Post-Biperiden Myocardial Depression and Acute Pulmonary Oedema: A Case Report

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ABSTRACT

Biperiden, a muscarinic receptor antagonist, is utilised in antipsychotic therapies and has been associated with a restricted incidence of cardiac side effects. This case study details a 30-year male patient who had acute pulmonary oedema and cardiac depression after a biperiden overdose. The patient arrived at the emergency department exhibiting dyspnoea, agitation, and a low oxygen saturation level of 65%. The physical examination and imaging indicated pulmonary oedema and diminished heart motion. The intervention included non-invasive ventilation, nitroglycerin infusion, and diuretics. Throughout the clinical follow-up, the patient's respiratory and cardiac symptoms exhibited improvement, with no alterations noted in troponin levels. It can be inferred that the infrequent cardiac side effects of biperiden, particularly in cases of overdose, can lead to severe consequences such as acute pulmonary oedema. Consequently, it is imperative to effectively manage this illness to avert fatality.

Key Words: *Biperiden, Acute pulmonary oedema, Cardiac depression*.

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INTRODUCTION

A review of the literature reveals a preponderance of studies indicating that biperiden exerts a deleterious effect on cognitive and physiological processes. This effect is attributable to its capacity as an antagonist of muscarinic M1 receptors. One important negative consequence of selective impairment of verbal episodic memory is dose- and time-dependent manifestation. While leaving attention and motor skills unchanged, this impairment is especially seen in activities requiring verbal learning and extended recognition memory. This could provide a possible paradigm for looking at memory problems similar to those observed in Alzheimer's disease.[1,2]

Moreover, biperiden has been linked to acute urine retention, a side effect more usually reported in men, especially those with prostatic hypertrophy. A case study shows that women have also been recorded in this regard.[3] Biperiden has been linked in paediatric cases to delirium even at non-toxic levels, which emphasises the need for caution while prescribing it to little children.[4]

Notwithstanding the already indicated cognitive and physiological adverse effects, biperiden seems not to affect the reinforcing power of cocaine.

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For instance, it has been shown that biperiden lowers cocaine-induced ultrasonic vocalisations in rats, devoid of any effect on cocaine-induced locomotor activity.[5] Although biperiden has no direct relationship with cardiac tissue engineering or heart function, the larger background in cardiac health includes knowledge of the systemic impacts of several pharmacological substances. For example, research in the field of cardiac tissue engineering aims to create three-dimensional cardiac muscle structures in order to either replace or repair injured cardiac tissue, hence preserving heart function.[6]

This paper describes a case of a patient utilising biperidin who showed up to the emergency department with cardiac depression and acute pulmonary oedema.

CASE REPORT

A 30-year male patient presented to the emergency department with the primary complaint of shortness of breath. Additionally, symptoms of agitation, tremors, and cold perspiration were present. He reported experiencing dyspnoea, tachypnoea, and orthopnoea. The patient was diagnosed with a non-organic psychotic disorder after a review of medical history. He was presently receiving treatment with biperiden 2 mg three times daily, quetiapine 300 mg once daily, and risperidone 4 mg twice daily. It was determined that the patient had consumed two dosages of biperiden before his symptoms had become apparent.

The patient's vital signs were assessed upon admission. The patient's arterial blood pressure was recorded at 153/102 mmHg, and his oxygen saturation was observed to have decreased to 65% when assessed with ambient air. His respiratory rate was recorded at 18 breaths per minute.

The physical examination revealed crepitant rales following lung auscultation and the usage of accessory respiratory muscles. The patient was alert but showed reduced coordination and orientation. The abdominal examination produced no significant results.

Two intravenous access lines were set up to guarantee the patient's safety. He was attached to a non-invasive mechanical ventilator for respiratory support. The pH of venous blood gas measurement was 7.28, the pCO_2 was 59.9 mmHg, the pO_2 was 17.3 mmHg, the lactate level was 3.2 mmol/L, and the base excess (BE) was -22.4. The diagnosis of type 1 respiratory failure was considered. With a non-invasive mechanical ventilator and continuous positive airway pressure (CPAP) support, the patient's oxygen saturation levels showed improvement, and his tachypnoea showed a clear drop during the next follow-up period.

