

Tropical sprue

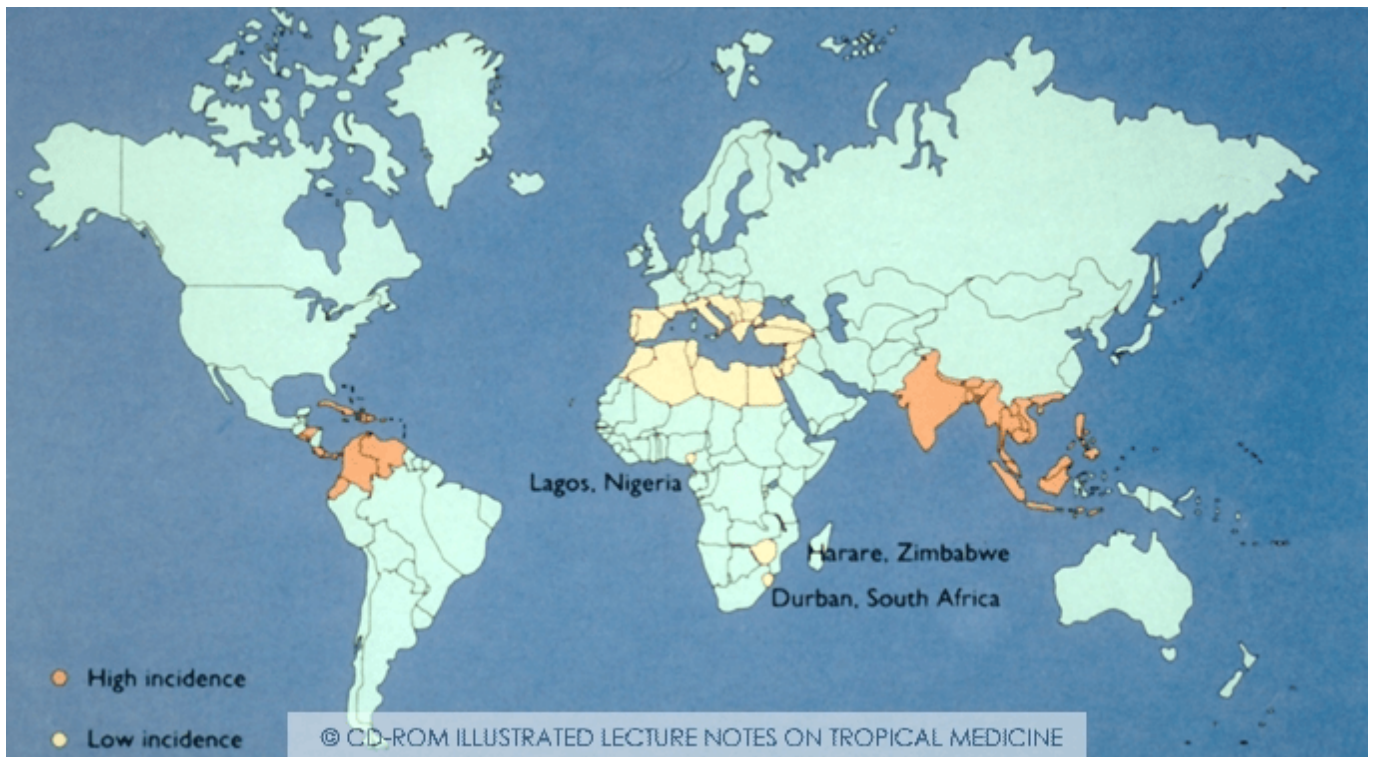
Tropical sprue	3
General	3
Clinical aspects	4
Diagnosis	5
Treatment	5

Tropical sprue

General

Tropical Sprue is largely limited to within about 30 degrees north and south of the equator. It was responsible for one-sixth of all casualties sustained by the Allied forces in India and Southeast Asia during World War II. Tropical sprue is an acquired disease of unknown origin. An infectious origin appears probable and the term “post-infectious malabsorption” is also used. Possibly there is an initial insult at the level of the jejunal-ileal enterocytes, followed by bacterial overgrowth with enterotoxigenic strains. The disease is characterised by abnormalities of the mucosa in the small intestine with chronic malabsorption, multiple nutritional deficiencies and anaemia. The malabsorption is generalised and affects absorption of proteins, fat, carbohydrates, minerals and vitamins (typical is iron and folate deficiency). Good response to treatment with doxycycline and iron-folate supplements is seen.

