

## A typical case of pellagra

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Mr. Lama 40 year old male from Sindhupalchowk district was admitted with chief complaints of loose motion & irrelevant talking of 5-7 days duration. He was a known case of alcoholic liver disease. He had been taking about 1.5 liters of locally made alcohol for the past 5 years. He gave history of having large volume, watery faeces without blood. According to his family members, Mr. Lama had poor memory for recent events and was talking irrelevantly 5-7 days prior to admission. There was no history of pain abdomen, headache, loss of consciousness, fever, yellowish discoloration of skin

& mucus membrane. On examination patient was confused, dehydrated, afebrile, icteric & pale. Scaly blackish dry skin on dorsum of hands & feet of one month's duration was present. Pulse was 152 per minute and BP 150/110mmHg on sitting posture. There were no abnormal findings in the chest & cardiovascular system. Non-tender & firm hepatomegaly with no bruit was noted. Liver Span was 15 cm with no enlargement of other organ. There was no free fluid in the abdomen. The patient was found to have loss of memory for recent events.



Before Treatment



After Treatment

### Investigations

Total WBC count was increased with neutrophilic leucocytosis with normal haemoglobin. Blood urea, creatinine, sugar & electrolytes were within normal limit. Liver function tests showed unconjugated bilirubinaemia with normal liver enzymes. PT-INR was 1.42. Serology for HIV/HBsAg was negative. X-ray chest & electrocardiogram were normal. Abdominal USG scanning revealed hepatomegaly.

On the basis of history & clinical features *alcoholic liver disease* with *pellagra* was diagnosed.

### Management

Initially this patient was managed with IV fluids. 60 mg niacin thrice daily was started orally. During his

stay in the hospital, he also received chlorthalidone & thiamine. Repeated blood test after initial treatment revealed normal bilirubin level after 7 days of treatment. Skin changes started showing improvement after two days and the skin became normal by tenth day whereas loose motion stopped by sixth day. His mental state improved by fifth day.

### Discussion

In early part of 20<sup>th</sup> century, pellagra was considered infectious disease.

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Joseph Goldberger, an epidemiologist, experimented on himself and his wife and proved this condition to be due to dietary deficiency<sup>1</sup>. Niacin, an essential component of nicotinamide adenine dinucleotide (NAD) and nicotinamide adenine dinucleotide phosphate (NADP), coenzymes for many oxidation-reduction reactions, is the generic term for nicotinic acid (pyridine-3-carboxylic acid) and derivatives that exhibit the nutritional activity of nicotinic acid. It can be formed from the essential amino acid tryptophan. In our body, an average of about 1 mg of niacin is formed from 60 mg of dietary tryptophan. Accordingly, estimates of the adequacy of dietary intake must take into account the content of both tryptophan and niacin. Many foodstuffs, especially cereals, contain bound forms of niacin from which the vitamin is not nutritionally available<sup>2,3</sup>.

Pellagra is a chronic wasting disease classically characterized by diarrhoea, dementia & dermatitis. Dry, black cracking and scaly skin are the typical changes seen in pellagra. Such skin changes seen in neck & upper chest is popularly known as “*Casal's necklace*”<sup>4</sup>. The dermatitis is bilateral, symmetric, and present in sites exposed to sunlight and is due to photosensitivity. The mental changes are less discrete. Fatigue, insomnia, and apathy may precede the development of an encephalopathy characterized by confusion, disorientation, hallucination, loss of memory, and eventually, organic psychosis. Diarrhoea, when present, results from widespread inflammation of the mucous surfaces. Other mucosal abnormalities include achlorhydria, glossitis, stomatitis, and vaginitis. The skin lesions are characterized by hyperkeratosis, hyperpigmentation, and desquamation<sup>3</sup>. The endemic disease is usually associated with a high intake of maize (American corn) or millet (sorghum, jowar), for example in parts of Africa and can be cured by the administration of niacin<sup>4,5,6</sup>. Family income is an important factor, since it determines the extent to which the foods containing the pellagra-preventing essential could be purchased, especially in industrial communities where the entire population is on the margin of subsistence<sup>1</sup>. It is a nutritional disease in developing countries due to deficiency of niacin whereas in developed countries it occurs with alcohol abuse and chronic small intestinal disease. It takes at least 8 weeks of niacin/tryptophan deficient diet for Pellagra to develop. Daily recommended dose of niacin is 16-21mg/day & therapeutic dose is 100mg orally every 8hourly<sup>4,5</sup>. WHO recommended daily intake of niacin equivalents is 6.6 mg (infants) to 21.1 mg (adults)<sup>6,7</sup>. Improvement with niacin occurs rapidly<sup>8-11</sup>.

## Conclusion

No biochemical test is of diagnostic value for pellagra, and diagnosis must be based on clinical features and response to replacement therapy<sup>3</sup>. Early diagnosis is crucial in the management of pellagra and requires a high degree of suspicion in a likely clinical background such as in our present case.

## References

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