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Single Case

Inadvertent Intoxication with Salbutamol, Treated with Hemodialysis: A Case Report and Brief Review of the Literature

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Intoxication · Albuterol · Salbutamol · Hemodialysis · Case report

Abstract
Introduction: Salbutamol is a moderately selective beta-2-adrenergic agonist. Various side effects can occur because of beta-1 and beta-2 receptor activation. Due to the large volume of distribution, it is not considered dialyzable. ***Case Presentation:*** A patient with salbutamol intoxication, which developed as a result of a medical error in a patient with sepsis, Down syndrome, and liver cirrhosis, is presented. Initial treatment was partially successful and antibiotic adjustments were made. After his respiratory failure worsened, the patient needed non-invasive ventilation, and previously undiagnosed chronic obstructive pulmonary disease was suspected. He was prescribed intravenous methylprednisolone but accidently received 5 mg of salbutamol (albuterol), which led to immediate severe arrhythmic tachycardia with hemodynamic collapse. After unsuccessful cardioversion and treatment with landiolol infusion, salvage hemodialysis was commenced to decrease suspectedly highly elevated serum salbutamol levels. After 30 min, sinus rhythm with normocardia was observed. After the hemodialysis termination, no rebound tachycardia was noted, but due to severe septic shock, the hypotension was ongoing and vasoactive medications were adjusted. However, the measured levels of plasma salbutamol and data from literature do not support the view that hemodialysis was the cause of the described improvement: the total amount of the drug cleared was very small (2.8% of total dose). ***Conclusion:*** Our results confirm a large volume of salbutamol distribution; the measured

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levels are within observed therapeutic levels; and the measured half-life time during hemodialysis (3.1 h) is comparable to observed half-life times in therapeutic settings. The observed favorable clinical benefit associated with dialysis may be fortuitous, highlighting potential bias toward positive clinical outcomes and unproven ("salvage") therapies.

Introduction

Salbutamol/albuterol is a short acting, moderately selective beta-2- adrenergic receptor agonist, mostly used in the treatment and prevention of acute bronchospasm in asthma, chronic bronchitis and other chronic reversible obstructive pulmonary diseases and acts as airway smooth muscle relaxant. Intravenous salbutamol can also be used in obstetrics as a tocolytic agent and as an adjuvant treatment for severe hyperkalemia. After oral and parenteral administration various side effects can occur because of beta-1 and beta-2 receptor activation: tremor, hyperglycemia (/hyperinsulinemia), hypokalemia, hyperlactatemia, hypotension (fall in peripheral vascular resistance), tachycardia (with palpitations), QT_c prolongation, ventilation mismatch (fall in pulmonary vascular pressure) and increased metabolic rate. Due to the large volume of distribution, it is not considered dialyzable. We present a case of inadvertent intoxication with salbutamol where multiple salbutamol plasma measurements were made during a salvage hemodialysis procedure and relation to the clinical presentation was made possible. The CARE Checklist has been completed by the authors for this case report, attached as online supplementary material (for all online suppl. material, see https://doi.org/10.1159/000536523).

Case Presentation

A 69-year-old male was admitted due to acute respiratory failure and septic shock following severe pneumonia. His previous medical history included Down syndrome, heart failure, hepatitis B with liver cirrhosis, and trans-aortic valve insertion 5 years previously. His medication is listed below.

On admission, the patient was hemodynamically unstable despite volume resuscitation and needed vasoactive support with noradrenaline and hydrocortisone, and supplemental oxygen was administered via facemask. Initial laboratory testing showed elevated inflammatory markers, high lactate values, mild acute kidney insufficiency with preserved diuresis and signs of disseminated intravascular coagulation (DIC). He empirically started on piperacillin/tazobactam. After a couple of hours, his respiratory failure worsened, and he was put on non-invasive ventilation (NIV). Clinical response to antibiotic therapy after 3 days was poor; he was still febrile and tracheal aspirations were purulent, therefore vancomycin was added to antimicrobial therapy. Blood cultures came back positive for *Streptococcus dysgalactie*, and antibiotic therapy was adjusted to meropenem. NIV was successfully discontinued a few days later, but as mild respiratory insufficiency persisted, previously unrecognized chronic obstructive pulmonary disease was suspected. He was receiving regular inhalations of ipratropium/fenoterol and salbutamol, intravenous morphine, and intermittent boluses of furosemide. The patient required constant parenteral potassium replacement. He was eventually prescribed intravenous methylprednisolone but accidently received 1 mL of salbutamol (albuterol) solution 5 mg/mL instead (see Fig. 1), which led to immediate severe tachycardia with hemodynamic collapse. The medication on the day of intoxication is listed below.

