SHUTTERSTOCK/PROSTOCK-STUDIO

Seafood poisoning

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Abstract: Seafood poisoning is common worldwide but is relatively unknown by clinicians and the general public and can be confused with other causes. This article discusses two common seafood poisoning cases—ciguatera and scombroid poisoning—and offers recommended nursing considerations.

Keywords: ciguatera fish poisoning, histamine fish poisoning, scombroid fish poisoning, seafood poisoning, seafood toxicity

Seafood poisoning is common worldwide, but is relatively unknown by clinicians and the general public and can be confused with other causes. This article reviews two common types of toxicity related to seafood ingestion: ciguatera and scombroid.

Ciguatera fish poisoning

Case scenario

A 38-year-old female presented to the ED with complaints of "blacking out continuously" while in her hotel room from midnight until 0200. She arrived in the ED at approximately 0300, complaining of dizziness, nausea, and blurred vision. The patient denied a history of trauma, abuse, or alcohol or drug use. Her health history did not reveal any cardiac or neurologic disorders, nor prescription or over-the-counter medication use. The patient relayed that she was from out of town, having arrived earlier in the day to attend a conference at the hotel. She had traveled internationally in the days leading up to the event, which complicated the initial history-taking process. Both flights were unremarkable. Her only activity was dinner with colleagues the night before at a local restaurant, where she consumed sushi and fish chowder.

The initial physical exam showed the patient to be in no acute distress; alert and oriented to time, place, and person but confused by what happened. Initial vital signs revealed a temperature of 98.6 °F (37 °C); heart rate 88, normal sinus rhythm without ectopy or ST segment changes; respirations 22; and BP 106/48. A finger-stick blood glucose was 110 mg/dL (normal, 70-100 mg/dL).

The ED care team initially focused treatment on dehydration because the patient appeared alert and well. Throughout the initial exam, the patient said she heard a rushing sound. Her heart rate concurrently decreased to 60 with a BP of 100/60. The patient had multiple episodes (more than 12), which started at the hotel room and continued throughout the initial hospital stay, of being unable to speak for up to 10-15 seconds.

Sinus bradycardia was noted on the monitor; intermittent episodes of 3rd-degree atrioventricular (AV) block were captured on the ECG. The patient experienced visual hallucinations—including seeing a clown in the corner of the hospital room and at times feeling disassociated with one's body—throughout the ED stay but was able to self-recover after each brief episode.

Twenty-four hours into the hospital stay, the patient felt severe pain in her left chest associated with shortness of breath and experienced asystole for more than 5 seconds on the cardiac monitor. The patient self-recovered and never had another episode of asystole.

After extensive evaluation, the patient's signs and symptoms began to resolve. Syncope and seizures, as well as a cardiac defect, were all ruled out before discharge. The patient was diagnosed with ciguatera toxicity due to ingestion of a seafood meal at the airport and the sushi and fish chowder consumed the night prior.

General symptoms include fatigue, malaise, and insomnia

The patient recalled diarrhea and bilateral upper-extremity tingling during lunch the day of, and near-syncope while presenting at the conference, although she was only able to recall this information after she was discharged and back with her primary care team.

Overview

Ciguatera fish poisoning (CFP) is the world's most frequently reported seafood poisoning. Approximately 50,000 global cases are reported annually, and it is the most common fish-related foodborne illness in the US.[1,2] In recent years, CFP has been on the rise in developed countries due to an increase in global sea temperatures and the globalization of the trade of tropical fish for human consumption, resulting in cases occurring far away from typical areas.[3]

CFP is caused by eating fish contaminated with ciguatoxin, a neurotoxin produced by dinoflagellates (marine plankton), such as *Gambierdiscus toxicus*. These plankton attach to algae, which are then eaten by fish that are subsequently consumed by larger predator fish, including barracuda, amberjack, moray eel, snapper, and certain types of grouper.[2-4]

Ciguatoxin is a neurotoxin that opens voltage-dependent sodium channels in cell membranes, triggering membrane depolarization.

