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Vitamin A deficiency: An adult patient with night blindness and cirrhosis in an uncommon geographical area

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Abstract

Background: Vitamin A Deficiency (VAD) is a common entity seen in developing countries, usually in pediatric patients or pregnant women, secondary to low dietary intake. Here we describe a case of a patient with chronic liver disease within the United States who developed symptomatic Vitamin A deficiency.

Case description: This case describes a 64-year-old female patient with symptomatic VAD, likely secondary to liver cirrhosis in the setting of Hepatitis C. The patient presented with night blindness and blurry vision. She was successfully managed with direct replacement of Vitamin A.

Conclusion: This case describes a rare presentation within a developed country and highlights the importance of symptom recognition for VAD to avoid permanent consequences such as blindness. This case also prompts the need for more investigation into the association of hepatic injury and VAD.

Keywords: Vitamin A deficiency; Liver cirrhosis; Night blindness; Case report.

Introduction

Vitamin A Deficiency (VAD) is classically associated with malnutrition, often isolated to developing countries in pediatric or pregnant patients, likely due to these patients' increased demand. Consequences are severe if such a deficiency is left untreated [1]. When symptomatic, patients most commonly initially experience impaired night vision, with sequelae of epithelial change and permanent blindness, along with immunological impairment [1,2]. Vitamin A deficiency has also been seen to rarely present in patients with chronic disease and malnutrition in more developed areas, often secondary to lipid malabsorption [1]. Hepatic stem cells have been associated to play a key role in storage of Vitamin A, with hepatic injury resulting in lower levels of stored Vitamin A [3]. We present a case of a 64-year-old female patient who developed symptomatic Vitamin A deficiency in the setting of liver cirrhosis secondary to hepatitis C within the United States.

Case presentation

A 64-year-old female patient presented to us for follow up of chronic anemia and liver cirrhosis secondary to Hepatitis C. She complained of fatigue, headaches, fullness in her neck, difficulty swallowing, blurred vision, and difficulty seeing in the dark. She stated she was unable to see anything in front her when it was dark. She could not see her grandson when he was in front of her. She had undergone and ophthalmology evaluation and was

thought to be having visual migraines. She had been treated for Hepatitis C approximately eight years ago and her last PCR was negative. Her last EGD and visit with a GI specialist had not been for many years. Prior lab work had revealed a folate deficiency and Vitamin D deficiency. The patient was currently on Vitamin D and folate replacement, as well as oral ferrous sulfate. The patient denied experiencing any dizziness or focal weakness, hearing loss, eye pain, or easy bruising or bleeding. She denied any other complaints or issues. Physical exam was unremarkable.

The patient's last endoscopy had revealed Grade 2 varices secondary to liver cirrhosis and splenic sequestration had been found on previous CT with no visible masses. Labs revealed a hemoglobin of 10.5 g/dl and other blood counts were unremarkable. Platelets were 129,000/µl.

A head CT was negative. The patient's ophthalmology evaluation had been negative, so a neurology and retinal consult were placed. An ultrasound of the head and neck revealed vascular congestion of bilateral internal jugular veins. Due to the night blindness, a Vitamin A level was checked and came back undetectable. She was started on Vitamin A replacement.

The patient was prescribed 100,000 units of intramuscular Vitamin A for three days, followed by a daily regimen of oral 20,000 units of Vitamin A. Upon follow up one month later, the patient reported she had received the intramuscular Vitamin A replacement, but was still working to obtain oral Vitamin A. She reported a 50-60% improvement of her night blindness. The patient was referred for further follow up testing at a vision rehabilitation center at one and three months after treatment for comparison to baseline findings and encouraged to continue to follow up with us to ensure symptomatic resolution.

