

PELLAGRA IN ASSOCIATION WITH HALLUCINATION AND OVER-SALIVATION

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ABSTRACT

A 33 yr old African female, living in Makkah, presented to ER with decreased LOC & diarrhoea which was observed by the attending nurse with no more available history .On examination: she was afebrile; Pulse rate was 100 bpm, BP 90/60 mmHg, R/R 18 breaths/min, O2 sat. 98% on room air. She was disoriented having hallucination, underweight, dehydrated, pale. She was diagnosed as acute confusional state, and she received IVF, PRBC, and correction of electrolytes. Patient has received empirical antibiotics since admission and for 3 days, and conservative treatment but has not improved yet! On the 4th day of admission, niacin deficiency was suspected. Therefore, niacin was started at dose of 100 mg BID per oral. By the 6th day of the hospital stay, patient had become oriented, communicating, with no diarrhoea, and the skin lesion started to improve. In conclusion, this case calls attention to pellagra when considering the differential diagnosis of hallucination and oversalivation.

KEY-WORDS: Pellagra; Over-Salivation; Hallucination

Introduction

Pellagra is defined by the systemic disease resulting from niacin (vitamin B3) deficiency, and it is clinically manifested by the **4Ds**: photosensitive dermatitis, diarrhoea, dementia, and death. Early symptoms of pellagra include lassitude, weakness, loss of appetite, mild digestive disturbances, and psychiatric or emotional distress (e.g., anxiety, irritability, and depression), Burning sensations, headache. This vitamin deficiency responds to treatment with nicotinic acid (niacin).^[1] Pellagra is observed in malnourished individuals. The diagnosis is often overlooked or delayed, occasionally with life-threatening consequences. It is International. Although the exact incidence of pellagra in other countries is not known, pellagra occurs mainly in developing nations in which corn and corn products are the major food sources. Pellagra also occurs in ethnic populations whose diets are deficient in niacin and/or tryptophan. No racial or sex predilection is reported. It is typically a disease that occurs in adults and may develop in adolescents and young children if they are exposed to a pellagrigenic diet and rarely occurs in infants.^[2] Untreated pellagra results in death from multi-organ failure. The morbidity of

pellagra is related to its effects on the organ systems involved. Cellular functions in multiple organs and tissues are affected by niacin deficiency; the clinical expressions of pellagra are diverse. Pathologic changes in the skin include vascular dilatation, proliferation of the endothelial lining, perivascular lymphocytic infiltration, hyperkeratinisation, and subsequent atrophy of the epidermis. Mucosal inflammation and atrophy involve most of the GI tract. Evidence of glossitis and atrophy of the papillae of the tongue are characteristic findings, along with gastritis and subsequent gastric mucosal atrophy. Acute inflammation of the small intestine and colon are also commonly noted.^[1] Pathologic changes in the nervous system can be found in the brain, spinal cord, and peripheral nerves. Findings include patchy demyelination and degeneration of the affected parts of the nervous system. The morbidity of pellagra is related to its effects on the organ systems involved. Systemic effects of the disease include malaise, apathy, weakness, and lassitude. GI tract involvement leads to a malabsorptive state and a subsequent failure to thrive. Neurologic manifestations include anxiety, depression, delusions, hallucinations, and stupor. In 1926, Goldberger reported that nicotinamide was a preventive factor of pellagra.^[1]

A search was conducted, but we did not find any case of pellagra does not involve other medical condition and associated with hallucination (which is uncommon presentation) and over-salivation.

Case Report

A 33 year old African female, living in Makkah, presented to ER with decreased LOC & diarrhoea which was observed by the attending nurse with no more available history.