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On presentation, he was somnolent, only responsive to pain, and in acute hypercapnic respiratory distress. He was tachypneic with a respiratory rate 35 per minute, receiving additional oxygen supplemented via 28% Venturi mask (4–6 L/min). Before the intoxication, his heart rate was 75/min, in sinus rhythm; afterward, he immediately presented with tachycardia (atrial fibrillation 170–200/min). Because of acute hypotension with tachycardia, the patient was immediately treated with volume resuscitation and noradrenaline (see below). This resulted in a stable but moderately low blood pressure (95/50–105/40 mm Hg).

The diagnosis of acute tachycardia with hemodynamic collapse and cardiogenic shock was made based on inadvertent intravenous salbutamol application (time 0 min). The lactate levels were also elevated.

Due to deteriorating condition, the patient was sedated with propofol, intubated, and mechanically ventilated. He was stabilized with volume resuscitation and titrated infusion of noradrenaline. The tachycardia was first treated with intravenous landiolol infusion (Table 1). The infusion was stopped as hypotension worsened (60/30 mm Hg) and heart rate persisted unchanged. The electrical cardioversion was performed according to protocol guidelines but was unsuccessful. He received potassium chloride 40 mEq to treat worsening hypokalemia.

Due to life-threatening cardiogenic shock and unresponsiveness to other standard medical interventions, small salbutamol molecular size and water solubility, we decided to treat the patient with hemodialysis as salvage therapeutic option, despite the known poor dializability of salbutamol. At dialysis initiation, the patient was hypotensive (81/34 mm Hg) and tachycardic (181/min); however, 30 min after dialysis commencement, the patient suddenly converted to sinus rhythm with pulse rate 80/min (see Fig. 1). The hypotension did not resolve, and the patient required continuing vasoactive support. The dialysis lasted for 3.5 h. After the discontinuation, his heart rate remained within the normal range and no rebound tachycardia was noted. The hypotension was however ongoing, attributed to severe septic shock; therefore, vasoactive medications were appropriately adjusted.

Lactate and potassium levels, and urine output are shown in Figure 1. He received potassium chloride infusion 40 mEq 120 min after admission. Correction during dialysis procedure must also be acknowledged. Glucose levels were elevated (range 7.1–13.7 mmol/L). Before and after dialysis procedure, serum, urine, and dialysis fluid specimens were collected for further analysis.

Despite aggressive treatment with targeting antibiotic-sensitive bacteria and all supportive measures, including multiple bronchial lavages, the patient had abundant purulent tracheal aspirations and remained febrile and hypotensive. The patient died 9 days after admission into MICU because of septic shock with multiorgan failure.

Therapy before admission: acetylsalicylic acid 100 mg, furosemide 40 mg, eplerenone 25 mg, pantoprazole 20 mg, allopurinol 100 mg and tenofovir 25 mg and was without known allergies. Family history was not available; he lived with his relatives and was able to do simple work.

