoia and auditory and visual hallucinations which resulted in him trying to escape his admission, and he was held on an involuntary psychiatric hold. He was treated with risperidone for his psychosis and IV fluids and electrolyte depletion, and then he became medically stable. When he was coherent, he reported new onset facial acne, cherry angiomas, fatigue, insomnia, subtle ataxia, and polydipsia.

So the clinicians looking after him decided to discuss this with poison control, and based on all the information that they had, suggested bromism as the most likely cause for his symptoms. So bromism, this isn't toxic masculinity, as the name might suggest, but rather bromide toxicity, which is actually a relatively rare condition today, but was relatively common, Victorian times, a little bit later, because bromide salts were actually originally used quite a bit as sedatives, but long-term use of bromide salts can lead to psychiatric conditions and things like paranoia, hallucinations, things that you've described.

And I haven't got a source for this stat, but it is vaguely written in the archives of the internet that in Victorian asylums, around 10% of patients who were there were actually there due to bromism, rather than actually due to their presenting psychiatric symptoms originally. Another fun fact about bromine

salts is it was used, or at least it's rumored to have been used to control certain urges. There was rumors that the British Army used to put it in the soldier's tea to maintain a degree of order. So why is bromide toxic?

Well, chemically, it's a halide, it's very similar to chlorine, and essentially the body mistakes it for chlorine. It gets distributed, absorbed. It's basically treated like chlorine in the body, except one of chlorine's roles is in neuronal signaling, and bromine just doesn't act in quite the same way. So it interferes, and it seems to slow down synaptic transmission, which is why it's a sedative, or why it was used as a sedative.

Interestingly, actually, it is still occasionally used as an anti-epileptic even today. Only in really severe intractable cases of epilepsy, because of that neuronal slowing, and that's why in large doses over a long period of time, you get toxicity effects causing the psychiatric symptoms. I want to talk a little bit about the abnormal biochemistry. We talked about the unusual negative anion gap, and actually this was an artifact, wasn't it?

Because the measured chloride was very high, and probably in this patient, it was relatively low, and the way we measure chlorine is we use what's called an ion selective electrode. Turns out it isn't that selective, and bromine cross-reacts with the chloride ion and produces this false reading, so it was over-reading the chloride as a result of the bromine being present. I want to talk a little bit about the phosphate as well. We didn't mention that, but he had a very low phosphate, didn't he?

And I think the paper put it down to being due to refeeding syndrome, because he had a very selective diet. But actually, I'm wondering if that could have been artifactual as well. There is really well-documented cases of iodide interfering with phosphate measurement. I couldn't find anything on bromide, but bromide and iodide are not a million miles away.

I'm wondering if it could have been interference there as well, although I've got no proof of that, there's speculation. So where did the bromide come from, and what happened in this case? Why was it there? So this individual had been reading about the negative effects of sodium chloride, but could only find information on lowering his sodium.

So he decided to conduct a personal experiment inspired by his studies in nutrition to remove chloride from his diet, and he consulted chat GPT. And chat GPT thought that it would be acceptable to swap sodium chloride for sodium bromide. And so his dietary sodium was all coming from sodium bromide rather than sodium chloride. This does raise a few questions about chat GPT, doesn't it?

Probably the first chat GPT inspired bromism case. That we know of. That we know of. Is it the first chat GPT inspired poisoning case?

Probably not. Possibly not. I'm not aware of any others. There was an early chat bot wasn't there that was a recipe bot where you could put in food items that you had in your fridge and it would create a recipe.

And it hadn't had guardrails coded in. So you could say I had arsenic and bleach in my fridge and it would give you a recipe containing arsenic and bleach. I found it quite amusing, but obviously there are health and safety risks associated with such things. For sure.

So four very, very different cases. All four of them, or most of them, actually one of them possibly inhaling something they shouldn't have done, but three of them eating something they shouldn't have done for sure. It just shows the diversity of things which can cause poisoning, doesn't it? Yeah.

And how as clinicians and as labs, we can actually support managing these patients. Cause all of these had involvement with such a diverse team, didn't they? The teams at the hospitals that directly

manage them, the lab team behind the scenes doing the analysis, both clinical analysis and the toxicology analysis. I hope you've enjoyed listening this week.

If you've liked us going through some of these cases in a slightly quicker fire fashion, do get in touch and let us know. It's a little bit of an experiment. Hope you all have an amazing week and we'll see you next time. Bye.