Seastrokes: A New Threat for North Carolina Swimmers?

A Case Report

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Key words: seastroke, spinal cord infarct, jellyfish sting, acute myelopathy

SUMMARY – *Spinal cord infarcts have been reported in young healthy patients following contact with the water on the beaches of North Carolina. Although their exact cause is not known, contact with a toxic marine animal has been proposed. We describe a patient who suffered a “seastroke” and review the pertinent literature.*

Introduction

A report of three unusual cases of brainstem and spinal cord infarctions occurring in young patients swimming at different beaches along the North Carolina (NC) coast was published in 1993¹. Although the etiology of the findings was never found, a possible relationship with an unknown toxic marine animal was proposed. Here we describe a new case with remarkably similar characteristics, that again presented after contact with the water in the same geographic area.

Case Report

A 16-year-old previously healthy female felt a “sting” on her foot while swimming at Wrightsville Beach, NC. Minutes after the episode, she presented with acute motor and sensory quadriplegia, flash pulmonary edema, and cardiogenic shock. She was intubated and placed on multiple pressors followed by gradual improvement. A brain and spine MRI were done. The brain MRI was unremarkable, but in the spine MRI showed extensive abnormal T2/STIR signal and expansion involving the anterior two thirds of the spinal cord. The abnormalities were present from C6 to T7 and from T10 to the conus medullaris. (Figure 1A-D). Diffusion-weighted imaging at those levels showed re-

stricted diffusion compatible with infarctions (Figure 1E ,F). No abnormal contrast enhancement was identified.

She continued to improve during her hospital stay although her motor and sensory functions did not return to normal. After approximately one month, she was transferred to a rehabilitation facility.

Discussion

In 1993, a new entity of brainstem stroke in previously healthy young patients swimming in the southeastern North Carolina coast was reported¹. In that report, three young males suffered brainstem and spinal cord infarctions. They all remembered stepping on or touching an unusual object in the water, described as “rough” or “seaweed”. Because of this, a possible contact with a toxic marine animal was proposed as the cause for their symptoms.

Later in 2001, a fourth case was reported, again with similar characteristics but this time in a lake near Lynchburg, in southwestern Virginia².

Months after this event, the media reported the discovery of *Craspedacusta sowerbii* in this lake, a freshwater jellyfish, but that is not known to be toxic. To the best of our knowledge, there have been no more similar reports after this last one.

Figure 1 Midsagittal T2 weighted images through the cervical (A), thoracic (B), and lumbar (C) spine show areas of high intramedullary signal intensity from C6 to T10 followed by a short normal-appearing spinal cord and a second area of high signal in the conus medullaris. Axial T2 image (D) through the lower cervical spine shows high signal intensity in the anterior two thirds of the cord. Midsagittal DWI (E) and corresponding ADC map (F) show high and low signals (restricted diffusion) respectively in the cervicothoracic and conal regions (arrows).

Recently, we encountered a new case that presented at our institution with a remarkably similar history to previous ones. Again, our patient remembered suffering a “sting” in her arm minutes before her symptoms began.

It is well known that some marine animals, especially some jellyfish species can cause poisoning syndromes. The effects can be localized and transient in the form of a mononeuropathy or severe with devastating health effects, even death^{3,4}. These severe poisonings are caused either by anaphylactic reactions or by direct actions of toxins⁵. Irukanji syndrome is an example of a serious reaction caused mainly by *Carukia barnesi*, a small jellyfish found mainly in the coasts of Australia. It is believed that after a minor sting, the venom produces a massive release of endogenous catecholamines causing severe muscle pain and cramps, vomiting, sweating, agitation, vasoconstriction, prostration, hypertension, and in some cases even acute heart failure and death⁶. The sting generally happens upon entering the water and typically affects young children. Most jellyfish stings are benign but severe symptoms may occur and treatment with antivenom and magnesium is recommended for them as well as hot water and lidocaine spray to reduce pain in lesser reactions^{7,8}. No Irukanji cases have been reported in the continental USA although some isolated Irukadji-like cases have been reported in South Florida⁹. In Carolina coast, the most venomous jellyfish is the Portuguese man-of-war (*Physalia physalis*) which can cause symptoms similar to those in our patient but its particular shape and relatively large size make it easy to identify. This dangerous type of jellyfish has also been reported to cause death¹⁰.

As brainstem and spinal cord strokes in young adults are extremely rare, unusual causes must be taken into consideration to explain them. Although the patients described here could represent a highly improbable coincidence, their geographic aggregation and similar characteristics makes us think of a possible common and hitherto unknown cause for them.

In our patient, it is unclear if the cardiogenic shock was the result or the cause of the spinal cord abnormalities. The fact that her brain was normal leads us to postulate that the systemic symptoms arose from the spinal cord abnormalities because if it had been the result of them, the brain would have likely also been involved. The pattern of MRI abnormalities in the anterior two thirds of the cord is typical of infarction as this corresponds to the territory supplied by

the anterior spinal artery and was confirmed by diffusion-weighted imaging showing restricted diffusion at the levels of the abnormalities (Figure 1). Since the anterior spinal artery network receives blood supply from multiple intercostal and lumbar arteries, segmental infarcts may occasionally be seen as in our patient.

Spinal cord infarctions have a poor prognosis, with little chance of recovery and significant sequelae as seen in our patient. It is unclear why in all of the reported patients the abnormalities involved only the spinal cord and/or brainstem but spared the brain. Some jellyfish toxins affect the calcium channels of neurons, a process that could lead to cytotoxic edema, failure of cellular energetics, and eventually irreversible damage such as seen in ischemia but these are not limited to the spinal cord. Although the exact mechanisms of neural tissue damage by jellyfish toxins are not known, two hypotheses exist¹¹. First, the toxins cause production of large cation-selective channels or pores that open and close cell membranes affecting voltages and potentials leading to nerve depolarization. Second, these toxins affect the passage of calcium and sodium across excitable cell membranes. This latter hypothesis has been proven when the heart is affected by jellyfish toxins as the drug verapamil restores normal function, but it has not been proven for their effects on the central nervous system. However, the effects of these toxins on parasympathetic ganglia have been explored. Ganglia contain large C-fibers which are a model with which to study hyperpolarization, and since neurons with hyperpolarization exist in both the central and peripheral nervous system, ganglionic C-fibers may reflect what goes on in the spinal cord and brain after jellyfish toxin exposure. These fibers, when exposed to the toxins, suffer abnormalities in the membrane transport of calcium and potassium which is further accentuated by inflammatory mediators also found in the toxins. Treatment with the anti-inflammatory drug indomethacin which blocks cyclogenase activity prevents some of the effects of the toxins on neurons and fibers. Affected neurons result in both parasympathetic and sympathetic symptoms and the Irukanji syndrome presents the typical picture of a catecholamine storm. Some toxins are also known to produce a post-synaptic block which may lead to immediate paralysis. How the jellyfish toxins reach the spinal cord is not known and some authors have thus postulated that damage to the spinal cord is not direct but due to systemic hypotension with subsequent infarctions.

Conclusion

We encountered a new case of an unusual form of acute myelopathy (“seastroke”) in a previously healthy young patient who had a history of swimming in the North Carolina

coastline and contact with an unusual object in the water just minutes before her symptoms started, a history similar to other reported cases. We believe that a toxin or toxic marine animal at this geographic location could be responsible for this condition.

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