Austin Cardio & Cardiovascular Case Reports

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Austin Publishing Group

Case Report

A Case with Recurrent Chest Pain and Chronic Lead Exposure

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Received: November 18, 2020; **Accepted:** December 15, 2020; **Published:** December 22, 2020

Abstract

Lead exposure may cause serious cardiovascular disease. However, the current threshold for lead poisoning is still unclear. We reported on a 44-year-old woman who had recurring coronary spasms induced by long-term low-level lead exposure, manifesting as acute myocardial infarction. This case emphasizes lead should be considered as a risk factor for cardiovascular disease even if blood lead concentrations are normal. Also, the accepted reference value of lead harmlessness should be lowered.

Keywords: Chronic lead exposure; Coronary vasospasm; Lead poisoning threshold

Abbreviations

LVEF: Left Ventricular Ejection Fraction; CAG: Coronary Angiography; LAD: Left Anterior Descending Artery; LAX: Left Circumflex Artery; RCA: Right Coronary Artery; Lp(a): Lipoprotein(a); Ca^{2+}: Calcium Ion

Case Presentation

A 44-year-old woman was brought to the emergency room of the first hospital due to paroxysmal left shoulder and forearm pain for 20 days and paroxysmal chest pain for 1 day. She was engaged in assembling batteries, has a two-year history of lead exposure. Chest pain occurs at work and worsens gradually. The emergency electrocardiogram showed electric axis left, ST-segment elevation in leads V1~V5, terminal negative T-waves in leads V2-V6, the amplitude of R wave in leads II, III, aVF, V1, V2 are reduced, only small q waves are visible and disappeared R wave in leads V3~V6 (Figure1). Cardiac troponin increased obviously. The echocardiogram showed a normal Left Ventricular Ejection fraction (LVEF) (65%) in the emergency room. On the first day after admission, severe chest pain still occurred accompanying with dyspnea, which persisted for 20 minutes until 3 mg morphine was injected. On the third day the second echocardiogram showed decreased LVEF (46%). After that, she was transferred to the second hospital. On the seventh day of hospitalization, she had a recurrence of chest pain. Coronary Angiography (CAG) showed that the Left Anterior Descending Branch (LAD) had full stenosis from the opening to the distal part, with 90% stenosis at the most severe point, full stenosis from the Left Circumflex Branch (LCX) opening to the distal part, and 90% stenosis at the most severe point. Diffuse disease of Right Coronary Artery (RCA), in severe cases up to 70% stenosis. (Figure 2A,B&C, Supplementary material online, Videos S1-S3). Considered the serious disease of the three arteries of LAD, RCA and LAX, then she was recommended with coronary artery bypass graft and transferred to our hospital (the third hospital). After discussing with the experts in our hospital, considered that the patient's CAG showed diffuse vascular occlusion, but there is no risk factor for coronary heart disease. Based on the medical history and clinical symptoms, we highly suspected coronary artery spasm, so diltiazem and nitrates were prescribed to her immediately. Chest distress disappeared after 2 days. Repeated coronary angiography revealed complete resolution of all stenosis (Figure 2D,E&F, Supplementary material online, Videos S4-S6). Repeated the echocardiogram showed a normal LVEF (60%). Serum lead level was normal (76.9μg/L) in our hospital, Lipoprotein(a) (Lp[a]) level is 851.1mg/L, sex hormone level and connective tissue-related examinations are normal. After excluding

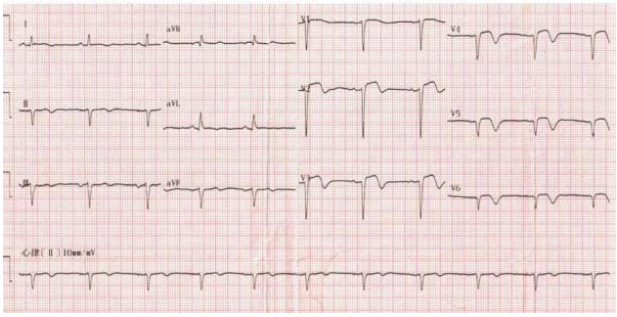

Figure 1: ECG obtained when patient developed chest pain demonstrating, electric axis left, ST-segment elevation in leads V1~V5, terminal negative T-waves in leads V2-V6, the amplitude of R wave in leads II, III, aVF, V1, V2 are reduced, only small q waves are visible and disappeared R wave in leads V3~V6.

