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Anti-Parietal Cell Antibody: Causative Agent Or Incidental Finding In A Patient Of Macrocytic Anemia With Ileal Tuberculosis: A Diagnostic Deilemma

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Abstract

Vitamin B12 deficiency in an adult male is caused by vegetarian diet, gastrointestinal surgery and pernicious anemia. Anti-parietal cell antibody, a screening tool for autoimmune atrophic gastritis and pernicious anemia can be found in 85-90% of patients with pernicious anemia. However, the presence is not diagnostic and can be found in several other autoimmune disease including 2.5 to 9% of healthy people. The presence of anti-parietal cell antibody causes symptomless chronic destruction of gastric mucosa and presents with severe neurological symptoms secondary to B12 deficiency. Similarly, chronic inflammation of ileal mucosa also known to cause b12 deficiency. India, endemic to tuberculosis is a known cause of macrocytic anemia.

Here we present the case of a young male consuming a mixed diet presented with easy fatiguability, fear of falling while walking, and slippage of slippers for the last 1month and diagnosed to have chronic distal, symmetrical, length-dependent large fibre sensory polyneuropathy secondary to b12 deficiency, and diagnosed to have ileal tuberculosis. However, the patient also had a positive anti parietal cell antibody which created a diagnostic dilemma in the present case.

Keywords: NIL

Introduction

Vitamin B12 deficiency is common in vegetarian group of people and people with gastric by pass surgery as well as Pernicious anemia .Pernicious anemia is defined as severe lack of intrinsic factor due to gastric atrophy .Pernicious anemia also has association with other autoimmune diseases like vitiligo, hyperparathyroidism, type 1 diabetes mellitus as well Addison's disease. Pernicious anemia shows higher life expectancy in women than men as men are predisposed to Gastric Cancer in later stages of life. Intestinal Tuberculosis is relatively common and is a major burden in India, as our patient experienced occasional diarrhoea and the endemicity of tuberculosis in India led to a suspicion of ileocaecal TB in our case.In our case 21 years young individual presented with selective vitamin B12 deficiency and had no any other predisposing association with other auto-immune conditions. This young non vegetarian male had both features of hematological as well neurological signs of vitamin B12 deficiency in just mere age of 21 . Also the association of ileocaecal TB in this patient explains the impaired of absorption vitamin B12 from terminal ileum, combined with pernicious anemia.

Case Presentation

A 21 year old unmarried Hindu male,consumer of non vegetarian diet belonging to poor socioeconomic status, who has no other significant past or family history. Presented with complaints of progressive easy fatigability for the last 15 months this is associated with palpitation on exertion and increased pallor over the nails and skin.This is also associated with self resolving yellowing of eyes, cheilosis and

hyperpigmentation of legs,hands and abdomen.He also complained of difficulty in walking for 5 months he described it as feeling of loss of control over his limbs during movement and fear of fall.This is also associated with difficulty in wearing slippers and slippage of slippers during walking with history of tingling and numbness in bilateral lower and upper limb in a bilateral symmetrical pattern. There is no history of loss of sensation to temperature or pain. Apart from this patient also complains of altered bowel habits - loose consistency of stools (2-3 episodes per day) which are non bloody ,non mucoid or non sticky. Associated with intermittent crampy abdominal pain in the periumbilical region.No history of fever ,nausea vomiting ,decreased appetite.

He had normal sensorium on presentation along with stable vitals.On examination he had brownish pale hair, Facial hair are sparse and inappropriately low for age. Severe pallor was there along with cheilosis, loss of tongue rugae, Hyperpigmentation on dorsum of hand and bilateral lower limb and abdomen.Atrophy of bilateral girdle muscle and proximal muscles and bilateral calf muscle. Normal power ,tone and reflexes in all the muscles. Impaired fine touch sensation in bilateral lower limb upto mid thigh, impaired proprioception, impaired vibration sensation in bilateral lower limb up to umbilicus. Stumping gait with a wide based stance. Sensory ataxia with and sensory paraesthesia suggestive of chronic distal ,symmetrical ,length dependent large fibre sensory polyneuropathy.

