CLINICAL VIGNETTE

Elevated Mercury with Normalization after Cessation of Seafood Intake

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Case Report

Twenty-three-year-old male presented with abdominal pain, fatigue and brain fog, present for two months. The pain would come and go and was not severe. He describes it as a 3/10 generalized pain which did not radiate. He has normal bowel movements and no pain medications. He reports eating poke bowls several times a week, usually with tuna. Vital signed include blood pressure 128/78, pulse 80, weight 125 lbs. Physical exam was normal. Specifically, normal bowel sounds and no tenderness to palpation. Labs included normal CBC, CMP, ESR, CRP, B12, and folate. Blood mercury was elevated at 22 ng/mL. The patient was asked to refrain from all seafood for a week and his symptoms resolved. Repeat labs were normal.

Discussion

The clinical significance of low to moderate levels of mercury exposure is an area of great controversy and recent interest.[1] The health hazards of high-level mercury exposure have been well established. The infamous outbreak of Minamata disease (methylmercury poisoning) in Japan in 1956 brought attention to the neurotoxic impact of mercury exposure, including cerebellar ataxia, sensory abnormalities, and speech disturbances.[2] More recently, there has been a shift in interest to both the short and long-term clinical impact of low-level mercury exposure. The Seychelles Child Development study, a large, longitudinal study examined the impact of methyl mercury exposure on child development. They reported no consistent association between low-level methyl mercury exposure and adverse developmental effects in children.[3] However, other studies have reported the short-term impacts of increased mercury exposure, including central and peripheral nervous system dysfunction, renal injury, and pulmonary irritation.[4]

Mercury exposure in humans is primarily derived from three sources: dental amalgams, fish consumption, and vaccines.[4,5] Of note, each exposes an individual to a different form of mercury with varying toxicologic impacts and clinical manifestations.[4] Fish consumption is associated with organic mercury, or methylmercury exposure. Of note, fish at the top of the food chain, such as swordfish and tuna, have larger amounts of mercury stored in their tissue. Methyl mercury concentrates primarily in the brain, liver, and kidney.[6] Most seen clinical manifestations impact the central nervous system and include paresthesias of the hands and feet, ataxia, and visual and hearing loss.[4] There is typically a latent period of multiple months between exposure to methyl mercury and manifestation of symptoms.[4] Interestingly, studies in the past have suggested an association between methyl mercury exposure and increased risk of myocardial infarction,[7] however larger studies have found no such association.[8]

There are multiple markers to assess mercury exposure. Mercury is differentially distributed throughout organ tissues; however, the most common method of mercury measurement is via urine, blood, hair, and fingernails.[1] Blood and urine will reflect more recent exposure to mercury however do not reflect total body burden of mercury.[6,8] Normal blood mercury levels are below 20 μg/L, however it is usually found to be below 10 μg/L. Urine mercury levels are less useful for detecting methyl mercury levels and are more helpful for detecting inorganic and metallic mercury exposure.[1]

This case highlights a underrecognized and uncommon diagnosis for common complaints of gastrointestinal upset, fatigue, and central nervous system dysfunction. As mercury can be deposited in a wide range of tissues, the impact and clinical manifestations of mercury poisoning can vary widely and are often underrecognized.[6] While environmental exposure to pathologic levels of mercury is uncommon, this case highlights the importance of obtaining a detailed nutritional history to better understand patients' potential mercury intake via fish consumption. With the growing popularity of poke in the Los Angeles area and nationally, abnormal levels of mercury may be increasingly common and should be included on the differential diagnoses for patients for whom a uniting diagnosis is unclear. Specifically, when exposure to mercury via fish consumption is a concern, blood mercury rather than urine mercury should be utilized as a diagnostic tool.

Conclusion

Obtaining a detailed nutrition history and exposure history to rule out mercury toxicity can be useful in uncovering the etiology of generalized central nervous system complaints, such as brain fog, and gastrointestinal upset, as in this patient. Mercury toxicity is unrecognized in the differential diagnosis for central nervous system, peripheral nervous system, and gastrointestinal complaints. It should be investigated if a uniting diagnosis is unclear.

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