Oral Abstracts

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FROM BEER TO HIPS - DIAGNOSIS AND TREATMENT OF COBALT TOXICITY

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Trace amounts of cobalt ions are essential for human life in that they are required for the synthesis of vitamin B12 (cyanocobalamin), while larger amounts have the potential for significant toxicity. Although the use of cobalt salts for intended self harm is uncommon, an understanding of cobalt toxicity can be gained by studying its early use as a hematopoietic, and the unintended consequences of the addition of cobalt to beer and its use in metal-on-metal hip implants. The major organ systems involved are the bone marrow, nervous, cardiovascular, and endocrine systems.

The first experiences with cobalt salts came from their medicinal use as a hematopoietic with one popular pharmaceutical known as Roncovite. Chronicled in 1949 in two works in the New England Journal of Medicine, toxic gastrointestinal effects were already recognized, but therapy was continued because cobalt was felt to be less toxic than iron.(1,2) By 1955 hypothyroid and thyromegally were well described.(3,4,5)

In 1966 the first cases of “beer drinker’s cardiomyopathy” were reported.(6) Essentially simultaneous cases were noted in heavy beer drinkers in the US and Canada. Other findings included liver and thyroid disease. Although at the time, this disorder was felt to be largely nutritional and treated with supportive care and thiamine, ultimately the outbreak was traced to the intentional addition of cobalt sulfate to beer to increase the longevity of the foam. Because of experimental difficulties reproducing the cardiac effects in animals, many researchers have concluded that cardiomyopathy requires poor nutrition and/or alcoholism to occur.

The most recent experience with cobalt toxicity comes from the use of cobalt and chromium in metal-on-metal hip implants. Excessive wear and dysfunctional devices liberates metal into local tissues and ultimately enters the systemic circulation. Case reports of thyroid dysfunction, peripheral neuropathy, cardiomyopathy, and loss of hearing and sight are attributed to metal release from these devices.(7,8,9)

There are no controlled trials in humans to best guide therapy for patients with clinical findings of cobalt toxicity and elevated blood concentrations. Removal of exposure seems reasonable in cases of documented toxicity from dysfunctional orthopedic implants, but thresholds for removal based on blood concentrations are ambiguous and must be balances by operative risks. Recent evidence cast doubt on the roles of hemodialysis and therapeutic plasma exchange.(10) In animal models, early administration of EDTA and NAC show the most promise,(11,12) while another model favors DTPA(13). Human case reports suggest benefits of CaNaEDTA(14) and DMPS(15).

In conclusion based on limited data this author would suggest the use of CaNaEDTA, NAC, DTPA or DMPS depending on availability along with daily thiamine for its potential role in treating cardiomyopathy.

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Learning Objectives:

1. Describe the epidemiology of cobalt poisoning
2. Explain the biochemical basis for cobalt toxicity
3. Discuss the evaluation of patients with suspected cobalt toxicity
4. Describe the best treatments for patients with cobalt toxicity