The toxin is unaffected by heat, freeze-drying, or gastric acid. One consuming the fish would not be able to detect the presence of this toxin by the taste, smell, or appearance of the fish.[1]

Clinical manifestations

Signs and symptoms of CFP typically occur within 1 to 3 hours, with increasing severity in 4 to 6 hours. Most people develop signs and symptoms within 24 hours.[5]

General symptoms include fatigue, malaise, and insomnia.[2] Gastrointestinal (GI) symptoms begin within 6 to 12 hours and include abdominal pain, nausea, vomiting, and diarrhea.

Musculoskeletal symptoms include myalgia and arthralgia.

Neurologic signs and symptoms, which present within the first 2 days of toxin consumption, include pruritus, vertigo, ataxia, blurred vision, paresthesias, headache, dizziness, seizures, hallucinations, and coma.

Other neurologic symptoms include temperature-related dysesthesias (cold stimuli perceived as hot or producing an abnormal, unpleasant sensation). Temperature-related dysesthesias may more commonly be experienced as burning pain upon cold exposure.[1] Additional neurologic symptoms may include tooth pain or the sensation that teeth are loose.[1]

Neuropsychologic symptoms are typically reported within days or weeks and include confusion, memory loss, lack of concentration, depression, anxiety, or irritability.

Cardiopulmonary signs can develop early in the illness and

include hypotension, hypertension, and dysrhythmias such as bradycardia, tachycardia, and AV block. Respiratory failure may also occur.[6] CFP is rarely fatal but death may occur if severe dehydration, shock, or respiratory failure is present.

Diagnosis

No clinical test is available to diagnose CFP. Diagnosis is made based on clinical presentation and recent fish-eating history. Multiple individuals reporting similar signs and symptoms after consuming the same fish strongly indicate CFP.

Treatment

Care is primarily supportive by treating signs and symptoms until they resolve, which typically occurs within a few days. However, some signs and symptoms may last for weeks or months. Supportive care may include I.V. fluid resuscitation and I.V. vasopressor support for hypotension.[2]

Calcium channel blockers have been suggested to manage neurologic signs and symptoms associated with CFP since the ciguatera toxin can affect sodium channels in nerve cells.[7] Calcium channel blockers may alleviate some of the neurologic manifestations by modulating the flow of calcium ions into the nerve cell, thereby reducing the abnormal nerve activity caused by the toxin.

Bradycardia may require I.V. atropine and respiratory failure may require rapid sequence endotracheal intubation.

I.V. mannitol has been recommended as a primary treatment for CFP to reduce the severity and duration of neurologic signs and symptoms by reducing neuronal edema and decreasing uncontrolled and repetitive action potentials after a stimulus.[4] Recent reviews have questioned this practice due to low evidence levels. Additional studies are needed for this recommendation. If Mannitol is given, it should follow adequate intravascular volume restoration.

Paresthesia, described as tingling in this case of CFP, is associated with the release of cytokines and complex actions on ion channels in cell membranes, causing cell excitability.[2] Amitriptyline can be used for pruritus and may be effective for paresthesia and depression.[3] Gabapentin can also be used for neuropathic signs and symptoms.[3]

Antimotility agents should generally be avoided in the treatment of CFP-associated diarrhea. These agents work by reducing peristalsis and increasing the time it takes for stool to pass through the intestines, allowing the toxin to stay in the body longer. Diarrhea may be beneficial in removing the ingested toxin.

Nursing considerations

Discharge instructions should include avoidance of any fish, caffeine, alcohol, and nuts for 6 months, as they may trigger a recurrence or worsen signs and symptoms.[2]

CFP signs and symptoms can persist for weeks or even months, and the severity can vary. Nurses should teach patients to avoid eating high-risk fish such as barracuda or moray eel and avoid consuming fish parts such as the head, intestines, liver, or roe as the toxin can be concentrated in these parts. Individuals recovering from CFP should consult with healthcare professionals for guidance on their specific situation, dietary restrictions, and managing signs and symptoms.[1]

CFP is a reportable illness in all states, so nurses must ensure that local public health authorities are contacted to assist in confirming the diagnosis and to prevent further consumption of contaminated fish.

