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Prolonged Inhalation Sedation in the Intensive Care Unit Using the AnaConDa Device (Case Reports)

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Summary

This report describes two clinical cases involving prolonged inhalation sedation using the AnaConDa device in the ICU. Both patients achieved and maintained adequate sedation levels throughout the treatment period. No significant adverse cardiovascular effects were observed.

Keywords: inhalation sedation; AnaConDa; prolonged sedation; sepsis

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Introduction

The challenge of sedation in the intensive care unit (ICU) remains a pressing issue despite the availability of several intravenous sedative agents [1, 2]. Each of these agents has potential drawbacks that may adversely affect patient outcomes. Nevertheless, a growing body of evidence, although still limited, supports the routine use of the AnaConDa device for inhaled sedation [3, 4]. The organ-protective properties of sevoflurane and the absence of reported tachyphylaxis suggest its potential efficacy in the ICU setting [5].

Case Report No.1

A 26-year-old man with a height of 186 cm and a weight of 77 kg (BMI 22.3 kg/m²) was admitted to the ICU with altered consciousness following substance use.

Emergency medical services (EMS) reported that the patient had experienced a generalized seizure prior to arrival at the hospital, which was effectively treated with 10 mg of diazepam. Due to the ambiguous clinical presentation of intoxication, no antidotal therapy was administered.

Urine toxicology revealed cannabinoids, cocaine, methadone and its metabolite (2-ethylidene-1.5-dimethyl-3.3-diphenylpyrrolidine), and alpha-PVP.

On admission, the patient had a Glasgow Coma Scale (GCS) score of 12, indicating severe stupor, with episodes of psychomotor agitation and central tachypnea due to intoxication syndrome, with a respiratory rate (RR) of 30 breaths per minute.

CT scan of the brain: Cystic gliotic lesions in the right frontal lobe.

Chest CT scan: Inflammatory changes in the lower lobe segments of the right lung, consistent with aspiration pneumonia secondary to decreased consciousness.

Laboratory tests showed significant abnormalities:

- Hemoconcentration: Hematocrit 49%, hemoglobin 163 g/dL.
- Mixed acidosis (arterial blood gas):
  - pH 6.97
  - PO_2 125 mmHg,
  - P/F ratio 357 mmHg
  - PCO_2 85 mmHg
  - HCO_3 19.6 mmol/L

- Base Excess (BE) –16.1 mmol/L
- Lactate 7.3 mmol/L

- Hyperglycemia: glucose 20.5 mmol/L.
- Systemic inflammatory markers:
  - Leukocytes 18.42 × 10^9/L
  - C-reactive protein (CRP) 62.2 mg/L
  - Procalcitonin (PCT) 3.5 ng/mL.

Due to marked psychomotor agitation, impaired consciousness, and tachypnea associated with the intoxication syndrome, the patient required pharmacologic sedation to prevent cerebral complications. Endotracheal intubation was performed, and the patient was placed on mechanical ventilation (SIMV mode) with the following parameters:

- Tidal volume (V_t) 500 mL
- Inspiratory time (T_{insp}) 1.35 sec
- Respiration frequency (F) 16/min
- Positive end-expiratory pressure (PEEP) 8 cm H_2O
- Fraction of inspired oxygen (FiO_2) 40%
- Assisted spontaneous breathing pressure (P_{asb}) 16 cm H_2O

With these settings, the monitoring parameters were as follows:

- Peripheral oxygen saturation (SpO_2) 99%.
- Minute ventilation (MV) 8 L/min
- End tidal CO_2 ($EtCO_2$) 37 mmHg.

Intravenous sedation with propofol was initiated at an initial dose of 2 mg/kg/h, achieving a Richmond Agitation-Sedation Scale (RASS) score of –4.

Despite the aggressive therapy, the patient showed hemodynamic instability requiring vasopressor and inotropic support with norepinephrine (initial dose 0.6 µg/kg/min) and epinephrine (0.4 µg/kg/min), with a calculated vasopressor-inotropic score (VIS) of 100 points [6]. Continuous invasive blood pressure monitoring was started.

The patient was started on antimicrobial therapy with ampicillin-sulbactam (3 g three times daily), fluid resuscitation (40 mL/kg/day), gastroprotective therapy with omeprazole (40 mg twice daily), and anticoagulation with nadroparin (2.850 IU anti-Xa twice daily).

With correction of blood gas and metabolic parameters, the following improvements were observed:

- pH 7.470
- PCO_2 36.9 mmHg
- P/F ratio 312 mmHg
- HCO_3 26.5 mmol/L
- BE 3.4 mmol/L
- Glucose 6.3 mmol/L
- Lactate 2.4 mmol/L

Consciousness fully recovered; however, as sedation was tapered, the patient developed severe anxiety, psychomotor agitation, tachycardia, and ventilator asynchrony. Because of these complications, continued mechanical ventilation was deemed necessary.

