

Burton's Line in Lead Poisoning

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Key Words

Lead poisoning · Plumbism · Burton's line

Abstract

Lead poisoning in both its acute and chronic forms has been recognised since the second century BCE. Lead colic, anaemia, renal tubulopathies and motor neuropathies are well recognised. This paper sketches the early history and remembers the important contribution of Henry Burton, who described the gums to be bordered by a narrow leaden-blue line, about the one-twentieth part of an inch in width, whilst the substance of the gum apparently retained its ordinary colour and condition. The sign though inconstant, is still a valuable clinical clue.

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In the second century BCE, the Greek botanist Nikander described the paralysis and colic caused by lead, and 3 centuries later the Greek physician Dioscorides observed that after exposure to lead, 'the mind gives way' [1]. The Romans were often similarly afflicted with colica pictonum caused by leaden amphora containing wine, until in 1616 Francois Citois described Poitou colic (colica pictonum) in detail, culminating in the recognition of the condition and its causes [2]. In Baden-Württemberg, a physician noticed that while wine drinkers developed colic, monks who did not drink wine were unaffected; a white oxide of lead added to sweeten the wine was responsible [3]. 'Devonshire colic' was the name used in the 18th century for plumbism caused by cider contaminated by lead lined cider presses. Voltaire's physician, Théodore

Tronchin gave rich account of industrial and iatrogenic lead poisoning [4], before the official *Inquiry* of Sir George Baker in 1767. In 1786, Benjamin Franklin, who had worked as a printer in his youth and therefore familiar with lead poisoning, was surprised that it still occurred:

'... the Opinion of this mischievous Effort from Lead is at least above Sixty Years old, and you will observe how long a useful Truth may be known and exist before it is generally receiv'd and practis'd on' [5].

Burton's Line

Burton's line is a blue-purplish line on the gums seen in lead poisoning. It is caused by a reaction between circulating lead with sulphur ions released by oral bacterial activity, which deposits lead sulphide at the junction of the teeth and gums.

Henry Burton whilst investigating the unproven benefits of medicinal lead in pulmonary and other disorders described this clinical sign in 1840 [6]. He recorded:

'... Dr Christian in 1829 & 1836 described the saliva is increased in quantity and bluish in colour ... In a total number of fifty patients who were examined whilst under the influence of lead, a peculiar discolouration was observed on their gums, which I could not discern on the gums of several hundred patients who were not under the influence of lead.

I believe the sign will enable physicians to establish ... a precise diagnosis in derangements of health depending on the unsuspected presence of lead; and also to obviate ... the inflictions of lead colic, during treatment of other diseases by saturnine [lead containing] preparations ... The edges of the gums attached to the necks of two or more teeth of either jaw, were distinctly bordered by a narrow leaden-blue line, about the one-twentieth part of an

inch in width, whilst the substance of the gum apparently retained its ordinary colour and condition ... no tumefaction, softening or tenderness about them; neither any peculiar foetor in the breath; nor increased salivary discharge ...'

He did not observe salivation in 36 cases of lead colic but noticed the neglect of dental hygiene independent of constitutional disease arising from lead. It differed, he said from scorbutus [scurvy] and mercurial poisoning.

A Londoner by birth, Henry Burton attended Tonbridge school and read medicine at Caius College, Cambridge, qualifying in 1826. He practised as Assistant Physician to St Thomas's hospital in 1828, and later was senior physician. He became a Censor at the Royal College of Physicians, London, in 1838.

Lead poisoning in both its acute and chronic forms has gradually declined with increasing surveillance of industrial and domestic exposure. But it has not disappeared [7]. The main route in industrial exposure is the respiratory tract [8, 9]. Chronic poisoning less commonly results from ingestion. Deteriorated lead-based paint in old homes and high levels of lead-contaminated house dust are the commonest sources. Lead's affinity for sulphhydryl groups impairs multiple enzyme systems, notably intracellular calcium channels. This causes defective haem synthesis, proximal renal tubular and osteoblast dysfunction. The Environmental Protection Agency's 'action level' for lead is 15 ppb. Ninety-five percent is stored in bone, 4% in brain, liver and kidneys, 1% in blood.

In children, who absorb lead more readily than adults, classical features are irritability, loss of appetite, weight loss, sluggishness, behaviour and learning difficulties, abdominal pain, vomiting, constipation, anaemia and renal failure.

In adults the main features are: pain, numbness or tingling of the extremities, muscular weakness, headache,

abdominal pain, memory loss, anaemia and renal failure, male reproductive impairment.

The symptoms are non-specific [10], but three main syndromes of chronic poisoning are described: (1) the gastrointestinal, (2) neuromuscular and (3) neurological types. The cerebral syndrome is most often seen in childhood. The blue line on the gums (Burton's sign) is a valuable but variable clue to diagnosis.

Neurological signs of acute poisoning typically are: paraesthesiae, pain, muscle weakness, encephalopathy (rare) with headache, convulsions, delirium, and coma.

Chronic poisoning is insidious with fatigue, sleep disturbance, headache, irritability, slurred speech, stupor, ataxia, convulsions, anaemia and renal failure. Neurodegenerative changes shown on MRI may complicate chronic plumbism [11]. Hyperkinetic and aggressive behaviour disorders occur and in children, refusal to play and learning regression [12]. In the peripheral nervous system, paraesthesiae, pain, muscle weakness occur and in chronic poisoning, similar symptoms, and occasional lead palsy with paralysis of the radial nerve with 'wrist drop' are characteristic. Sensory nerves are not clinically affected.

Laboratory findings include punctate basophilia, variable anaemia and azotaemia. L-line X-ray fluorescence technique is a predictor of lead toxicity and measures cortical bone lead content. The blood level of lead is raised: an 'unsafe level' is 10 µg/dl (0.5 µmol/l) or higher.

If blood levels exceed 45 µg/dl of blood, chelation treatment is with ethylenediaminetetraacetic acid intravenously, which may be combined with the dimercaprol (BAL).

Burton's line remains a useful if not wholly reliable diagnostic clue in a disease which should have disappeared.

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