Investigations If Relevant

Hemoglobin-3.1;Total leukocyte count -2.28;

Differential leukocyte count(Neutrophil/Lymphocyte/Monocytes)84.5/12. 3/ 3.1;Platelet count;8.82

Serum Iron: 134.3, Serum Ferritin: 182.36 LDH: 1694.3, TIBC: 362.9, Transferrin saturation: 32.49%,

Peripheral blood smear : Macrocytic red cells showing anisopoikilocytosis, macroovalocytes, target cells and polychromatophils.

Vitamin D: 4.8ng/ml

Vitamin B12: <83 (211-911)pg/ ml ,**Serum Folic acid**: >20.0 ng / ml

St**ool routine and microbiological examination**: within normal limits

Stool occult blood : negative

Stool culture: No growth

Thyroid Function test: TSH-2.042,Ft4: 1.01, Ft3: 1.9

Viral markers: Non reactive

Tissue transglutaminase : 1.091(<4)

Anti parietal cell antibody (APCA) : Positive

CE-MRI BRAIN WITH WHOLE SPINE SCREENING: Ring enhancing lesion with scolex within in right frontal lobe-neurocysticercosis.

T2/STIR hyperintensity involving the bilateral dorsal columns of spinal cord, from cervico-medullary junction upto conus medullaris-likely subacute combined degeneration of spinal cord.

Ring enhancing lesions in muscle of neck left strap muscles and right sternocleidomastoid muscles-Myocysticercosis.

CECT ABDOMEN – hepatosplenomegaly with thickening of terminal ileum and ICJ with stricture in distal ileum – likely tubercular

Colonoscopy – Ulcerated ileo colic valve ,transverse ulcers terminal ileum ? Tuberculosis