Citation: Fu F, Miao Z, Huijun Y, Daoquan P and Ling L. A Case with Recurrent Chest Pain and Chronic Lead Exposure. Austin Cardio & Cardiovasc Case Rep. 2020; 5(1): 1037.

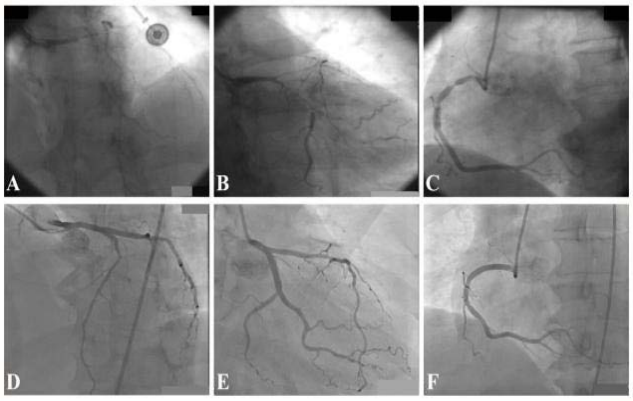

Figure 2: (A) Angiogram of left coronary artery system showing diffuse and extensive stenosis of the left anterior descending artery. (B) Coronary angiography revealed diffuse and extensive stenosis of the left circumflex artery. (C) Angiogram of right coronary artery with proximal stenosis. (D-F) Repeated Coronary angiography showed that all the stenosis was relieved by take diltiazem and nitrate.

hyperthyroidism and pheochromocytoma, she was discharged home on a regimen of diltiazem and nitrate, with advice to stop exposure to lead dust.

Discussion

Lead is a common toxic metal. Studies have shown that lead exposure causes endothelial injury by promoting oxidative stress, reducing nitric oxide production and release, increasing adrenergic activity, augmenting endothelin production, promoting inflammation, disrupting $Ca2^{+}$ signaling in vascular smooth muscle, thereby causing cardiovascular disease [1]. Endothelial injury is the main pathogenesis of coronary artery spasm. Therefore, lead may play a positive role in inducing coronary spasm. Previous literature has reported cases of coronary spasm associated with chronic lead poisoning [2]. The Bureau of Toxic Substances and Disease Registration [3] recommends limiting the blood lead concentration of adults exposed to lead at work to 60μg/dl. However, the current threshold for lead poisoning is still unclear. Some researchers injected low-dose lead acetate into Wistar rats for 30 days, finally observed that low-level blood lead concentration (12μg/dl) would increase systolic blood pressure and vascular phenylephrine reactivity [4]. This shows that the threshold of lead poisoning may be very low or even non-existent. In our opinion, some considerations should be given to this case. First, the blood lead concentration mainly reflects the recent/current lead exposure, with a short half-life (30 days), and the patient has left work for 2 months when the blood lead concentration was measured in our hospital. Due to limited hospital conditions, the bone lead level was not measured. The patient's blood lead level has likely risen at work. Secondly, high Lp(a) is another characteristic of this patient. Some scholars studied the serum Lp(a) levels between 77 patients with coronary artery spasm and 81 patients without cardiovascular disease and concluded that the incidence of elevated serum Lp(a) level was not significantly different between the two groups, suggesting that Lp(a) may not play an important role in the pathogenesis of coronary spasm [5]. The patient had no high-risk factors for coronary artery disease. The sex hormone level and connective tissue-related examinations were normal. Thirdly, reports of coronary artery spasm causing heart failure are not uncommon [6]. Therefore, this will cause the LVEF to drop in a short time. This may explain why the patient's LVEF decreased in a short time. Finally, we followed the patient for four years, after stopping lead exposure, she intermittently took $Ca2^{+}$ blocker and nitrate therapy, and she is still doing well, not complaining of any cardiac symptoms. This case emphasizes lead should be considered as a risk factor for cardiovascular disease even if blood lead concentrations are normal. For patients with chest pain, we should be careful to ask if they have a history of lead exposure. Also, the accepted reference value of lead harmlessness should be lowered.

Conclusion

As this case illustrates, although blood lead levels are normal for patients with chest pain, after eliminating common causes, we should consider whether there is a history of metal lead exposure.

Acknowledgment

We sincere thanks to all the peer reviewers and editors for their opinions and suggestions.

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