Tropical sprue occurs chiefly in the Caribbean, India, Nepal and Southeast Asia, in both the indigenous populations and immigrants. Cases have been reported from Mauritius, Fiji, southern Italy, Guyana and Central America. In Africa the disease is apparently very rare, although cases have been reported from Zimbabwe.



Map of areas with post-infective malabsorption, also known as tropical sprue. Copyright Wellcome History

Clinical aspects

Tropical sprue can have an insidious onset or can start acutely. The symptoms are those of chronic malabsorption. Generally it presents as a clinical triad of painful tongue, weight loss and persistent abdominal discomfort with diarrhoea. Patients are noticeably tired, both physically and mentally. Amenorrhoea is very common. There is loss of weight with muscle atrophy. Hypoalbuminaemia leads to oedema. Due to malabsorption of carbohydrates there is increased gas production in the intestines, with borborygma, a bloated feeling in the abdomen and intestinal cramps. The D-xylose absorption test is abnormal in more than 90% of cases. Fat malabsorption leads to steatorrhoea with more than 10 g of fat in the faeces. This occurs in 95% of patients. The stools are pale, very odorous and quite voluminous, up to 5 times the normal amount. Dehydration, hyponatraemia and hypokalaemia are very common. Calcium deficiency may lead to tetany with positive Trousseau's sign. Hypokalaemia leads to reduced tendon reflexes and U-waves on the electrocardiogram. There is usually a deficiency of vitamin B12, folic acid and sometimes also iron. Anaemia occurs and is typically macrocytic with megaloblastic bone marrow. In long-term cobalamin deficiency there may be peripheral neuritis and involvement of the spinal cord, chiefly of the dorsal columns (proprioception). The tongue is red and

painful. As well as glossitis there may be stomatitis with superficial erosions. Deficiencies in fat-soluble vitamins (A, D, E, K) lead to prolongation of the coagulation time and osteomalacia. Vitamin A deficiency is characterised by a dry, rough skin with follicular hyperplasia and Bitot's spots on the conjunctivae. In severe deficiency night-blindness and xerophthalmia may occur.

Differential diagnosis:

Tropical sprue is a pan-enteric inflammatory process often mistaken for gluten-sensitive enteropathy.

The differential diagnosis is that of chronic malabsorption. It includes persistent giardiasis, isosporiasis, strongyloidosis, intestinal capillariasis, gluten enteropathy (coeliac disease), chronic pancreatitis, intestinal tuberculosis, intestinal amyloidosis, Whipple's disease, the blind-loop syndrome, bacterial overgrowth, diverticula and jejunocolic fistulae. Crohn's disease is rare in developing regions.

Diagnosis

Tropical sprue should be suspected in anyone with megaloblastic anaemia and malabsorption who has lived in an endemic region or has visited these regions. Biopsy of the jejunum shows typical abnormalities. Intestinal villi become shorter and broader (blunting without flattening as in gluten enteropathy). In the intestinal wall there is an inflammatory infiltrate, chiefly consisting of lymphocytes, plasma cells and a few eosinophils. The enterocytes exhibit large vacuoles. Radiography of the small intestine shows non-specific changes. There is flocculation of the contrast material and segmentation of the barium column, distension of the lumen and thickening of the mucosa. The mucosal folds in the small intestine are irregular and thickened, which gives the impression of a stack of coins. In advanced cases, no mucosal folds at all can be seen. A flat mucosa is very unusual and should lead to suspicion of a different disease (e.g. gluten enteropathy).

Treatment

Treatment is based on tetracyclines 250 mg QDS or doxycycline 100 mg daily for 3 to 6 months. Folic acid supplements (5 to 10 mg daily) and multivitamins and if necessary iron should be added to the treatment. Response to treatment is generally swift with an initial improvement within three days. Further recovery takes place in the course of the following three months.