ILEAL TISSUE FOR CBNAAT : MTB DETECTED

UGI ENDOSCOPY - GASTRIC ATROPHY

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Figure 1

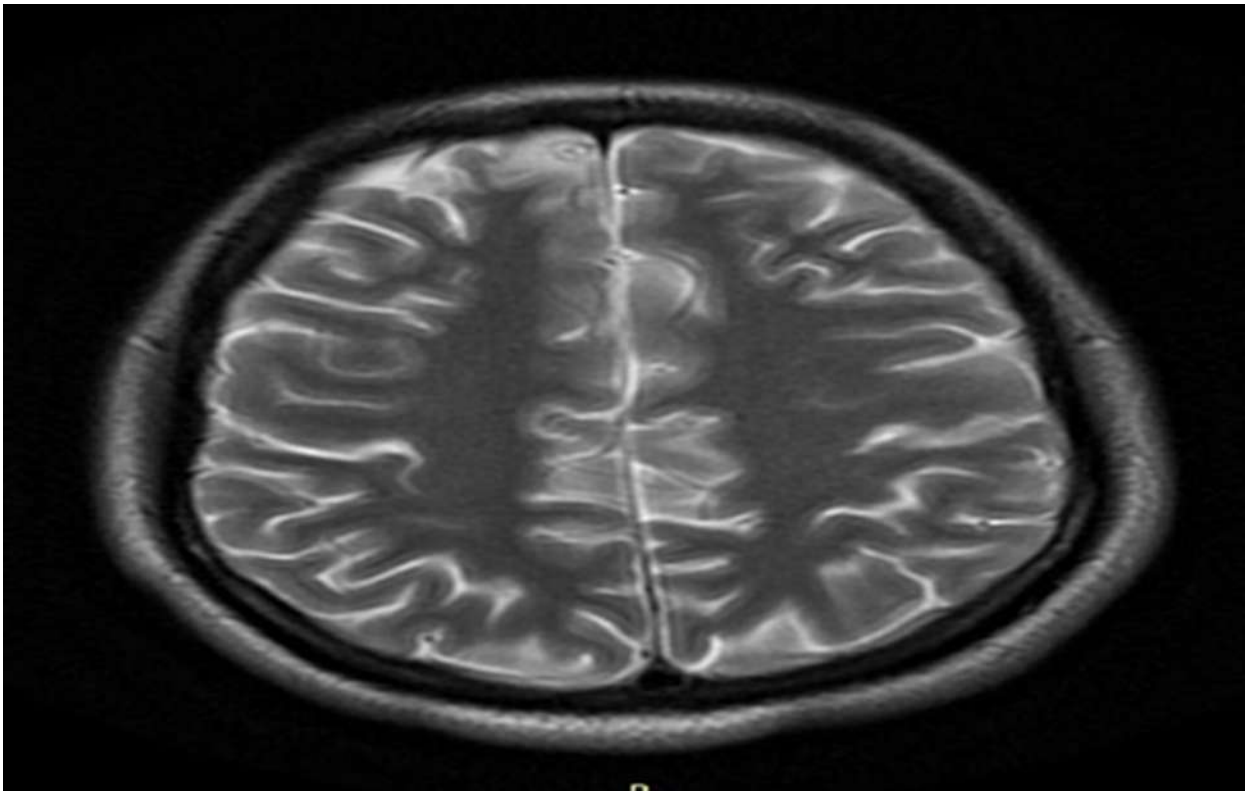

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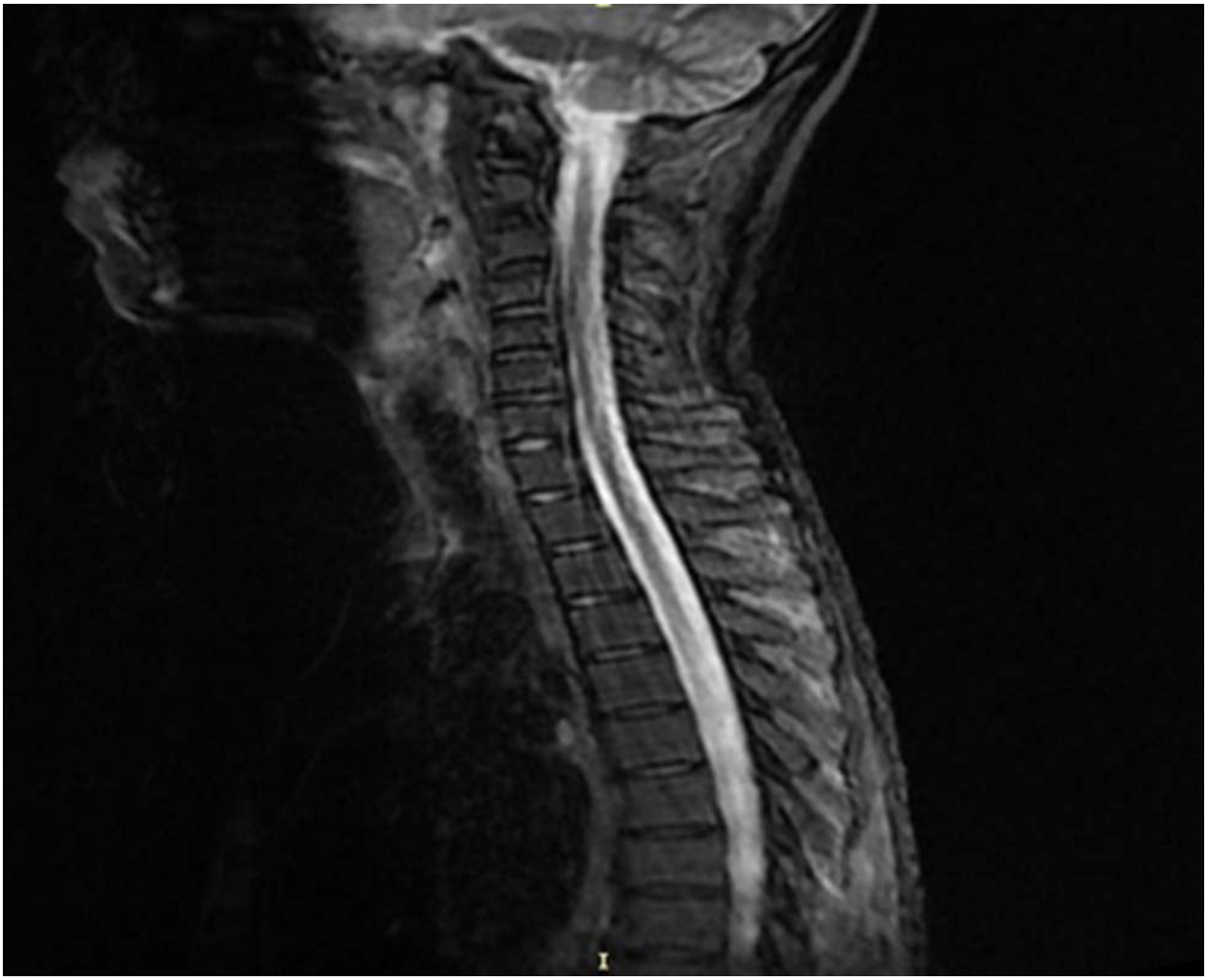
Figure 2



