For their systematic review of arsenic and cardiovascular disease, Navas-Acien et al. (1) thoroughly searched and rigorously reviewed reports from 1966 through April 2005. This methodology, exemplary in a modern context, could not have uncovered citations pertinent to a documented episode of arsenic-mediated cardiotoxicity that happened in the United Kingdom long before 1966. That epidemic, here called “arsenic-beer drinkers’ disease,” warrants attention for both historical interest and possible insights into the pathogenesis of cardiac myocyte disorders.

In 1900, in and near Manchester, England, an epidemic occurred of a multisystem disease with over 6,000 cases with more than 70 deaths. With replicated testing, the proved culprit was accidental contamination of beer by arsenic (2). The syndrome included the usual signs and symptoms of arsenic poisoning, with skin, nervous system, and gastrointestinal manifestations. Unusual in arsenic poisoning, but especially prominent in this epidemic, were cardiovascular aspects. In a superb clinical description, Dr. Ernest Reynolds wrote the following: 1) “cases were associated with so much heart failure and so little pigmentation that they were diagnosed as beri-beri...”; 2) “so great has been the cardiac muscle failure that—undoubtedly the principal cause of death has been cardiac failure”; and 3) “at the post-mortem examinations the only prominent signs were the interstitial nephritis and the dilated flabby heart...” (3, p. 169).

For several years, there were lively relevant entries in The Lancet (3–7). It was determined that the source of the arsenic was contaminated sulfuric acid used to treat cane sugar. The result in the affected beer was 2–4 parts per million (approximately 0.2–0.4 mg/liter), an amount not believed, by itself, likely to cause serious toxicity (4, 7). For example, Gowers reflected that 10 times the amount of arsenic involved was prescribed for epilepsy over substantial periods of time without toxicity; thus, “the amount of arsenic...was not sufficient to explain the poisoning” (4, p. 99).

It was observed that some seemed to have a “peculiar idiosyncrasy” (5, p. 218), and that “many persons became ill who drank less beer than others who were not affected” (6, p. 673). The governmental investigative body (Royal Commission Appointed to Inquire into Arsenical Poisoning) suggested that “alcohol predisposed people to arsenic poisoning” (2, p. 673). A more probable and more modern view would be the converse, that arsenic predisposed susceptible persons to alcoholic cardiomyopathy.

Recognized 65 years after the arsenic-beer episode, a condition known as “Quebec-beer drinkers’ cardiomyopathy” (8) was eerily similar in some respects. Heart failure epidemics among chronically heavy beer drinkers in Omaha, Nebraska, Minneapolis, Minnesota, and Quebec, Canada (9), included rapidly developing symptoms. Deaths were common, but those who recovered did well despite return, by many, to previous beer habits. Quebec investigators (8) tracked down the etiology, which proved to be the addition of small amounts of cobalt chloride by certain breweries to improve the foaming qualities of beer. Removal of the cobalt additive ended the epidemic in all locations; biochemical mechanisms were not established. As with the arsenic-beer episode, there must have been other factors, since most exposed persons did not develop the condition.

Viewing, in 1969, the arsenic and cobalt episodes, Alexander commented: “This is the second known metal induced cardiotoxic syndrome produced by contaminated beer” (9, p. 413). Cobalt or arsenic, substantial amounts of alcohol, and other predisposing factors were needed to produce these syndromes. It is unclear whether they represented synergistic or additive myocardial toxicity by the respective contaminants plus alcohol. What is clear is the reminder that most clinical entities are pluricausal in nature.

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REFERENCES

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