NEWS FROM THE PIT

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It's in the Blood: DIC and VICC

To understand VICC, we need to better understand DIC.

By Tyler Hoelscher, MD

In medicine, we all have patients that we remember. My very first memorable patient was in my third year of medical school, the first week of my clinical rotations on the labor and delivery ward. We had a mom who rolled in with a placental abruption, where the placenta tears off of the uterus, causing mom to bleed and starving the baby of oxygen. This one resulted in a crash C-section, a very fast surgery. The obstetricians had the baby out within minutes, and thankfully he was fine. Mom was closed up and taken to the recovery room. We got called back when mom started bleeding, and bleeding. It turned into a very long night with another trip to the OR.

Midway through the second operation, the anesthesiologist noted the patient was bleeding from her IV sites. An IV requires a tiny puncture, and as we've previously discussed, the body is very capable of preventing much bleeding from most small injuries. This signified an ominous turn for the patient, as she continued bleeding even more from her surgical incision. The patient had developed disseminated intravascular coagulation, or DIC.

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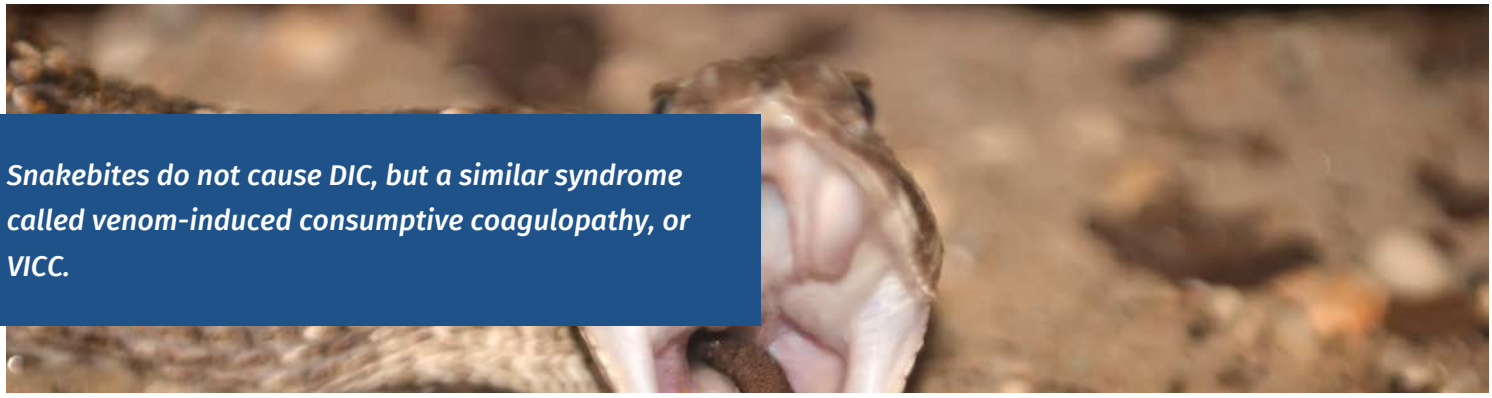
Essentially the uterus shearing from the placenta caused trauma to the blood vessel beds and exposure to amniotic fluid resulting in the profound overactivation of her clotting cascade. Rather than simply clotting the bleeding vessels, her blood was forming tiny, worthless clots throughout her body, leaving the rest of her blood thinned to the point of oozing from any site of tissue injury. 15 units of blood later, she was closed up and handed off to the ICU, where she got to meet her baby, and made a full recovery.

Now let's shift over to my favorite topic: snakes. If you've read the previous articles I've written, you'll notice I used very similar language to describe DIC as I use to describe the coagulopathy associated with snakebites, which is because they are very similar, but snakebites do not cause DIC. This is a point of confusion in medical education today. If you open a number of textbooks you will find snakebite listed among the causes of DIC. It's very easy to understand why this mistake would occur, the diagnosis of DIC is supported by several scoring criteria using laboratory values related to clotting. In DIC you see low platelets, low fibrinogen levels, low clotting proteins signified by prolonged prothrombin time or PT, and markers of clotting, like an elevated D-dimer or fibrin degradation products.