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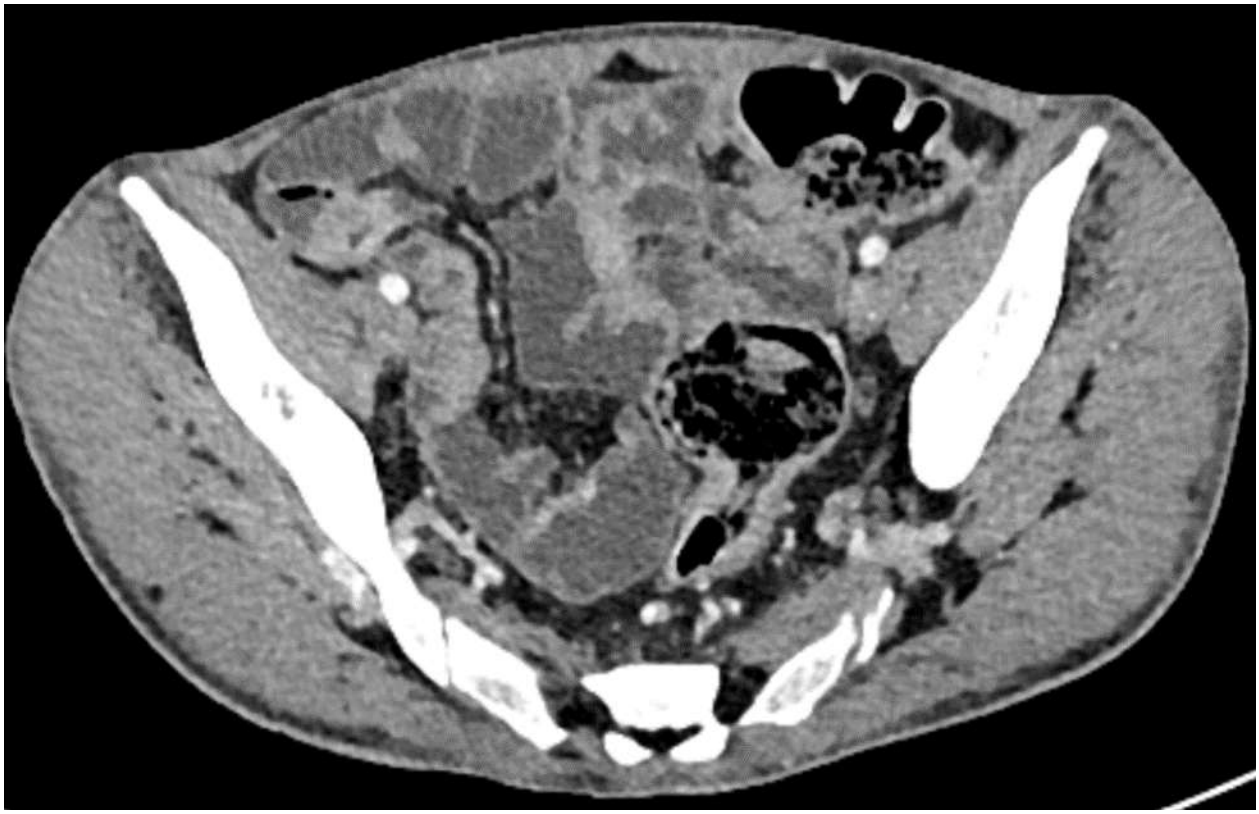

