Case Report

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An unusual case of Lactic Acidosis

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Introduction

Lactic acidosis is an important cause of metabolic acidosis in hospitalised patients. This usually occurs either due to over production or under utilisation of lactate[1] . Most cases of lactic acidosis are due to marked tissue hypoperfusion or hypoxia in systemic shock.

Asymptomatic lactic acidosis has been reported previously during acute severe asthma and attributed to fatiguing respiratory muscles, hypoxaemia and liver ischaemia. It has also been linked to β2 agonist therapy in asthma, although lactic acidosis causing increasing dyspnoea in the asthmatic patient has only been recorded rarely.

Case presentation

We present a case of lactic acidosis in a patient with acute severe asthma who did not have any overt signs of sepsis or tissue hypoperfusion.

Mr IL was a 49 years old male who was known to have moderate asthma. He had multiple previous admissions to hospital with exacerbation of asthma but had never required an intensive care admission and had never been intubated. His other comorbidities included atrial fibrillation, ischaemic heart disease and depression.

His usual medications included salbutamol, budesonide and salmeterol inhalers, aspirin, atorvastatin and digoxin. He was a mechanic by trade with no obvious occupational sensitisation. He had no pets at home. He was a smoker with a 20 pack year history. Recent lung function tests showed an FEV1/FVC of 0.68 with a post bronchodilator FEV1 of 4.17 L (95% predicted).

He was admitted with a 1 week history of worsening shortness of breath, dry cough and wheeze. His baseline blood tests including full blood count, C reactive protein, liver and renal function were normal. Chest radiograph was unremarkable. Arterial blood gas showed no evidence of hypoxia or acidosis. He was treated as acute severe asthma with back to back nebulisers, intravenous hydrocortisone and magnesium sulphate resulting in gradual improvement in bronchospasm and peak expiratory flow rate.

Despite optimal treatment, his breathing started to deteriorate. Arterial blood gas at this time showed lactic acidosis with normal oxygenation (Table 1). There was no clinical or biochemical evidence of haemodynamic compromise or sepsis. A presumptive diagnosis of lactic acidosis secondary to salbutamol was made. The nebulisers were withheld and he has transferred to high dependency unit for closer monitoring. The acidosis completely resolved in the following 12 hours on stopping salbutamol and the patient made an uneventful recovery.

Discussion

Lactate is a product of anaerobic glucose metabolism and is generated from pyruvate. Normal plasma lactate concentration is 0.5-2 meq/L. Most cases of lactic acidosis are due to marked tissue hypoperfusion or hypoxia in systemic shock[2] .

Lactic acidosis can occur in acute severe asthma due to inadequate oxygen delivery to the respiratory muscles to meet an elevated oxygen demand[3] or due to fatiguing respiratory muscles[4] . A less recognised cause of lactic acidosis is treatment with salbutamol. The mechanism of this complication is poorly understood.

Salbutamol is the most commonly used short acting βagonist. Stimulation of β adrenergic receptors leads to a variety of metabolic effects including increase in glycogenolysis, gluconeogenesis and lipolysis[5] thus contributing to lactic acidosis.

Table 2 shows an assortment of previously published case reports and case series of lactic acidosis in the context of acute asthma.

Table 1: Serial Arterial Blood Gases (On admission, 4 hours later and on stopping salbutamol)

	00:22	**04:06**	**07:42** Salbutamol withheld	**10:50**	**11:35**	**12:24**	**14:29**	**17:33**	**23:32**
FiO2	100%	60%	60%	60%	60%	40%	40%	35%	28%
pH (7.35-7.45)	7.36	7.28	7.26	7.32	7.34	7.37	7.37	7.39	7.41
pCO2 (4.5-6.0 kPa)	4.87	4.74	4.15	3.31	3.98	3.9	4.7	5.08	5.49
pO2 (11-14 kPa)	27	19.2	16.5	19	18	14.1	12.5	13	11.8
HCO3 (22-28 mmol/L)	22	16.3	13.6	12.4	15.6	16.6	19.9	22	25.6
BE (2- -2)	-2	-9.1	-12	-12	-9	-7.6	-4.4	-1.5	1.4
Lactate (0.5-2 mEq/L)	1.8	7.6	9.7	9.3	7.6	6.8	3.6	1.4	1.1

Table 2: Details of etiology and consequences of lactic acidosis in previously published case reports

Reference	**n**	**Suggested etiology of lactic acidosis**	**Effect of lactic acidosis**
Roncoroni et al, 1976 [6]	25	Uncertain: increased respiratory muscle production, decreased muscle or liver metabolism	None observed
Appel et al, 1983 [7]	12	Increased respiratory muscle production, decreased muscle or liver metabolism	8 out of 12 developed respiratory acidosis, 6 required invasive ventilation
Braden et al, 1985 [8]	1	2 agonist, steroid and theophylline therapy	None
O'Connell & Iber, 1990 [9]	3	Uncertain: intravenous 2 agonist versus severe asthma	None
Mountain et al, 1990 [10]	27	Hypoxia and increased respiratory muscle production	None
Maury et al, 1997 [11]	1	2 agonist therapy	Inappropriate intensification of 2 agonist therapy
Prakash and Mehta, 2001 [2]	2	2 agonist therapy	Contributed to hypercapneic respiratory failure
Manthous, 2001 [12]	3	2 agonist therapy	None
Stratakos et al, 2002 [3]	5	2 agonist therapy	None
Creagh-Brown and Ball, 2008 [13]	1	β2 agonist therapy	Patient required invasive ventilation
Veenith and Pearce, 2008 [14]	1	β2 agonist therapy	None
Saxena and Marais, 2010 [15]	1	2 agonist therapy	None

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Conclusion

In this case, the patient developed lactic acidosis secondary to treatment with salbutamol nebulisers. The acidosis resolved spontaneously without any specific treatment.

Lactic acidosis secondary to β agonist administration may be a common scenario which can be easily misinterpreted and confuse the clinical picture. Acidosis itself results in hyperventilation which could be mistaken for failure to treat the

response. This may in turn lead to inappropriate intensification of treatment.

Competing Interests

None declared

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