A case of lactic acidosis complicating assessment and management of asthma

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Abstract

Introduction: Lactic acidosis often occurs in severely unwell patients presenting to Accident and Emergency. It is commonly associated with either hypoxia or decreased tissue perfusion secondary due to cardiovascular collapse or sepsis.

Case presentation: We present a case of severe lactic acidosis in the presence of normal tissue perfusion and oxygenation in a 31-year-old patient with poorly-controlled asthma. Acidosis promptly reversed on discontinuation of inhaled beta-agonists.

Conclusion: Lactic acidosis secondary to inhaled beta-agonist administration may be a common scenario which can be misinterpreted very easily and can confuse the clinical picture. Further studies will be needed to establish the exact aetiology of this lactic acid production.
tate. We excluded all common causes of a metabolic aci-
dosis in this clinical setting including hypoxia,
hypovolemia and sepsis. We suspected that the lactic aci-
dosis may have been secondary to nebulised salbutamol,
and consequently reduced the dosing interval. This
resulted in a reduction in the serum lactic acid level. When
the nebulised salbutamol was subsequently stopped the
lactic acidosis promptly reversed (Table 1). The patient
was transferred to the ward and discharged home une-
ventfully.

In this particular patient, salbutamol and its resultant
metabolic acidosis caused us difficulty in assessment and
management of her asthma.

Discussion
Salbutamol is a beta agonist associated with a multitude
of systemic side effects. One of the least recognised side
effects of salbutamol with clinical consequence is lactic
acidosis. Lactic acidosis is commonly associated with
either hypoxia or decreased tissue perfusion either due to
cardiovascular collapse or sepsis [2]. There are many views
regarding the pathogenesis of lactic acidosis in asthma;
the most accepted one being due to fatiguing respiratory
muscles [3]. Another accepted explanation could be due
to effects of ischemia and hypoxia on liver which was
unlikely in the above case as this patient had normal liver
function [4].

In healthy volunteers salbutamol will increase the oxygen
consumption by increasing metabolic rate and serum lac-
tate. This will affect patients with severe asthma, who
already limited ventilatory reserve, by increasing their
symptoms [5]. In pregnant patients the lactate levels
increase when given Ritodrine (beta agonist) for tocolysis
[6]. An increase in serum lactate has also been docu-
mented paediatric patients, followed by administration of
nebulised salbutamol [7]. This resolved after the discon-
tinuation of the drug. Lactic acidosis is much more prom-
inent in intravenous salbutamol than nebulised
salbutamol, so intravenous salbutamol should not be
routinely prescribed [8].

Conclusion
In this patient, resolution of wheezing was accomplished
by intensive treatment with salbutamol but at the cost of
lactic acidosis. This was important because acidosis by
itself can result in hyperventilation and a sense of dysp-
noea which could be easily mistaken for failure to
respond to the treatment, as this particular case exempli-
ifies. Lactic acidosis secondary to inhaled beta-agonist
administration may be a common scenario which can be
misinterpreted very easily and can confuse the clinical pic-
ture. Further studies will be needed to establish the exact
aetiology of this lactic acid production.

List of abbreviations
FBC – Full Blood count, CRP – C Reactive Protein, CXR –
Chest X-ray, ABG – Arterial Blood Gases, A&E – Accident
and Emergency, ITU – Intensive Treatment Unit, PEFR –
Peak expiratory flow rate, ECG – Electrocardiogram.

Competing interests
The author(s) declare that they have no competing inter-
ests.

Authors’ contributions
Both author(s) are involved in writing this case report.

Acknowledgements
This patient was consented for publication for this case report.

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Table 1: Arterial blood gas analysis before and after stopping salbutamol. The patient’s clinical condition and blood gases improved
dramatically after cessation of salbutamol. ABGs after 11 hours and 48 hours post-withdrawal of salbutamol are shown.

| Time/Date       | pH   | PaCO2 KPa | PaO2 KPa | HCO3 mmol/l | BE   | Lactate mmol/l | PEFR L/min | Symptoms/Treatment                      |
|-----------------|------|-----------|----------|-------------|------|----------------|-------------|----------------------------------------|
| 11/1/06 21.20 hrs | 7.39 | 3.03      | 20.5     | 13.6 mmol/l | -11  | 14.6 mmol/l    | 230         | Minimal wheeze, SOB* Reduced dose of Salbutamol |
| 11/1/06 24.00 hrs | 7.32 | 3.01      | 18.6     | 11.7 mmol/l | -14  | 11.06 mmol/l   | 240         | No wheeze, hyperventilation*           |
| 12/1/06 3.00 hrs | 7.28 | 2.29      | 11.4     | 8.3 mmol/l  | -19  | 15.2 mmol/l    | 230         | SOB**, hyperventilation                |
| 12/1/06 7.00 hrs | 7.3  | 2.13      | 13.8     | 8.0 mmol/l  | -18  | 10.09 mmol/l   | 300         | Minimal wheeze Nebulised salbutamol discontinued |
| 12/1/06 23.00 hrs | 7.43 | 3.46      | 12.3     | 17.6 mmol/l | -7   | 6.3 mmol/l     | 300         | No wheeze Continued improvement       |
| 14/1/06 10.00 hrs | 7.53 | 3.39      | 9.6      | 21.0 mmol/l | -2   | 1.1 mmol/l     | 320         | Discharged to ward                   |
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