Therapy on the day of admission to the MICU: noradrenaline (norepinephrine) 10 mg in 50 mL, 5 mL/h (0.18 μg/kg/min), vancomycin (1st day, 1 g I.V., 4 h before A*), tazobactam (7th day, 4.5 g I.V.), magnesium sulfate (10 mmol I.V., 110 min before A*), fenoterol/ipratropium inhalations (0.5 mg/0.261 mg/mL, 2 mL, 20 h before A), pantoprazole (40 mg I.V.), paracetamol (1 g, 5 h before A), morphine (2 mg, 4 h before A), thiethylperazine (6.5 mg I.V., 5 h before A), furosemide (40 mg I.V., 1 h before A), spironolactone (25 mg P.O.), clemastine (2 mg I.V.), tenofovir (25 mg P.O.), bisacodyl (10 mg P.R.), salbutamol (5 mg, inhalation, 5 and 2 h before A). The patient did not receive the planned methylprednisolone (40 mg I.V.) at the time of A.

*A = salbutamol/albuterol administration.

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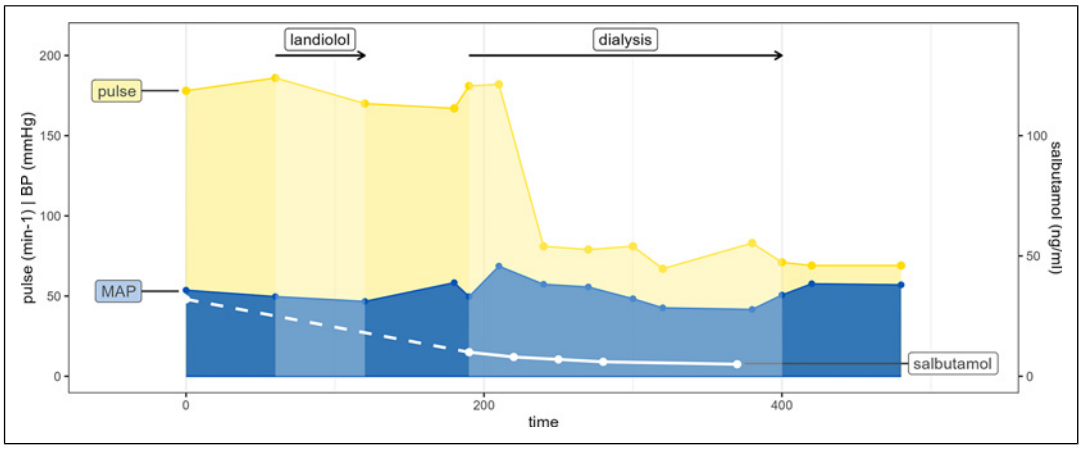

Fig. 1. Heart rate (min^{-1}) and mean arterial pressure (MAP, mm Hg) versus salbutamol levels (ng/mL).

Table 1. Timeline

Time (local)	Time elapsed, min	Therapeutic action taken
2:00 p.m.	0	Salbutamol application
2:20 p.m.	20	MICU admission
3:00 p.m.	60	Landiolol infusion start Rapibloc, 6 mg/mL, titrated infusion, total dose 180 mg, start at time 60 min, end at time 120 min
4:00 p.m.	120	Landiolol infusion stop; total dose 180 mg
4:00 p.m.	120	Electrical cardioversion (EC)
5:10 p.m.	190	Hemodialysis commencement • Dialysis machine Fresenius 4008, dialysis filter Fresenius FX80 (1.8 m^2) • Regional citrate anticoagulation • Blood flow 300 mL/h, lowered to 280 and 270 mL/h after 1st and 2nd hour, respectively
	~200	Symmetric muscle fasciculations, patient receives midazolam
5:45 p.m.	220	Normocardia; 30 min after hemodialysis commencement
8:40 p.m.	400	Hemodialysis termination

Clinical examination at day of admission to the MICU: the patient's body weight was (approximated) 76 kg, height 160 cm, BMI 29.7 kg/m^2. With a body temperature of 37.6°C, his skin was cold and clammy. Auscultation revealed diffuse loud moist inspiratory crackles and some expiratory crackles and wheezes. The heart rhythm was irregular and tachycardic; due to loud respiratory phenomena and tachycardia heart sounds were not distinguishable. The abdomen was soft, nontender; no peripheral edema or tremor was noted.

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The resuscitation measures colloidal plasma volume substitute, Gelaspan, 1,000 mL and titrated vasopressor I.V. infusion (norepinephrine), up to 0.66 μg/kg/min. Central venous access: two one-lumen dialysis catheters Medcomp®, 15 and 20 cm in the left femoral vein.