An electrocardiogram (ECG) was performed once the patient had stabilised and showed a 2-mm ST-segment elevation in leads V1–V4 together with a heart rate of 96 beats per minute (bpm). This was consistent with an incomplete bundle-branch block pattern (Figure 1). Thoracic ultrasonography showed the existence of pleural effusions; a point-of-care ultrasonic (POCUS) examination indicated an akinetic apex and a global hypokinetic heart (Figure 2).

The initial assessment's biochemical analysis produced the following results: Alanine aminotransferase (ALT) of 54.7 U/L, aspartate aminotransferase (AST) of 35.5 U/L, creatine kinase (CK-MB) of 38.3 U/L, and lactate dehydrogenase (LDH) of 318 U/L. The patient's high-sensitivity troponin T level was 34.33 ng/L, with no subsequent increase observed during the follow-up period.

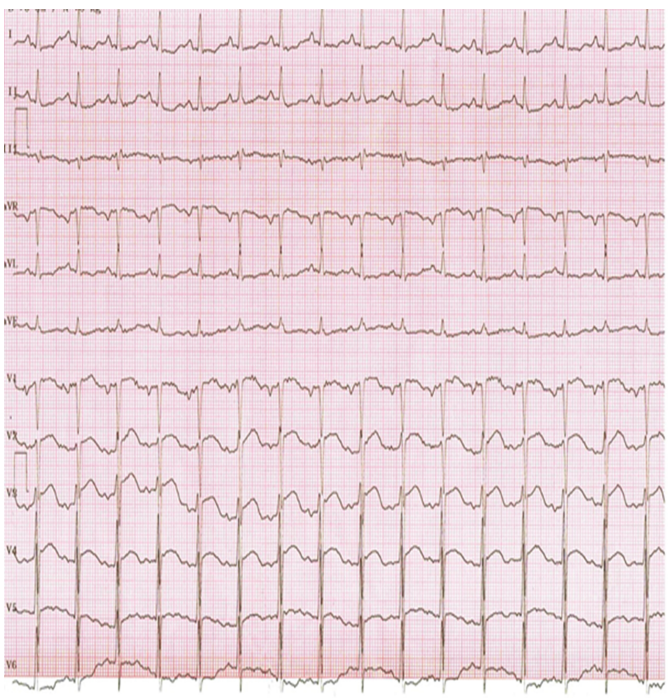

Figure 1: ECG

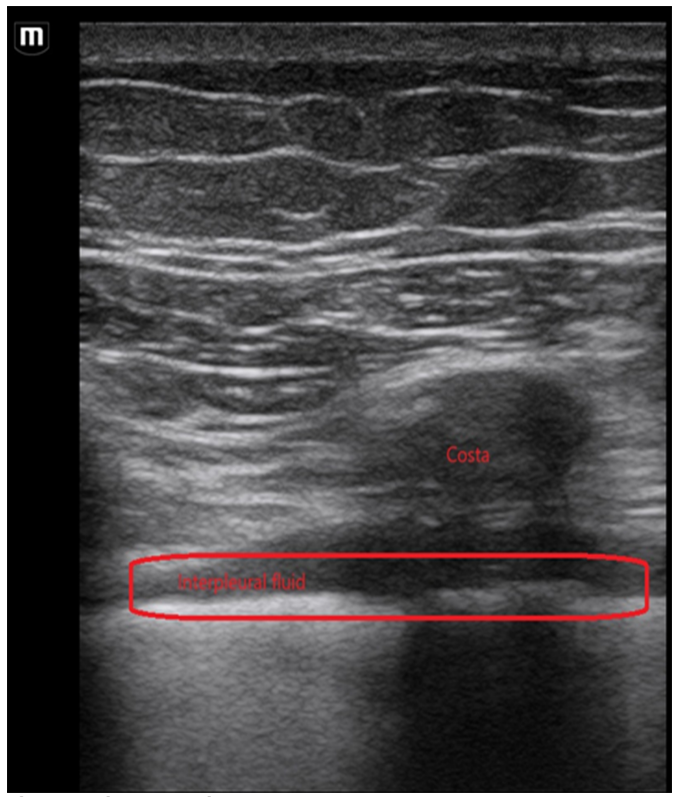

Figure 2: Thorax USG image.