Furthermore, the derangement of these labs is identical to the way a snakebite affects these labs, but like I just said, snakebite does not cause DIC, rather it causes a similar syndrome called venom-induced consumptive coagulopathy, or VICC.

To understand VICC, we need to better understand DIC. Of all the disruptions to a body's natural clotting ability, DIC is probably the worst. Most commonly DIC occurs from terrible infections, in states of sepsis, but it is also seen in major trauma, obstetrical emergencies like my patient, severe burns, cancer, and a scattering of rarer conditions. DIC really makes blood your enemy. Sepsis-associated DIC is the hallmark here. Patients will come to the hospital sick, wind up in the ICU, and then as DIC develops, those tiny clots will build up in the blood vessels in the skin and organs. Patient's skin will turn purple and die as blood flow is cut off, and then kidneys will fail, livers will shut down, and adrenal glands have a tendency to clot, start to die, and then bleed into the abdomen. Simultaneously patients will start to bleed from their IV's, from breathing tubes, from urinary catheters, and unfortunately mortality in DIC is astronomical, approaching 50% in some studies.



Snakebites do not cause DIC, but a similar syndrome called venom-induced consumptive coagulopathy, or VICC.

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Now this should give us pause. I just said that DIC has a mortality rate of about 50%, whereas a death from a rattlesnake envenomation is very rare, well under 1% in patients receiving appropriate antivenom. Moreover, when a patient bitten by a rattlesnake dies, it's almost never from bleeding or organ failure, it's from anaphylaxis, an allergic reaction. Snakebite coagulopathy also tends to be transient, often rapidly reversible with antivenom. One last, very important point is that DIC is a disorder of bleeding and clotting, and the sequelae of clots blocking blood flow to organs and skin is not seen in North American rattlesnake bites. All in all, rattlesnake envenomation seems to mimic DIC, but with several key differences.

From a more nitpicky perspective these differences make sense. DIC is usually caused by severe damage to endothelial tissue lining the body's blood vessels which causes activation of the clotting cascade. In rattlesnake envenomation the venom itself is responsible for the coagulopathy, with many venom components independently activating the clotting cascade, destroying components of the clotting cascade before they are activated, and altering platelet function, so while the labs look the same, the causes and effects of the two syndromes are quite different.

Taking these into account, toxicologists in Australia have created the term venom-induced consumptive coagulopathy, or VICC to better describe snake envenomation. Now Australia has its fair share of snakes, but whereas we in America are very well versed in viper envenomations from our crotalids, like rattlesnakes and cottonmouths, Australia sees mostly envenomations from a group of snakes called elapids, like the brown snake and taipan. Elapid snakes tend to be less hemotoxic than vipers, and more neurotoxic, but there are well documented cases of significant coagulopathy developing after elapid bites, which was then called VICC. Since not every snakebite in Australia becomes coagulopathic, it is a useful way to characterize the specific envenomation that your patient has, with or without VICC.

Rattlesnake envenomation seems to mimic DIC, but with several key differences.

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In the US, we have not widely adopted the term VICC, and there are probably a few reasons for that. First, it is a relatively new term in medicine, and things tend to change slowly. Also, if you have a significant envenomation from a rattlesnake, you are going to have VICC. If almost all rattlesnake envenomations cause VICC, you really don't need to say your patient had a "rattlesnake envenomation with VICC", you can just say "rattlesnake envenomation". Most physicians who treat snakebites will understand what is being described. Maybe we will start to adopt this term, but in the meantime, I appreciate our Australian colleagues teasing out the subtle differences in these two diseases to help us better understand how envenomation affects our patients.