Differential Diagnosis If Relevant

Our approach and differentials were based on two major headings, on one side cause of pancytopenia was to be evaluated and the other hand cause of Malabsorption was to be ruled out . We started with base line investigations and patient was found to have pancytopenia , and the history of loose stools from around 15 minutes suggested chronic diarrhoea. Stool routine,microscopy and cultures were negative for pathogens,ova and parasites. Thus Malabsorption syndromes were to be ruled out.

TREATMENT If relevant

1. TAB.REDLAR XT 1 TAB PO OD

2.Cap DRIVE D3 60 K IU PO ONCE A WEEK

3.INJ ELDERVIT 400 Ml IV OD (INITIALLY)

4.INJ VITCOFOL 2 Ml IM ONCE A WEEK

5.TAB PREDNSILONE60 Mg PO OD FOR 14 DAYS

6.TAB ALBENDAZOLE 400 MG PO OD

7.TAB SHELCAL 500 Mg PO BD

8.TAB CALCITRIOL 0.25 MG PO OD

9.TAB HRZE 3 TAB PO OD –FOR 2 MONTHS (AS PER CATEGORY I DOTS)

10.TAB PYRIDOXINE 40MG PO OD - CONTINUE TILL ATT

11.INJ VITCOFOL 2ML IM ONCE WEEKLY – FOR 4 WEEKS (NEXT DOSE ON 01/08/2, /08/08/21 AND 15/08/21) THEN ONCE MONTHLY –CONTINUE

Outcome And Follow-Up

Our patient was started on daily intramuscular B12 supplementation(methylcobalamine) and was evaluated in terms of hematological indices as reticulocyte index as well clinical parameters as improvement in sensory symptoms as well improvement in ataxia, after significant improvement patient was advised for IM B12 supplementation once every third month for the rest of life. Also patient was started on ATT for ileocaecal tuberculosis as CAT -1 ATT for 6 months and OPD follow for monitoring of LFT was advised monthly. For Neurocysticercosis patient was given steriods to

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prevent seizures for 3 days and started on Albendazole which was continued for 21 days. Patient is being followed up monthly in OPD basis and lab parameters as well neurological symptoms have improved . Our patient also has started on new job as he is feeling much better and fear for falling while walking has significantly improved.

Discussion

Pernicious anemia is third leading cause of megaloblastic anemia. Megaloblastic anemia is a hematologic manifestation of chronic atrophic gastritis which usually affects the corpus, fundus and antrum of the stomach that denudes the gastric mucosa of gastric parietal cells. The disease is common in Northern European population rather than Asian population showing incidence of men: women ratio of 1:1.6. The median age group of onset is 70-80 years with only 10% of population with less than 40 years of age ,even the younger onset of disease is common in common in black population as well Latin Americans . The gastritis arises from activation of pathologic Th1 CD4 T cells to gastric H/K ATPase that is present on gastric mucosal secretory membranes. The onset of autoimmune gastritis is marked by circulating parietal cell antibody to gastric H/K ATPase. Asymptomatic chronic gastritis usually precedes Gastric atrophy by 10-20 years. Gastric parietal cells have two major essential biologics: intrinsic factor and HCl acid. Pernicious anemia leads to antibody directed against parietal cells as well as to intrinsic factor which causes loss and neutralizing intrinsic factor antibody that impairs cobalamin absorption. Acid loss leads to iron deficiency anemia that precedes cobalamin-deficient pernicious anemia by 20 years. Laboratory diagnosis is based on parietal cell antibody with or without intrinsic factor antibody, cobalamin-deficient megaloblastic anemia and elevated serum gastrin from loss of acid secretion.Intrinsic factor antibodies is the blocking type against the B12 binding site present in 70% of patients, and the other is antibodies to parietal cells present in 90% of patients, but it is less specific. Autoimmune gastritis is associated with autoimmune thyroiditis and type 1 diabetes mellitus, polyglandular autoimmune syndromes.