Discussion

Vitamin A is a fat-soluble vitamin necessary for epithelium differentiation and proliferation, immunity, and vision [4]. Subclinical VAD is defined as a serum or plasma concentration of <0.70 umol/L and severe VAD as <0.35 umol/L [5]. In humans, Vitamin A must be obtained from the diet. Preformed Vitamin A, such as retinyl esters, are obtained from animal products, while provitamin A carotenoids must be obtained from plants [6]. Example foods include milk, liver, cheese, and eggs, as well as vegetables such as carrots, leafy greens, and orange-yellow fruits such as mangos [5].

Vitamin A deficiency is most prevalent in developing countries due to poor dietary intake and is a major cause of preventable blindness in children [5]. VAD typically affects pediatric populations and pregnant women due to increased demand for development, fetal growth and breastfeeding [7]. In developed countries, VAD is much rarer, but has been found to occur secondary to malabsorptive conditions such as gastric bypass patients, irritable bowel disorder, pancreatitis, and cystic fibrosis [8-10]. Isolated cases have also been seen in conditions that cause direct hindrances to intake such as specific diets in autism spectrum disorder [11].

A lack of Vitamin A results in a stepwise degeneration of the rods and cones within the eye. In the eye, rods are responsible for night vision and motion detection, while cones are crucial for color and light absorption [4,12]. VAD first affects rods due to Vitamin A's role as a precursor to rhodopsin, a main component of rods, resulting in the classic night blindness symptom, with eventual progression to degeneration of the cones and loss of daytime vision [12,13]. Vitamin A is also necessary for epithelium differentiation and proliferation within the eye [2]. Without Vitamin A, ocular epithelial atrophy occurs [4]. This results in Bitot Spots, white/grey spots present on the peripheral retina, then progression to xeropthalmia, and eventual permanent blindness [13].

Cases have previously reported deficient circulating Vitamin A forms in chronic alcoholic patients and Non-alcoholic fatty liver disease, typically coinciding with deficiencies of other fat-soluble vitamins [14,15]. Other cases have also demonstrated possible VAD in chronic cholestatic disease such as primary biliary cholangitis, primary sclerosing cholangitis, and biliary atresia, with a documented need for more investigation into the possibility of a pathological role of VAD in the development of such biliary diseases [16].

Absorption of Vitamin A is largely dependent on gastric and pancreatic enzymes, bile salts, and formation of chylomicrons, all which are amplified by dietary fat [6]. Preformed Vitamin A, usually in the form of beta-carotene, is converted to retinol within the brush border of the small intestine [17]. Hepatocytes have been shown to be one of the main cell types responsible for the uptake of retinols, while Hepatic Stellate Cells (HSC) are a key cell for their storage [18]. The liver stores a significant portion of the bodies Vitamin A, ranging from 60-95% and is thought to be responsible for providing these stores to circulation when dietary intake is insufficient [15].

Hepatocytes uptake chylomicrons containing large amounts of retinyl-esters, which are either stored in HSCs or hydrolyzed and released into circulation as retinol [3]. HSCs are only able to store retinyl esters in their dormant state [3]. Acute hepatic injury results in activation of HSCs, thus releasing their stores of retinyl-esters [16]. When that injury is prolonged or chronic, HSCs remain in an activated state with no ability to store retinyl esters, resulting in lower circulating and storage levels of Vitamin A precursors [3].

Conclusion

Our patient's presentation of symptomatic Vitamin A deficiency, likely secondary to chronic liver injury in the setting of Hepatitis C, was a rare presentation in a developed country such as the United States. VAD tends to be associated with severe malnutrition in developing worlds, but there are other causes that can result in deficiency of Vitamin A outside of these areas. The recognition of VAD symptoms is crucial, as other associations of VAD after hepatic injury have also been described, prompting that more investigation is needed to establish such a relationship. VAD in our patient was easily corrected through Vitamin A replacement with resolution of symptoms, improvement in quality of life, and prevention of permanent blindness. Though considered a rare entity within developed areas, there must be a heightened awareness to recognize symptoms and address deficiency to avoid permanent consequences.

Declarations

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Conflicts of interest: None.

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