Cobalt intoxication diagnosed with the help of Dr House

Kirsten Dahms, Yulia Sharkova, Peter Heitland, Sabine Pankuweit, Juergen R Schaefer

In May, 2012, a 55-year-old man was referred to our clinic for severe heart failure (New York Heart Association class IV). He had raised brain natriuretic peptide of 1053 ng/L (normal <55 ng/L) and his estimated ejection fraction by echocardiography was 25%. His medical history was mostly uneventful, apart from the fact that he had had both hips replaced by prostheses. Coronary artery disease had been excluded by heart catheterisation; cardiomyopathy was therefore regarded as the cause of heart failure. Additionally he had fever of unknown origin, hypothyroidism, and reflux oesophagitis. His mediastinal lymph nodes as well as the lymph nodes at his left hip were enlarged. At this side he had had hip replacement surgery in November, 2010, when a metal-on-polyethylene prosthesis (head Zimmer CoCrMo Protasul, metal [Zimmer, Winterthur, Switzerland]; inlay Aesculap NH 413 Chirulen PE [Aesculap, Tuttlingen, Germany]) was implanted to replace a broken ceramic-on-ceramic hip prosthesis (implanted December, 2001; head Aesculap NK 561 Biox fort, inlay Aesculap NH 103 Plasmacup). All symptoms appeared within the past year before his admission to our centre. Searching for the cause combining these symptoms—and remembering an episode of the TV series “House” which we used for teaching medical students (series seven/episode 11)—we suspected cobalt intoxication as the most likely reason. We did radiography of the hip and measured cobalt and chromium. The radiograph showed a myositis ossificans-like picture attributable to metal debris at the left-sided hip. The measurement of cobalt and chromium in the blood showed severe increase of these metals. In a heparin-blood sample the cobalt concentration was 15000 nmol/L (normal <15·3 nmol/L) and chromium was 942 nmol/L (normal <9·6 nmol/L). The cobalt concentration in 24 h urine was 6140 nmol/L (normal <17 nmol/L) and chromium urine concentration was 52300 nmol/L (normal <11·5 nmol/L). We initiated 2,3-dimercaptopropane-1-sulfonate treatment and referred the patient to his former orthopaedic clinic, where he received a new left ceramic hip prosthesis, and subsequently—because of the severe heart failure—an implanted cardioverter-defibrillator. Most likely because of remaining ceramic particles, the metal head of the hip replacement was severely damaged (figure). Shortly after the hip replacement, the patient’s plasma cobalt and chromium concentrations decreased, and the patient stabilised and recovered slightly. In July, 2013 (14 months after removal of the metal hip), heparin-blood concentration of cobalt was 1460 nmol/L and chromium was 365 nmol/L. Cardiac function improved to 40% and there were no new episodes of fever or signs of oesophagitis. However, the patient’s hearing and vision recovered only slightly.

Cobalt intoxication has been a well known cause of cardiomyopathy for over 50 years; however, it has mostly been known in the context of so-called Quebec beer drinkers’ cardiomyopathy and hard steel work-related exposure to cobalt. The stability of cobalt in combination with chromium and molybdenum (usually Co 70%, Cr 25%, Mo 5%) made this metal an excellent and stable compound in hip prosthetics. Numerous studies have investigated metal exposure due to metal hip arthroplasties.

Contributors
KD, YS, and JRS looked after the patient and wrote the report. SP and PH managed and measured the blood samples. All authors reviewed the text. Written consent by the patient to publish this report was obtained. JRS was supported by the Dr R Pohl Foundation.

References