The authors placed a urinary catheter during the patient's hospitalisation and infused nitroglycerin at a rate of 3 µg/min to control preload. The authors administered furosemide at a rate of 20 mg per hour to promote diuresis. The patient's respiratory function improved as the treatment regimen continued, and his level of cooperation and orientation returned to normal. A follow-up arterial blood gas analysis demonstrated a pH of 7.33, pCO_2 of 47 mmHg, and pO_2 of 75 mmHg. Subsequent monitoring indicated a reduction in the pleural effusion and a resolution of tachypnoea and dyspnoea. The authors discharged the patient with improved consciousness, restored cooperation, and orientation, and recommended psychiatric outpatient follow-up for medication management.

DISCUSSION

Rare but important clinically, medicine-induced acute pulmonary oedema can be brought on by the use of ketamine, phenylephrine, naloxone, and nonsteroidal anti-inflammatory drugs (NSAIDs). Commonly used in emergency settings for acute agitation, ketamine has been linked to the development of pulmonary oedema through mechanisms including the inhibition of epithelial sodium channels and sympathomimetic effects, perhaps causing transient tachycardia and hypertension, particularly in patients with pre-existing cardiac conditions.[7] Likewise, rapid changes in heart-filling pressures brought on by phenylephrine following caesarean deliveries might cause unexpected pulmonary oedema. This emphasises the need for careful haemodynamic control in pregnant women.[8]

Moreover, naloxone, an opioid antagonist, has been found as a possible cause of acute pulmonary oedema because of the fast physiological changes that could accompany the reversal of an opioid's impact. The case of a patient who needed significant breathing support following a naloxone injection helps to highlight this.[9] The pharmaceutical agent known as biperiden is utilised in the treatment of several symptoms associated with Parkinson's disease, including stiffness, tremors, spasms, and impaired muscle control.[10]

A review of the existing literature reveals that medicines utilised in antipsychotic treatment, including opioid medications and ketamine, have been associated with the development of pulmonary oedema. In this case, it is emphasised that this phenomenon can be observed following the administration of biperiden. It is imperative to acknowledge the cardiac consequences of antipsychotic medications, which have the potential to result in fatal outcomes.

Usually showing as hypotension and bradycardia, biperiden, used as an antipsychotic medicine, hardly affects the cardiovascular system. Slow medication delivery has been claimed to help reduce these cardiac effects.[10] In this situation, biperiden—acting as a muscarinic receptor antagonist—was taken in dosages above the therapeutic range, which most certainly contributed to the recorded cardiac adverse effects. The reduced cardiac motion was shown by bedside POCUS evaluation. Although biperiden rarely results in such adverse outcomes, the noted impact in this patient resulted in acute heart failure, which subsequently led to pulmonary oedema.

Changes caused in preload and afterload help to define the degree of cardiac impact. As our example shows, acute pulmonary oedema might cause morbidity and may be fatal. This disorder can develop from the effects on calcium transport in cardiac muscles, without myocardial necrosis, which clarifies the lack of increases in troponin levels.

In conclusion, the absence of myocardial injury explains the lack of fluctuation in troponin levels. However, a drop in heart rate may result in severe pulmonary oedema and even lethal consequences.

PATIENT'S CONSENT:
The preparation of this article was conducted with the explicit consent of the patient, with the utmost confidentiality maintained concerning their personal information.

COMPETING INTEREST:
The authors declared no conflict of interest.

AUTHORS' CONTRIBUTION:
OZ: Literature search and manuscript writing.
DK: Literature search and proofreading.
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