

Massive Bilateral Pleural Effusion Due to Amlodipine Intoxication: Case Report

Elnaz Vafadar Moradi*, Morteza Talebi Doluee, Seyed Mohammad Mousavi and Seyed Reza Ahmadi Koupaei

Department of Emergency Medicine, Faculty of Medicine,
Mashhad University of Medical Sciences, Mashhad, Iran.

***Corresponding Author**

Elnaz Vafadar Moradi, Department of Emergency Medicine, Faculty of
Medicine, Mashhad University of Medical Sciences, Mashhad, Iran.

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Abstract

One of the rare but serious side effects of amlodipine is pleural effusion, which is the accumulation of fluid in the space between the lungs and chest wall. This condition can cause difficulty breathing, chest pain, coughing, and fatigue. In severe cases, it can lead to respiratory failure and death. A 19-years-old woman admitted in our Emergency department with generalized weakness after ingestion of 40-tablets of Amlodipine 5mg to suicide about three hours ago. Her vital signs were stable at admission; on the second day, she complained of dyspnea. Chest Computed Tomography revealed massive bilateral pleural effusion. An echocardiogram showed normal left ventricular ejection fraction (55%), no regional wall motion akinesia, and normal right ventricular size. She was followed for possible hypoglycaemia, there was no need for Glucagon administration during the treatment. Liver function test and metabolic panel revealed normal during hospitalization. The transudate PE was completely resolved after 8- days, and she discharged home after removal of the chest tubes. As we review reports on amlodipine intoxication, hypotension was a common finding in all cases, but few cases report pleural effusion and there wasn't any report for bilateral pleural effusion despite normal cardiac activity. Our patient was only 19-years-old without past medical history of disease and bilateral pleural effusion as a presentation of amlodipine intoxication in this situation seems rare.

Keywords: Amlodipine, Toxicity, Pleural Effusion

1. Introduction

Amlodipine is a commonly prescribed medication for the treatment of hypertension and angina. It belongs to the class of calcium channel blockers, which work by relaxing the blood vessels and improving blood flow. However, like any other medication, amlodipine can cause side effects if taken in excessive amounts or for prolonged periods.

One of the rare but serious side effects of amlodipine is pleural effusion, which is the accumulation of fluid in the space between the lungs and chest wall. Bilateral pleural effusion refers to the presence of fluid on both sides of the chest. This condition can cause difficulty breathing, chest pain, coughing, and fatigue. In severe cases, it can lead to respiratory failure and death. The mechanism behind this toxicity is not fully understood, but it is believed to be related to the drug's effect on the endothelial cells lining the blood vessels.

2. Case Presentation

A 19-years-old woman admitted in our Emergency department with generalized weakness after ingestion of 40-tablets of Amlodipine 5mg to suicide about three hours ago. Her vital signs revealed as follows: Blood Pressure 70/40mmHg, Pulse Rate 125 beats per minutes, Respiratory Rate 18 per minutes, Saturation in air room 94% and Temperature Axillary 37°C. She received Gluconate Calcium 1 gram/q6h, Hydrocortison 100mg/stat, and IV crystalloid normal saline. The patient was hypotensive despite receiving crystalloid, so norepinephrine was administered, therefore hypotension resolved by NE. On the second day, she complained of dyspnea. Chest Computed Tomography (CT) revealed massive bilateral pleural effusion (PE) (Figure 1). We had placed right sided chest tube on the second day and left sided on the third day, clear amber yellow fluid drained to chest bottles. Each chest tube drained 200cc transudate PE daily. An echocardiogram showed normal left ventricular ejection fraction (55%), no regional wall motion akinesia, and normal right ventricular size.

