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# Burton's Line

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A 39-year-old man with diabetes who had been taking ayurvedic medication for 2 years presented with loss of appetite of 2 months duration with vomiting and severe colicky abdominal pain of 1 week duration. On examination, pallor was present. Cardiovascular, respiratory, and abdominal examination results were within normal limits.

## CLINICAL SUMMARY

On investigation, the patient's hemoglobin level was 6.1 g, peripheral blood smear showed microcytic hypochromic anemia with basophilic stippling (**Figure 1**), and reticulocyte count was 10%. Stool examination, upper gastrointestinal endoscopy, ultrasound scan of abdomen, hemoglobin electrophoresis, and serum iron, serum ferritin, and total iron-binding capacity levels were normal. Urine porphobilinogen was negative. Fasting blood glucose was 70 mg/dL, and postprandial blood glucose was 250 mg/dL. Urine ketone bodies were negative.

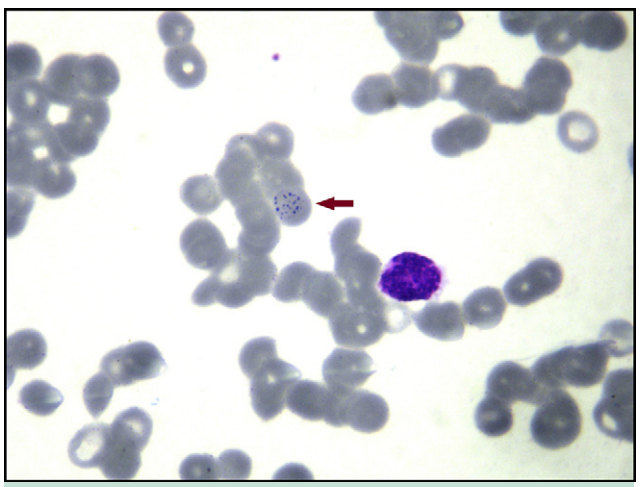

**Figure 1** Basophilic stippling.

**Figure 2** Burton's line.

On examination of the oral cavity, a purple blue line (Burtonian line) was present in the gingiva (**Figures 2** and **3**). Blood lead level estimation was done by atomic absorption and found to be 77.17 $\mu$g/dL (reference range $<$ 10 $\mu$g/dL). Treatment was started with D-penicillamine 250 mg, 3 times per day. After 1 month of treatment, the patient showed symptomatic improvement in his appetite and abdominal pain. D-penicillamine treatment was continued until the patient's blood lead levels were less than 10 $\mu$g/dL, which took 6 months from the day of initiation of treatment. The ayurvedic medication was subjected to chemical analysis for lead content, and it contained 16,307.27 $\pm$ 447 $\mu$g lead per capsule.

## DISCUSSION

Ayurveda is a traditional form of medicine, and ayurvedic medications can contain herbs, minerals, heavy metals, or animal products made in standardized or nonstandardized formulations.[1-3] Clinical manifestations of lead toxicity in-

See another article on this topic by Desai and Staszewski in the current issue.

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**Figure 3** Burton's line.

clude symptoms referable to the central nervous system, peripheral nervous system, hematopoietic system, renal system, and gastrointestinal system. The peripheral blood smear may show evidence of hemolysis, normochromic or hypochromic microcytic anemia, and basophilic stippling of red blood corpuscles. The reticulocyte count may be elevated because of increased red blood corpuscle destruction. The reaction of circulating lead with sulfur ions released by oral microbial activity may cause the deposition of lead sulfide at the interface of the teeth and gums, referred to as "Burton's line." The exact pathogenic mechanism of lead-induced abdominal colic is unknown. Several cases of acute abdominal colic due to lead toxicity have been reported.[4,5]

The whole blood lead level is the most sensitive and specific test in the evaluation of lead toxicity. In adults, a level as low as 20 $\mu$g/dL is associated with headache, irritability, and difficulty performing fine tasks. Levels of 60 to 80 $\mu$g/dL are associated with anemia, renal insufficiency, abdominal colic, and constipation. In adults, encephalopathy is associated with levels greater than 100 $\mu$g/dL.

Treatment of lead toxicity includes removal of the patient from the source of exposure, optimization of the nutritional status of the patient, and chelation therapy. Chelation functions by binding with lead and forming a water-soluble complex that is excreted in urine. The efficacy of treatment is monitored by post-chelation decreases in blood lead concentration. The agents recommended for chelation in lead poisoning include dimercaprol (British anti-Lewisite), calcium sodium ethylenediaminetetraacetic acid, succimer, and D-penicillamine.

## CONCLUSIONS

Lead is a multiorgan toxin. Lead poisoning should be considered in the differential diagnosis of abdominal pain with anemia. A complete history including ayurvedic drug intake, general physical examination for the presence of a Burtonian line, and examination of the peripheral blood smear for basophilic stippling will aid in the diagnosis of lead poisoning in a patient.

## References

1. Schilling U, Mück R, Heidemann E. Lead poisoning after ingestion of ayurvedic drugs. *Med Klin (Munich)*. 2004;99:476-480.
2. Kales SN, Christophi CA, Saper RB. Hematopoietic toxicity from lead containing Ayurvedic medications. *Med Sci Monit*. 2007;13:CR295-298.
3. Prpić-Majić D, Pizent A, Jurasović J, Pongracić J, Restek-Samarzija N. Lead poisoning associated with the use of Ayurvedic metal-mineral tonics. *J Toxicol Clin Toxicol*. 1996;34:417-423.
4. Sood A, Midha V, Sood N. Pain in abdomen—do not forget lead poisoning. *Indian J Gastroenterol*. 2002;21:225-226.
5. Frith D, Yeung K, Thrush S, Hunt BJ, Hubbard JG. Lead poisoning—a differential diagnosis for abdominal pain. *Lancet*. 2005;366:2146.