Discussion

Salbutamol/albuterol is water and alcohol soluble moderately selective beta-2-adrenergic agonist with a molar mass of 239 g/mol. It is used for the symptomatic management and prevention of bronchospasms related to asthma, exercise-induced bronchospasm, and chronic obstructive pulmonary diseases. Common adverse effects include hypokalemia, tremor, increased lactate, hyperglycemia, and sinus tachycardia [1]. Salbutamol is usually administered as aerosol; however, intravenous application is also possible, but the evidence is limited [1, 2]. Regardless the route of administration, the therapeutic (bronchodilator) action is associated with blood levels 5–20 ng/mL [1]. In the available literature, we found no experimental data regarding salbutamol (albuterol) dialyzability, but due to the drug characteristics, it is not regarded dialyzable [3].

In the presented case, the patient deteriorated immediately after salbutamol administration; severe tachycardia, a cardinal sign of salbutamol intoxication, was present. Also, the patient developed hypotension (peripheral vasodilation), respiratory failure (possibly worsened by pulmonary vasodilation), and later, symmetric muscle fasciculation were observed. Laboratory findings revealed hypokalemia, hyperglycemia and hyperlactatemia. Although the patient had several conditions, which contributed to the development of these signs, the sudden nature suggests that high plasma salbutamol was the primary cause.

The results of various pharmacokinetic studies are shown in Table 2. Briefly, salbutamol is metabolized and excreted with half-life within 2.7–6 h (Goldstein et al. [4]: 3.8, Fairfax et al. [6]: 6.0 + 1.0 (SD), Lin et al. [12]: 3.8, Powell et al. [13]: 4.8–5.5, and Walker et al. [7]: 2.7–5.0). The removal consists of renal excretion and conjugation, and the study of pharmacological properties of salbutamol metabolites suggests that they have no agonist or antagonist activity [7].

Observed salbutamol levels at the therapeutic and experimental doses are shown in Table 2. The observed adverse effects were mainly tachycardia and tremor. Interestingly, the observational data from a small cohort of patients with self-poisoning after salbutamol ingestion with high serum salbutamol levels showed only moderate side effects [9]. This report demonstrated almost linear relationship between higher levels of salbutamol, hypokalemia, and tachycardia. Of note, the measured levels were up to 10-times higher than referenced linear range for the HPLC assay used – this might have introduced measurement error, for which it is almost impossible to account.

Using data from the literature referenced above, we made some calculations pertaining to our case. After administration and equilibration of salbutamol 5 mg intravenously, the serum concentration would be 32 ± 13.1 ng/mL. The first measured level (10 ng/mL) in our clinical case was 190 min after administration. Using the available serum levels during hemodialysis procedure and approximation using first order kinetics indicated a half-life 3.1 h, which is consistent with published data (see above). The approximated maximum plasma level after (quick) distribution phase was 20.2 ng/mL, which is within predicted levels. Two things must be considered. First, the approximation is made using levels, taken from the samples during hemodialysis, but as shown below, the amount of drug removal with hemodialysis is negligible, so this should not affect the measured levels. Second, due to the high volume of

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Table 2. Therapeutic and experimental salbutamol/albuterol levels from the literature