To achieve adequate depth of sedation, propofol was increased to > 4 mg/kg/h [5]. Due to this high requirement, inhaled sedation with sevoflurane was initiated using the AnaConDa device at a rate of 5 mL/h, adjusted based on gas analyzer readings to maintain a target minimum alveolar concentration (MAC) of 0.5–1.0% vol.

The patient remained on SIMV mode ventilation with the following parameters:

- V_t 550 mL
- T_{insp} 1.4 sec
- F 16/min
- PEEP 6 cm H_2O
- FiO_2 40%
- P_{asb} 12 cm H_2O

With these settings, the monitoring parameters were as follows

- SpO_2 96%.
- MV 8.8 L/min
- $EtCO_2$ 40 mmHg.

During the first 12 hours, the patient remained hemodynamically unstable and dependent on sympathomimetic support. However, within 24 hours of initiation of inhaled sedation, the signs of acute cardiovascular failure subsided and vasopressor therapy was discontinued. The patient regained full consciousness.

With normalization of acid-base balance and arterial blood gas parameters (pH 7.37, PCO_2 37.3 mmHg, P/F ratio 353 mmHg, HCO_3 21.1 mmol/L, BE 3.1 mmol/L, glucose 6.8 mmol/L, lactate 1.9 mmol/L), along with restored alertness, adequate muscle tone, and spontaneous breathing, the patient was successfully extubated (Table 1).

Six hours after extubation, the patient was transferred to the appropriate clinical unit and discharged from the hospital the following day after refusing further treatment.

Case Report No.2

A 43-year-old female patient (height 170 cm, weight 68 kg) was admitted to the ICU with a clinical presentation of septic shock (hypotension with BP 70/40 mmHg, HR 125/min, severe tachypnea) due to obstructive pyelonephritis. Laboratory findings showed signs of systemic inflammation (leucocyte count 20.24 × 10^9/L, platelet count 78 × 10^9/L, CRP 229.4 mg/L, PCT 10.6 ng/mL) and metabolic acidosis (pH 7.29, BE -9.1 mmol/L, SvO_2 53%, lactate 7.1 mmol/L).

Emergency right ureteral stenting was performed and comprehensive intensive therapy was initiated, including vasopressor support (norepinephrine at 2.3 µg/kg/min, VIS score 230), fluid resuscitation (30 mL/kg), antimicrobial therapy (cefepime+sulbactam 4 g twice daily + metronidazole 500 mg three times daily + fosfomycin 4 g three times daily), and noninvasive lung ventilation (NILV).

Table 1. Changes in instrumental and laboratory data of patient No. 1.

Parameter	Values at different study steps					
	prior to MLV	MLV, sedation				after MLV (extubation)
		Intravenous (propofol)		Inhalation (sevoflurane)		
		12 h	24 h	12 h	24 h	
P/F ratio, mm Hg	357	377	342	362	451	353
MAP, mm Hg	87	51	79	68	104	92
Norepinephrine, μg/kg/min	—	0.6	0.6	0.6	0.25	—
Epinephrine, μg/kg/min	—	0.4	0.1	0.08	—	—
pH(a)	6.97	7.13	7.34	7.47	7.38	7.37
PCO_2, mm Hg	85	76	47	36	34	37
Lactate, mmol/L	7.3	1.3	1.6	2.4	1.7	1.9
PEEP, cm H_2O	—	8	7	6	6	—
EF, %	62	62	62	62	62	62

Note. For Tables 1–3: MLV — mechanical lung ventilation; MAP — mean arterial pressure; pH(a) — arterial blood pH; PEEP — positive end-expiratory pressure; EF — ejection fraction.

However, the patient's condition continued to deteriorate: persistent hypotension refractory to sympathomimetics, tachypnea of central origin, and episodes of confusion alternating with psychomotor agitation were observed. These symptoms were interpreted as manifestations of hypoxic encephalopathy.

It was decided to initiate mechanical ventilation. The patient underwent endotracheal intubation and mechanical ventilation was performed with a Drager XL ventilator in SIMV mode: V_t 480 mL, T_{insp} 1.4 seconds, F 15 breaths/minute, PEEP 8 cm H_2O, FiO_2 60%, and Pasb 16 cm H_2O. With these settings, monitoring parameters were as follows: SpO_2 99%, MV 7.2 L/min, and $EtCO_2$ 32 mmHg. Given these parameters, sedation was necessary. Initial arterial blood gas analysis at the start of mechanical ventilation showed: pH 7.209, PaO_2 108 mmHg, $PaCO_2$ 37 mmHg, bicarbonate (HCO_3) 14.4 mmol/L, base excess (BE) –13.5 mmol/L, and lactate 4.0 mmol/L.

Due to persistent hypotension, invasive hemodynamic monitoring was performed using the PICCO method with the following parameters: cardiac index (CI) 3.34 L/min/m², stroke volume index (SVI) 28 mL/m², systemic vascular resistance index (SVRI) 1852 dyn×s×cm^{-5}×m², cardiac power index (CPI) 0.66 W/m², global end-diastolic volume (GEDV) 686 mL/m², extravascular lung water index (ELWI) 14 mL/kg, global ejection fraction (GEF) 16%, and cardiac function index (CFI) 5.2 min^{-1}. Inotropic support was initiated with dobutamine at 5 μg/kg/min.

