IMAGE FOR EDUCATION

Bluish Discoloration of Periodontal Tissue

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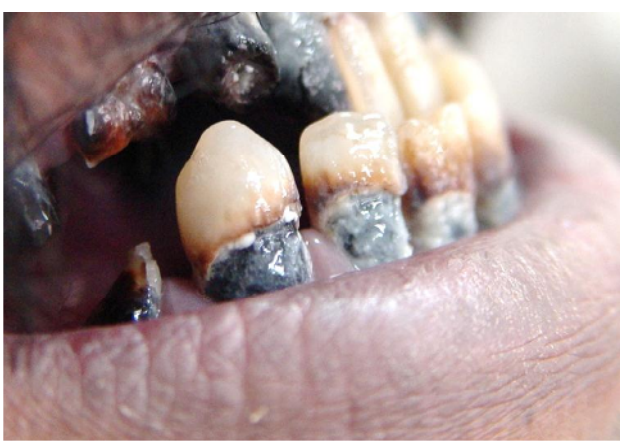
QUESTION

Case: A 31 year old male opioid addict referred to Addiction Rehabilitation Center, an affiliated clinic to Addiction Research Centre for opioid abstinence therapy. He was skinny in a poor condition. He complained of impaired memory, temporary daily agitation, severe weakness, mild paresthesia, impotence and metallic taste. He was also suffered from importunate spastic abdominal cramps and colic pains which have been repeated several times during the last 6 months.

On physical examination, his blood pressure was slightly increased (135/80). Stomach was soft without any tenderness and hepatomegaly. Force of proximal and distal muscles were decreased and he was unable to stand up without any help after kneeling. Oral examination showed yellowish rotten teeth with a diffuse dark bluish discoloration of cervical tooth (Figure 1). Dermatologic inspection revealed no other pigmentation on the other parts of the body.

What are the differential diagnosis of and the proper approach to this patient?



ANSWER

Differential diagnosis: The main complications of the patient were recurrent spastic abdominal cramps and colic pains, fatigue and severe muscle weakness and dark bluish discoloration of periodontal tissue. The leading differential diagnoses could accordingly include (1) lead poisoning (2) hypoadrenocorticism (Addison's disease) (3) AIDS (Kaposi sarcoma) (4) bismuth stomatitis and (5) smoker's melanosis.

A case with severe fatigue, muscle weakness, abdominal pain, and abnormal discolorations is suggestive of Addison's disease. However, such cases are hypotensive with diffuse pigmentation on the other parts of the body rather than oral cavity (1,2). Deposition of heavy metals (lead, bismuth, mercury, silver, gold, etc.) is tended to create discolorations in oral cavity due to reaction between sulfur ions released by oral bacteria with circulating metal molecules (3). There are other causes of bluish discolorations in oral cavity, which are summarized in table 1 (2,4-18).

Approach: As the first step to diagnose, complete blood count, peripheral blood smear and blood lead level (BLL) assays are recommended.

Laboratory tests of this patient revealed hypochromic microcytic anemia (Hb <10 mg/dL), basophilic stippling on blood smear and a high blood lead level (840 µg/dL). This is consistent with lead poisoning. To assess the level of target organ damages, urinalysis, routine serum biochemistries and liver function tests (LFT) are required. Radiographic imaging helps to substantiate the diagnosis in doubtful cases by showing increased metaphyseal density of long bones (lead lines) (11).

In this patient, a marginal increase in serum creatinine (1.4 mg/dL) was detected. LFT and serum biochemistries were normal

Treatment: Primarily, the exposure should be decreased. The mainstay of treatment of lead poisoning is chelation therapy. However, it is only indicated for symptomatic patients with BLL exceeding 70 µg/dL in adults and 45 µg/dL in

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children (10,11). For mild symptoms with BLL between 70 and 100 µg/dL in adults and 45-70 µg/dL in children, succimer (350 mg/m^2 tid for 5 days then bid for 14 days) is the drug of choice which is administered orally. Moderate to severe cases (BLL > 100 µg/dL, encephalopathy, abdominal colic, severe arthralgia or myalgia, target organ damage) should be admitted to receive 75 mg/m^2 dimercaprol (British anti-lewisite; BAL) intramuscularly every 4 hours for 5 days and 1000-1500 mg/m^2 Edetate Calcium Disodium (CaNa2EDTA) via daily infusion. In case of opioid adulteration, abstinence treatment with methadone maintenance is also indicated (9,10). Supportive treatments including intravenous fluids and hemodialysis in case of renal failure may also be necessary.