On examination: she was afebrile; Pulse rate was 100 bpm, BP 90/60 mmHg, R/R 18 breaths/min, O₂ saturation 98% on room air. She was disoriented having hallucination, underweight, dehydrated, pale. She had scaly skin changes over her hands, forearms and legs. Also, she had red smooth tongue with oversalivation. There was no clubbing, cyanosis, or jaundice. CVS examination: normal S1 and S2, no murmurs. Chest: clear equal air entry, no wheezes or crackles. Per Abdomen examination: abdomen was soft, lax, no tenderness, liver span 14 cm (hepatomegaly), and PR negative. CNS examination: opening her eyes spontaneously, not obeying commands, talking inappropriately, moving 4 limbs, no neck stiffness and meningeal signs are negative.

Laboratory studies revealed normal WBC and platelet, low Hb (microcytic hypochromic anaemia), hypokalaemia due to diarrhoea, hypoalbuminemia, low iron and ferritin. She had normal renal function test, LDH, ALT, AST, Na, Po₄, and Mg. CXR: normal findings. ECG: sinus tachycardia with regular rhythm. CT brain: normal findings. U/S abdomen: fatty liver with minimal ascites. She had negative stool analysis and C/S, CSF analysis, toxicology screen, brucella, salmonella, malaria film, HIV, tuberculin test, and septic screen. Niacin level testing is not readily available.

Hospital Course: She was diagnosed as acute confusional state, and she received IVF, PRBC, and correction of electrolytes. Patient has received empirical antibiotics since admission and for 3 days, and conservative treatment but has not improved yet! Patient was still confused, having diarrhoea, and oversalivation. On the 4th day of

admission, niacin deficiency was suspected. Therefore, niacin was started at dose of 100 mg BID per oral. By the 6th day of the hospital stay, Patient had become oriented, communicating, with no diarrhoea, and the skin lesion started to improve. On the 7th day, the patient was able to tolerate oral intake with minimal pain on swallowing which was improved with mouth wash. At the end of the 15th day, she was discharged on niacin.

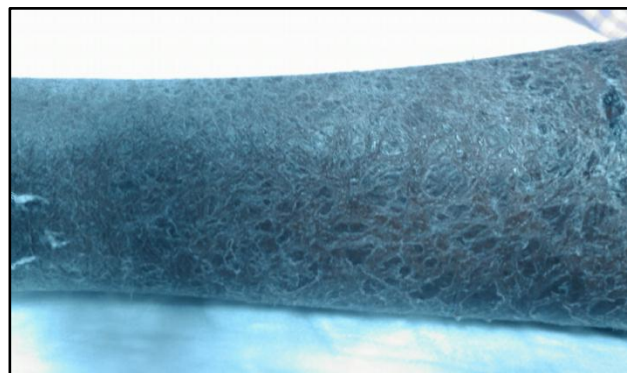


Figure-1: Dry Scaly Skin Changes over the Leg

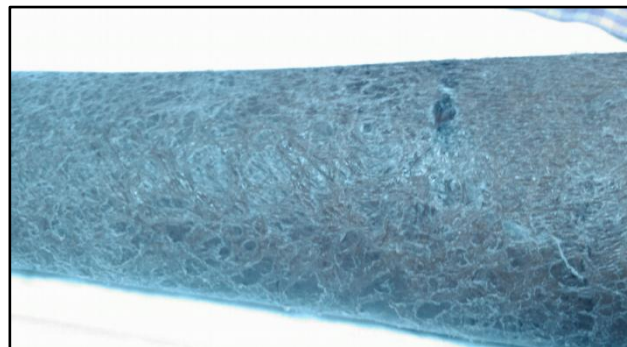


Figure-2: Dry Scaly Skin Changes over the Other Leg

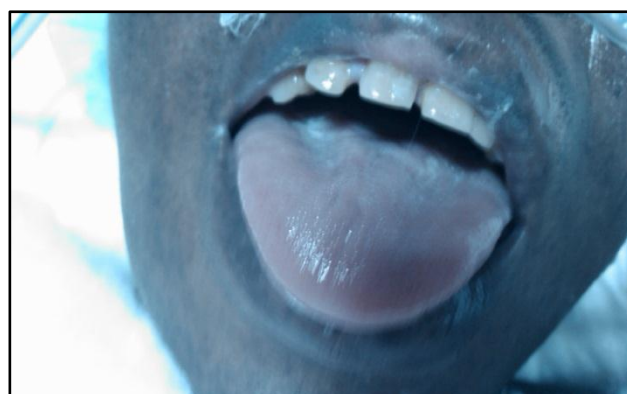


Figure-3: Glossitis which leads to Oversalivation

Discussion

Pellagra is the most frequent clinical feature of nutritional deficiency in adults in rural populations in the Third World, whose staple diet is niacin deficient jawar or maize with inadequate animal protein, fruits and vegetables.^[2]