India is the country which has highest burden of Tuberculosis in the whole world, India has 193 per 1 lakh population of active diagnosed tuberculosis according to data of WHO of 2019. Abdominal Tuberculosis is relatively uncommon extrapulmonary tuberculosis, yet is a common tuberculosis considering burden of TB in India. Over all it constitutes about 5% of all cases in the world. Intestinal tuberculosis consists of two types ulcerative and ulcerohypertrophic . Ulcerative and stricturous type are usually observed in small intestine and ulcerohypertrophic is usually observed in colon and ileocaecal area. Common symptoms of intestinal TB includes, abdominal pain, early satiety, postprandial fullness, malabsorption, diarrhea, hematochezia and constipation alongside with fever, weight loss and classical symptoms of Tuberculosis.

Once the treatment is started for B12 deficiency the earliest sign of treatment response is an increase in reticulocyte count, usually within three days of treatment. Following changes in the decrease of biochemical markers such as MMA and plasma homocysteine levels have been observed in the first five days of treatment. Normalization of serum cobalamin usually occurs following two weeks of therapy. The macrocytosis correction takes place during the first month of treatment. Neurological signs and symptoms usually become symptomatic after the hematological signs and symptoms with no defined time line, as we can observe in our case neurological signs and symptoms have almost followed hematological symptoms and the resolution of gait ataxia and fear of falling has resolved in almost 10 days of hospitalization, receiving B12 therapy. This case helps us to understand that despite ileocecal tuberculosis is common in India , other causes for Vitamin B12 deficiency should be looked for.

Learning Points/Take Home Messages 3-5 Bullet Points

1. Detailed examination of the patient can give clue to the underlying disease/deficiency as in our case signs of anemia and neurological signs gave clue to B12 deficiency even in patient with non-vegetarian diet.
2. Diseases endemic and common in our region must be ruled in/out as they have impact on the clinical outcome and severity of the diseases they present with.
3. Complete diagnostic workup may lead to diagnostic dilemma as co-existing diseases

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superimpose but usually define the cause of severity of the disease and symptoms.

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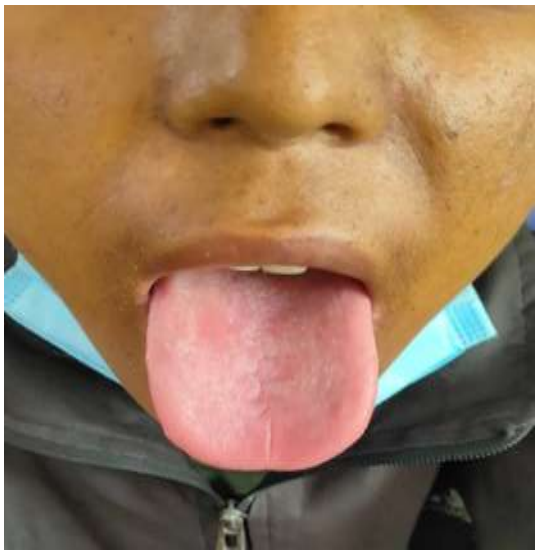
Figure 1



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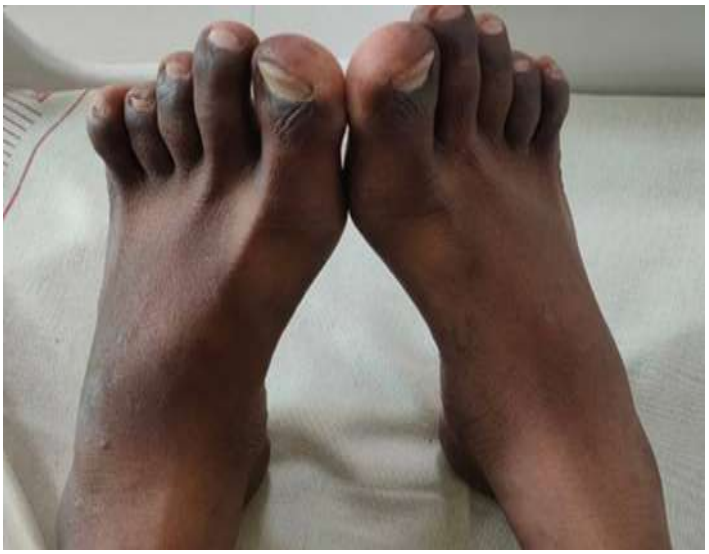