Etiology: Chronic and acute lead poisoning could be the result of occupational or environmental lead exposure or rarely ingestion of lead contaminated substances (3,11). However, some cases might be the victims of lead adulteration which is a new dilemma (12-18).

Outcome: The patient received 450 mg/m^2/d BAL and 1500 mg/m^2/d $CaNa_2EDTA$ in divided doses for five days. He was well in further follow-ups, though he complained occasional mild abdominal discomforts.

Limitation: In classic discoloration of oral cavity due to lead toxicity, marginal gingiva is described to be involved (Burton's sign) (11), which in this case has not been observed.

Table 1. Differential diagnosis of bluish discoloration of periodontal tissue

Disease	Etiology	Clinical Manifestation
Heavy metal deposition		
Lead Poisoning	- Environmental and occupational exposure to lead contaminated, paints fumes, etc. - Long term abuse of adulterated opioids and marijuana - Drinking from leaden amphora containing wine (ancient Romans) known as colica pictonum - Drinking from cider produced through a lead lined press known as Devonshire colic	Diffuse dark bluish discoloration of periodontal tissue, spastic abdominal pain, fatigue, muscle weakness, hypertension, anemia, peripheral neuropathy, renal failure, encephalopathy (rare)
Bismuth stomatitis	Long term treatment with bismuth compounds	Diffuse dark bluish discoloration on gingival sulcus, buccal mucosa and tongue. Metallic taste and burning sensation in the mouth may also be present.
Argyria	Constant Inhalation of highly concentrated silver containing fumes.	Diffuse blue-gray discolorations on the skin and, to a lesser degree, on the mucosal membranes
Amalgam tattoo	Embedding of mercury/silver containing material into adjacent oral mucosal membrane	Single or multiple localized blue gray discolorations of variable dimensions in gingiva and alveolar mucosa. Floor of the mouth and the buccal mucosa may also be involved.
Chrysiasis	Long term treatment with gold salts (usually for rheumatoid arthritis)	Diffuse dark bluish or faint purple gingival discoloration
Addison's disease	Increased adrenocorticotropic hormone production induces melanocyte-stimulating hormone	Multiple localized pigmentations of skin and mucosa of all over the body, weakness, nausea and vomiting, abdominal pain, constipation or diarrhea, weight loss and hypotension.
Kaposi sarcoma	Multifocal vascular malignancy	Multiple localized brown to purple lesions of hard palate, gingiva and tongue
Smoker's melanosis	Increased production of melanin due to long term smoking	Diffuse brown–black discolorations of gingiva, buccal mucosa , lateral sides of tongue, palate and floor of mouth which are spontaneously resolvable after cessation of smoking
Melanocytic nevus	Accumulation of nevus cells in the basal epithelial layers and/or connective tissue	Single or multiple localized dark brown, gray, blue, or black of hard palate, buccal mucosa, lip, gingiva, labial mucosa, soft palate, retromolar pad and tongue
Oral melanoma	Proliferation of malignant melanocytes along the junction of the epithelial and connective tissues and within connective tissue	Single or multiple localized dark brown, gray, blue, or black of hard palate, buccal mucosa, lip, gingiva, labial mucosa, soft palate, retromolar pad and tongue. The lesions are rapidly enlarging associated with ulceration, bleeding, pain and bone destruction
Minocycline induced pigmentation	Long-term treatment with minocycline (usually for refractory acne vulgaris)	Diffuse gray bluish discolorations of the alveolar bone, which can be seen through the thin overlying oral mucosa (especially the maxillary anterior alveolar mucosa)
Physiologic (Racial) Pigmentation	Increased melanocytic activity in African, Asian and Mediterranean populations	Diffuse discoloration of marginal gingiva. Pigmentation of the buccal mucosa, hard palate, lips and tongue may also be seen
Scurvy	Reduction in oxygenated hemoglobin due to vitamin C deficiency	Diffuse bluish red appearance of gingiva, weakness, lethargy, bone pain, myalgia, easy bruising, petechiae, poor wound healing and emotional changes

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