Case report

An atypical chemical burn

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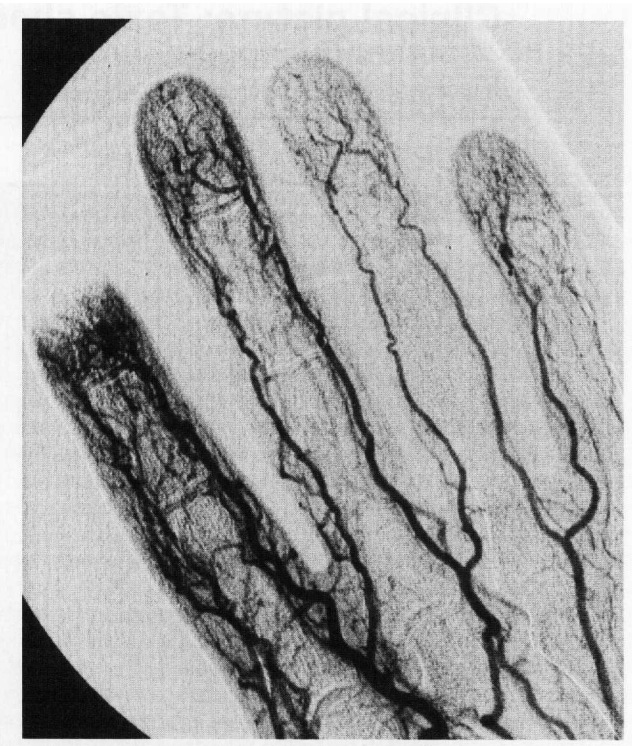
A 48-year-old male painter visited the emergency room, in May, 1999, complaining of throbbing pain in the tip of his right index finger. He said that he always wore gloves while working, had had no previous trauma, smoked 25 cigarettes per day and used no medication. On examination, the fingertip had a blanched appearance, no capillary refill, and intact sensibility. We thought that he had an arterial embolus and started acetylsalicylic acid. He returned the next day complaining of increased pain. The fingertip had become black and necrotic, and the proximal finger was swollen and erythematous. Femoral angiography showed hyperaemia just proximal to the necrotic fingertip with no visible obstruction or embolus (figure). The patient then recalled that the previous day he had used a new cleaning fluid at his job. We called his employer who informed us that this fluid contained hydrofluoric acid. We applied calcium gluconate gel to the finger, repeating the application every 4 h for 3 days, until the pain ceased. His finger healed cleanly and he had no complaints when last seen in October, 2000.



Brachial arteriography showing hyperaemia of the index finger with an ischaemic tip

Hydrofluoric acid is used in a variety of industries (production of plastics, metal cleaning, electronics and chip manufacturing) and household products (rust removers, aluminum brighteners, heavy duty cleansers).[2] The incidence of HF burns is surprisingly low, no more than 1000 cases a year are reported in the United States.[2] It causes severe burns and systemic effects, even when superficially unimpressive. The detrimental effect of hydrofluoric acid will continue for days subcutaneously if not treated adequately.[2] Tissue damage is caused by two separate mechanisms. First, the hydrogen ion causes a superficial burn. Second, the fluoride ion penetrates deeper tissues, causing liquefaction necrosis of soft tissue, bone decalcification, and intense pain. Onset of symptoms depends on the concentration of the spilled hydrofluric acid. Concentrations over 50% cause immediate pain and typical blanching of the involved skin. A delay in symptoms of 1–6 h is seen with concentrations between 20–50%; concentrations of less than 20% can give a delay up to 24 h. In these cases the skin shows a slight erythema or no injury at all.[1]

The characteristic symptoms of a hydrofluoric acid burn were described for the first time in 1809 by Thenard and Gay-Lussac.[3] Typical symptoms are blanching of the affected skin surrounded by erythema and excruciating pain (out of proportion to the burn). Destruction of deeper tissues is indicated by greyish discolourment of the skin, blistering and subcutaneous deposits of insoluble salts, frank necrosis and deep ulceration, with decalcification of underlying bone.[1,2] Treatment starts with removal of stained clothes and copious irrigation with water. The next step is generous application of calcium gluconate gel (2·5%), which must be repeated at least every 4 h. Relief of pain indicates successful treatment and should be continued until the patient is pain-free for at least 45 min. Application of calcium gluconate gel is the most simple and least invasive treatment for hydrofluoric acid burns. However, if the pain does not diminish or worsens during calcium gluconate gel therapy, alternatives are local infiltration, intra-arterial calcium gluconate perfusion, or intravenous regional perfusion with a calcium gluconate solution,[1,4] and surgical excision in case of frank necrosis or blisters.[5] Systemic intoxication can occur with an affected area of 160 cm^2. Changes in levels of serum calcium, magnesium and fluoride levels cause a prolonged Q-T interval, severe abdominal pain, nausea, and vomiting. A missed diagnosis results in symptomatic progression, leading to amputation or even death.

The Lancet 2001; **358:** 1510

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