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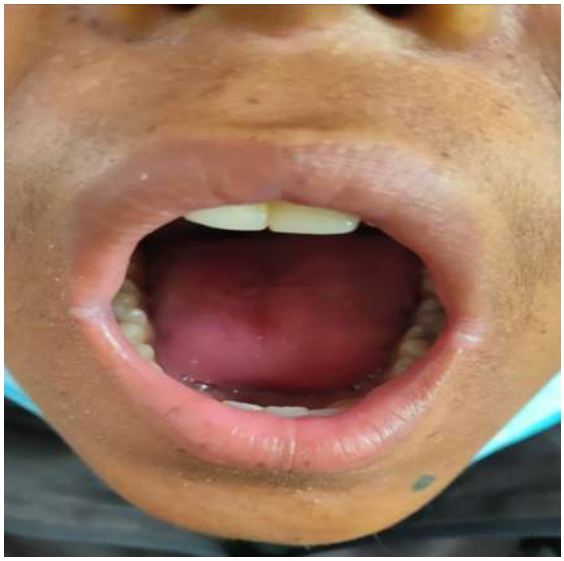

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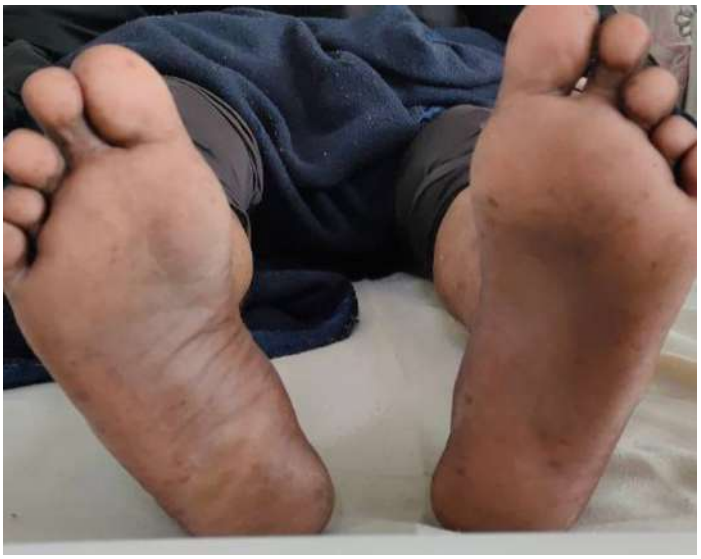

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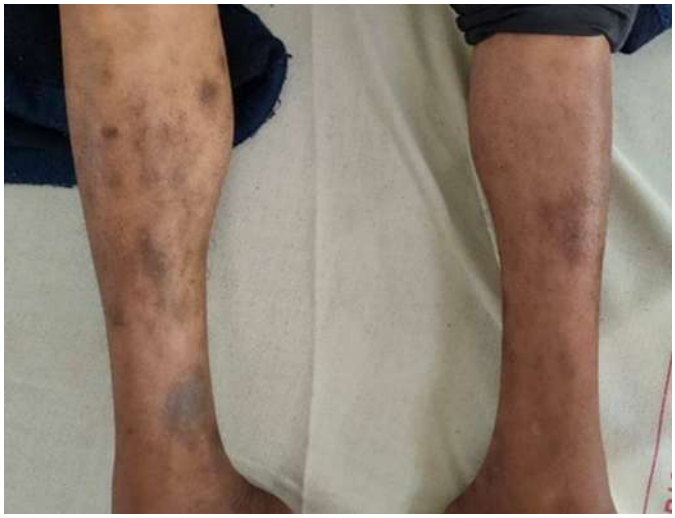

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