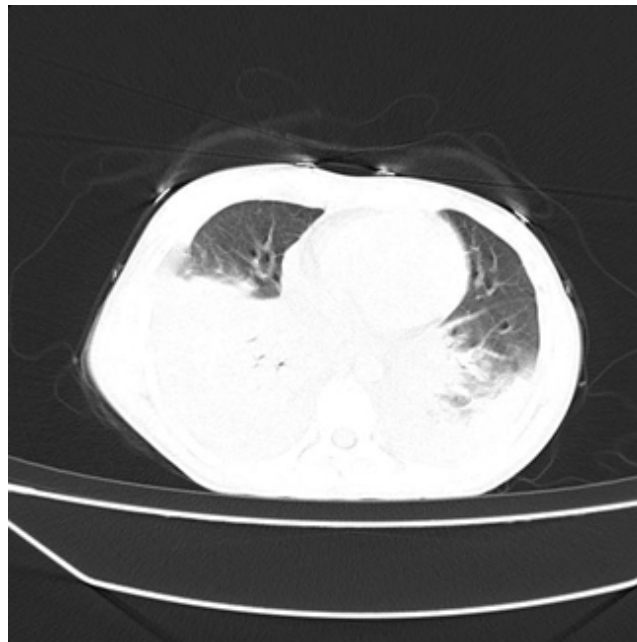


Figure 1: Chest CT Scan Revealed Massive Bilateral Pleural Effusion.

She was followed for possible hypoglycaemia, her Blood Sugar was as followed from the first day till discharged home after 8 days: 130, 148, 154, 85, 76, 71, 80 and 83 (mg/dlit). There was no need for Glucagon administration during the treatment. Liver function test and metabolic panel revealed normal during hospitalization. The transudate PE was completely resolved after 8- days, and she discharged home after remove the chest tubes.

3. Discussion

As you know better than us, Calcium Channel Blockers (CCB) can decrease blood pressure by acting on calcium-channels in vessels and heart. They also inhibit secretion of insulin in pancreas which lead to hypoglycaemia [1]. The vasodilation effects of these drugs can lead to pulmonary capillary dilation and permeability increase and the later effect cause peripheral edema [2]. Amlodipine is widely available as a dihydropyridine CCB and its overdose lead to cardiovascular collapse, cardiac dysrhythmia and hypotension. Our patient had hypotension and bilateral pleural effusion without significant hypoglycaemia and any cardiovascular dysrhythmia.

Yilmaz and his co-workers had studied on fourteen patients with CCB poisoning, of these 9-patient had pleural effusion, of which 5 were taking amlodipine [3]. They believed that vasodilation and impaired perfusion leads to pleural effusion. Our patients also had bilateral pleural effusion which was not seen yet.

Kute and colleagues reported another case report of amlodipine intoxication with refractory hypotension and non-cardiogenic pulmonary edema who had 48-hours delayed in refereeing to hospital. They treated her with IV crystalloid, vasopressor and supportive mechanical ventilation [4]. It seems that a similar pathophysiology like precapillary vasodilation leads to non-cardiogenic edema in their patient and bilateral pleural effusion in our patient. We also treated our patient with IV crystalloid and vasopressor, she responds well to the treatment and there was no need for mechanical ventilation.

Koliastasis et al. reported a 72-years old woman with amlodipine intoxication and refractory shock and hyperinsulinemia. While resuscitated the patients, she was intubated due to pulmonary edema. Treatment with high dose insulin in combination with Dextrose50%, and furosemide was initiated. After eight-days she was weaned from mechanical ventilation and discharged home after 14-days [5]. In this report patients had another symptoms of amlodipine intoxication, hyperinsulinemia, but our patients didn't have it and only had mild hypoglycaemia after 4-days of hospitalization. Hypotension is the most clinical symptoms in patients which is commonly respond to IV resuscitation and sometimes vasopressors.

As we review reports on amlodipine intoxication, hypotension was a common finding in all cases, but few cases report pleural effusion and there wasn't any report for bilateral pleural effusion despite normal cardiac activity. Our patients was only 19-years-old without past medical history of disease and bilateral pleural effusion as a presentation of amlodipine intoxication in this situation seems rare.

4. Conclusion

This case report provides valuable insights into potential adverse effect of amlodipine when taken in excess. It serves as a reminder for healthcare professionals to be vigilant in monitoring patient's medication use and educating them on proper dosing techniques.

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Conflict of interest.

None

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Data availability. Patient's data and file is available.

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