Author	Dose	Observed levels	Max levels, ng/mL	Adverse effects
Goldstein et al. [4]	/	10–20 ng/mL	20	Not available
Morgan et al. [5]	Loading 400 μg + infusion 10 μg/min (total 1,600 μg)	10 ng/mL	10	Increased heart rate
Fairfax et al. [6] – observational	300–2,000 μg inh. (mean 900 μg)	3.7 + SD 2.1 ng/mL	3.7 + 2.1	Hand tremor
Fairfax et al. [6] – experiment	8 μg/kg/h I.V. (15 h; approx. 8,520 μg total dose)*	20.3 +3.2 (SD) ng/mL	20.3 + 3.2	Heart rate (mean±SD) 97.4±10.7/min, palpitation, shaking of the hands, mild euphoria
Walker et al. [7]	8mgP.O.	100 nmol/L (up to)	23.93	Not reported
Janson [8]	5 μg/kg I.V.	59 nmol/L	14.1	Tachycardia, hypokalemia, palpitations, tremor
Janson [8]	150 μg/kg inh	30 nmol/L	7.2	Same
Lewis et al. [9]	89±83 (SD) mg P.O.	18–449 ng/mL	449	Hypokalemia, tachycardia, tremor
Schulz et al. [10]	/	4–20 ng/mL	160	/
Our study	5,000 μg I.V.	//	32**	Tachycardia 170–190/min Hypokalemia Hyperlactatemia
Our study	5,000 μg I.V.	10 μg/L***	10	Same

*Total dose not reported, approximated calculation using a body weight 71 kg [11].
**Calculated using volume of distribution by Morgan et al. [6].
***After 190 min.

distribution, it is highly likely that in the (unobserved) quick distribution phase immediately after administration, plasma levels were very much higher. For example, if the drug was initially distributed only to the plasma compartment (body weight * 0.065 * (1-hematocrit) [14], 3,300 mL), the concentration would reach or 1,520 ng/mL. This is well above the level associated with potential toxicity (30 ng/mL) and above potentially lethal level (>160 ng/mL) [1].

Table 3 shows salbutamol plasma levels after admission: the levels were diminishing during hemodialysis and there was a clear and consistent removal of the drug from the blood. However, due to low serum levels, the total removed amount was small. This is in concordance with a high volume of distribution. However, serum levels taken 780 and 1,140 min after hemodialysis commencement (970 and 1,330 min after intoxication) show a much slower clearance. This indicates that the distribution and elimination phase were still ongoing during hemodialysis.

Table 3. Salbutamol levels, blood flow, and amount of drug cleared from plasma during hemodialysis

Time, min	Elapsed time, min	Pre-filter level, mg/L	Post-filter level, mg/L	Absolute difference, mg/L	Relative reduction (pre-post)/pre	Blood flow, mL/min	Lower estimate of amount removed with dialysis, mg*
0		0.010	/	/	/	/	/
30	30	0.008	0.004	0.004	0.5	300	0.036
60	30	0.007	0.004	0.003	0.43	280	0.0252
90	30	0.006	0.003	0.003	0.5	260	0.0234
180	90	0.005	0.003	0.002	0.4	270	0.0486
210	30	/	/	/	/	270	0.0081**
Total removal, mg							0.141
780		0.004					
1,140		0.003					

*The amount is estimated based on available data.

**The amount removed is estimated using an estimated (low end) pre-filter level 0.004, post-filter level 0.003; i.e., absolute difference 0.001 and relative reduction 0.25.

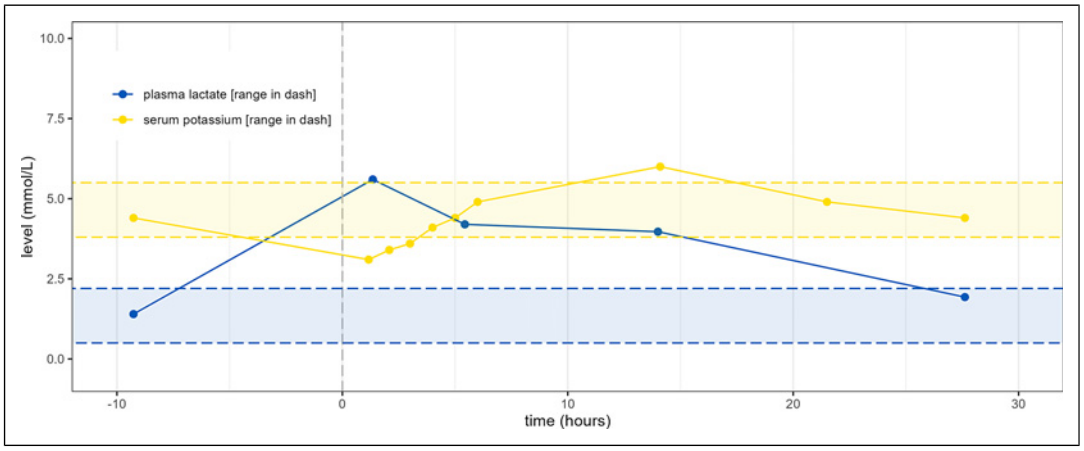

Fig. 2. Lactate and potassium blood levels before and after inadvertent salbutamol administration. Normal range for serum potassium and plasma lactate levels is indicated in colored area.