Pellagra is caused not only by a poor diet but also can be secondary to conditions that interfere with niacin intake, absorption, or processing, such as Crohn's disease, severe ulcerative colitis, prolonged diarrhoea, gastrectomy, hepatic cirrhosis, chronic alcoholism, or anorexia nervosa. Metabolic derangements, such as Hartnup disease or carcinoid syndrome may also lead to pellagra. Pellagra has been increasingly reported in HIV patients. Drugs that interfere with niacin or tryptophan metabolism, such as isoniazid, pyrazinamide, 6-mercaptopurine, 5-fluorouracil, phenytoin, azathioprine, chloramphenicol, and ethionamide can also cause pellagra-like symptoms. Lastly, a diet rich in leucine can also lead to pellagra because this amino-acid interferes with the enzymatic conversion of tryptophan to niacin.^[3]

The early symptoms of pellagra include loss of appetite, generalized weakness, irritability, abdominal pain and vomiting. Later symptoms are bright red glossitis, chronic or recurrent diarrhoea (watery, but occasionally bloody), which leads to a state of malnutrition and cachexia.^[4] The characteristic skin rash is characterized by pigmentation and scaling, particularly involving the sun exposed areas. As pellagra advances, neuropsychiatric symptoms such as photophobia, asthenia, depression, hallucinations, memory loss, and psychosis begin. The patient become disoriented, confused, and delirious; stupor and death result if untreated.^[1,3]

The diagnosis of pellagra is based on the characteristic clinical presentation and rapid response to oral niacin supplementation.^[1]

The differential diagnosis includes phototoxic and photoallergic skin reactions, porphyrias and pseudoporphyria, lupus erythematosus, and polymorphous light eruption. Also, primary pellagra should be differentiated from secondary pellagra.^[1]

This patient presented with dry scaly skin changes over her legs and diarrhoea. Also she had over salivation with red smooth tongue and

hallucination, which are rare presentations of pellagra.

Treatment

The administration of niacin has a dramatic curative impact on pellagra. The daily recommended dose is 300 mg of nicotinamide in divided doses, and treatment should continue for 3–4 weeks. Large therapeutic amounts of niacin should be provided in the form of nicotinamide, which does not produce the side effects encountered when nicotinic acid is administered. Acute inflammation of the tongue and mouth, as well as diarrhoea, subsides in a few days. The dementia and dermatitis usually improve significantly within the first week of therapy. In chronic cases, a longer recovery period is required, but appetite and general physical health improve rapidly. It is also recommended to administer a vitamin B complex preparation or a yeast product since patients with pellagra very often have a deficiency of other B vitamin compounds.

Conclusion

In conclusion, this case calls attention to pellagra when considering the differential diagnosis of hallucination and oversalivation.

References

1. Hegyi J, Schwartz RA, Hegyi V. Pellagra: dermatitis, dementia, and diarrhea. *Int J Dermatol.* 2004;43(1):1-5.
2. World Health Organization United Nations High Commissions for Refugees. Pellagra and its prevention and control in major emergencies. Geneva: WHO: 2000; 4-5.
3. Karthikeyan K, Thappa DM. Pellagra and skin. *Int J Dermatol.* 2002;41(8):476-81.
4. Oztürk F, Koca R, Aydin M, Cantürk MT, Akpolat I, Küçüködük S. Pellagra: a sporadic pediatric case with a full triad of symptoms. *Cutis.* 2001;68(1):31-4.

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