Scombroid fish poisoning

Case scenario

A 50-year-old female presented to the ED with a sudden onset of intense facial flushing, headache, palpitations, wheezing, and dyspnea 10 minutes after eating a tuna burger at a local restaurant. The patient had self-administered albuterol inhaler and oral diphenhydramine prior to arrival. She had no known allergies or exposures. The patient was awake and alert, with initial vital signs: temperature 98.6 °F (37 °C); heart rate 160 (supraventricular tachycardia); respirations 40; BP 150/110; SpO_2 96% on room air. Scattered inspiratory wheezes were noted on auscultation.

Immediate ED interventions included supplemental oxygen at 4 L/minute via nasal cannula and bilateral I.V. access with 0.9% sodium chloride infusion. Medications administered included I.V. diphenhydramine, I.V. famotidine, and I.V. methylprednisolone.

The patient exhibited intense shaking chills, which dissipated as her facial flushing slowly resolved. The patient was given a differential diagnosis of anaphylaxis versus scombroid fish poisoning (SFP), with the final diagnosis of SFP. The patient's clinical status was stable upon discharge. She was given prescriptions for oral ranitidine and prednisone. She was instructed to use diphenhydramine for the recurrence of mild signs and symptoms and to return to ED if she had any difficulty breathing.

Overview

SFP, also known as histamine fish poisoning, is caused by ingesting dark-fleshed fish such as tuna and mackerel.[8,9] Other fish not of the Scombridae family but can cause SFP include mahi mahi, salmon, swordfish, tilapia, trout, anchovies, and sardines.[8,9]

Occurrences are common throughout the world. In the US and

Europe, SFP accounts for up to 40% of seafood-borne illness outbreaks.[9] Between 2011 and 2021, 165 outbreaks and 512 illnesses were reported to the US Foodborne Disease Outbreak Surveillance System.[9]

Pathophysiology

SFP occurs when fish are improperly stored after being caught (temperatures greater than 40 °F [4 °C]). Dark fish meat has a high concentration of the amino acid histidine within the muscle tissue. Bacteria metabolize histidine into histamine. The presence of histamine may result in a metallic or peppery flavor; however, the meat can also be normal in appearance and taste. Histamine and other toxins cannot be destroyed by cooking, freezing, or subsequent refrigeration.

Clinical manifestations

The severity of signs and symptoms depends on the histamine quantity consumed, the rate of deactivation of histamine, and the individual's sensitivity to histamine. Signs and symptoms of SFP are generally of short duration and may be confused with other conditions, leading to underreporting. Most individuals with SFP have signs and symptoms consistent with histamine poisoning within 90 minutes.

Signs and symptoms include cutaneous flushing of the face and neck; pruritus; angioedema; and GI manifestations such as nausea, vomiting, abdominal cramps, and diarrhea. In rare cases, bronchospasm, hypotension, and myocardial ischemia may occur. SFP has been mistaken for acute cardiac disorders or other shock states in these rare cases.

Diagnosis

Diagnosis is made by clinical examination, with a recent history of fish ingestion. Assessment of the patient's plasma histamine level may be useful. A key assessment finding is allergic-like reactions in unrelated persons after consuming the same fish.

SFP is typically self-limiting and mild signs and symptoms usually resolve within 24 to 48 hours.

Treatment

SFP is typically self-limiting and mild signs and symptoms usually resolve within 24 to 48 hours. SFP is treated with H1 antihistamines, such as diphenhydramine, and H2 antihistamines, such as cimetidine and famotidine.[8] Inhaled bronchodilators may be needed for bronchospasm. However, corticosteroids are ineffective.

Nursing considerations

Obtaining an accurate health history, including recent fish consumption, is crucial for accurate diagnosis. Patient education should include prevention of SFP. The key preventive measure is proper handling and storing fish: they should be immediately chilled below 40 °F (4 °C) and adequately refrigerated after catching.[8]

Nurses should be aware of the clinical manifestations of SFP to facilitate rapid diagnosis and treatment. Nurses should also ensure that the local public health authorities are contacted to assist in confirming the diagnosis and prevent further consumption of contaminated fish.

Conclusion

Fortunately, both of these seafood-associated diseases are relatively self-limiting and respond well to interventions when not delayed, but they do require a degree of awareness and suspicion, especially in areas where seafood consumption is common. Clinicians should also be alert for patients with similar signs and symptoms who may have recently returned from endemic areas. ■

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