In accordance with the available data, the main adverse effect of elevated salbutamol blood levels is tachycardia with palpitations and hand tremor. However, the available record of exceptionally high salbutamol levels [9] describes sudden tachycardia with an intravenous "bolus" dose.

Although salbutamol can cause elevated serum lactate levels [11, 15, 16], we should evaluate lactate levels in our case with caution. If we consider normal lactate level before intoxication and very rapid increase before that, we can speculate that, at least partly, the hyperlactatemia due to salbutamol was observed (type B). However, the clinical course indicates that the patient was then in a state of cardiogenic and septic shock; therefore, low tissue perfusion was the cause of ongoing lactate acidosis (type A). This explains the prolonged time to serum lactate normalization (see Fig. 2).

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We were able to include precise plasma salbutamol measurements in an acute setting, and the data is in concordance with published data. However, this is a single-patient report with multiple concomitant conditions. The main limitation of this report is that the results are not directly applicable to all patients.

Our case of inadvertent intoxication with salbutamol presented with an extremely high heart rate and worsening of clinical condition. Therapeutic interventions with intravenous antagonist (landiolol) and cardioversion were ineffective. Therefore, despite known poor dialyzability, the decision to commence hemodialysis was taken. Hemodialysis procedure is not itself without risk; the risk is greater in older patients [17], patients with heart disease [18] and patients with liver disease [19]; therefore, hemodialysis in such a setting should be considered with great caution and utmost care. The almost immediate response with the resolution of tachyarrhythmia 30 min after onset of hemodialysis was seen as a direct consequence of ongoing hemodialysis procedure. However, the data presented in this case report does not support this.

- The total amount of drug cleared with hemodialysis in 3.5-h period is small (0.141 mg, 2.8% of total dose) – this is in line with published guidelines, that suggest low albuterol/salbutamol dialyzability [3].
- Although there could be "immediate" effect of an undiluted drug after bolus administration, the large distribution volume is in line with quick removal of the drug from plasma.
- The measured levels are within or at the high end of predicted therapeutic levels.
- The pharmacokinetic profile suggests that an observed half-life (3.1 h) during hemodialysis is completely comparable to observed half-lives in therapeutic settings (2.8–6 h).
- As a therapy for hyperkalemia, even high salbutamol levels do not seem to be highly effective; only moderate hypokalemic effect was present in our patient.

The observed favorable clinical benefit associated with dialysis may be fortuitous; by the time tachycardia was resolved, serum levels fell from 10 to 8 ng/mL; however, hemodialysis procedure removed only approximately 0.036 mg of salbutamol. This observational kinetic study is highlighting potential bias toward positive clinical outcomes and unproven ("salvage") therapies.

Statement of Ethics

Ethical approval is not required for this study in accordance with local or national guidelines. Written informed consent from the patient could not be obtained due to patient's passing away. Written informed consent was obtained from the patient's next of kin to perform the additional laboratory studies, for publication of the details of their medical case, and any accompanying images.

Conflict of Interest Statement

The authors declare that they have no competing interests.

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Author Contributions

N.B. and A.J. collected, analyzed, and interpreted the data; wrote the draft manuscript; and are recognized as equal first authors. D.G. designed the study and was a major contributor in writing the discussion. S.K.Z. was a major contributor in writing the case presentation and the discussion. All authors read and approved the final manuscript.

Data Availability Statement

All relevant data generated or analyzed during this study are included in this published article. Further inquiries can be directed to the corresponding author.

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