Due to the presence of acute kidney injury with preserved urine output and lactate acidosis, a combined extracorporeal therapy approach was used within the first 24 hours of hospitalization. This included hemodiafiltration with an EMIC2 filter and hemoadsorption with the Cytosorb system.

To maintain adequate sedation, a multidrug protocol was required that included high doses of ketamine, midazolam, fentanyl, and dexmedetomidine (Dexdor). The dosing regimen included dexmedetomidine at 0.8 μg/kg/h, ketamine at 150 mg/h, midazolam at 7.5 mg/h, and fentanyl at 0.2 mg/h. Based on these requirements, inhalation sedation with sevoflurane was initiated using the AnaConDa system.

Sevoflurane sedation was monitored by gas analyzer readings and continued for a total of 72 hours (Table 2). During this time, extracorporeal therapy was discontinued and both vasopressor and inotropic support were gradually tapered. The patient's VIS score decreased over three days from 230 to 87 and finally to 30. Laboratory tests showed normalization of acid-base balance and blood gas levels (Table 3), and signs of systemic inflammation resolved.

Table 2. Central hemodynamic parameters of patient No. 2.

Parameters	Values during sedation				
	Intravenous	Inhalation (Sevoflurane)			
		12 h	24 h	48 h	72 h
MAP, mm Hg	85	92	94	89	85
HR, bpm	120	110	99	107	95
Norepinephrine, μg/kg/min	1.4	0.87	0.5	0.3	0.15
Dobutamine, μg/kg/min	5	3.57	2.0	1.19	2.0
CI, L/min/m²	3.34	3.93	3.86	3.69	6.04
SVRI, dyn×s×cm^{-5}×m²	1852	1766	1696	2043	952
ELWI, mL/kg	14	16	16	16	14
GEF, %	16	18	19	23	27
GEDV, mL/m²	686	810	869	790	870
PEEP, cm H_2O	8	10	9	8	8

Note. HR — heart rate; CI — cardiac index; SVRI — systemic vascular resistance index; ELWI — extravascular lung water index; GEF — global ejection fraction; GEDV — global end-diastolic volume.

Table 3. Changes in instrumental and laboratory parameters of patient No. 2.

Parameter	Values during study steps					
	MLV, sedation					after MLV (extubation)
	Intravenous	Inhalation (Sevoflurane)				
		12 h	24 h	48 h	72 h	
P/F ratio, mm Hg	262	330	285	255	332	383
pH (a)	7.43	7.47	7.49	7.42	7.48	7.54
PCO_2, mm Hg	40	37	38	42	40	34
Lactate, mmol/L	3.9	2.0	2.34	2.5	0.85	1.23
BE, mmol/L	2.1	3.2	0.4	2.5	5.6	4.3
HCO_3, mmol/L	26.5	26.9	24.7	27.1	29.5	27.3
SvO_2, %	87	86	87	85	88	89
Leucocytes, 10^9/L	16.3	13.6	9.3	13.4	16.2	11.5
Platelets, 10^9/L	55	43	23	77	60	64

The patient's level of consciousness outside of sedation was assessed daily using the FOUR score. The score was 12 on the first day, 14 on the second day, and 16 on the third day. When the patient regained full consciousness, full muscle tone, and adequate spontaneous respiration, she was successfully extubated.

Subsequent rehabilitation in the intensive care unit was uneventful. After three days, the patient was transferred to a specialized unit and later discharged from the hospital.

Discussion

Despite advances in surgical techniques for the treatment of patients with acute abdominal pathology [7], the availability of a broad spectrum of antibacterial agents [8], the development of extracorporeal therapies [9], and improvements in early diagnosis [10], sepsis remains a critical problem in modern clinical practice [11]. The high mortality, complexity and prolonged duration of treatment, including prolonged stays in intensive care units, necessitate a continuous search for new strategies to mitigate the consequences of sepsis and septic shock.

A particular challenge is that patients with septic shock often require prolonged mechanical ventilation. This in turn is associated with several secondary complications, including ventilator-associated pneumonia, the need for prolonged sedation, and subsequent difficulties in early patient mobilization.

Traditional sedatives in the ICU include propofol and midazolam [12–14]. However, prolonged use of these drugs is associated with several challenges that may further complicate the already complex prognosis in this patient population.

A major concern is propofol infusion syndrome [15, 16]. Continuous administration at doses greater than 4 mg/kg/h for more than 72 hours can alter mitochondrial respiratory chain function, leading to impaired oxidative phosphorylation and accumulation of anaerobic metabolic by-products [15].

Midazolam, on the other hand, is associated with rapid development of tolerance [17]. In cases requiring prolonged sedation, this may result in reaching the maximum tolerated dose relatively quickly without achieving adequate sedation, even in combination with opioid analgesics.

Dexmedetomidine, a selective α_2-adrenergic receptor agonist, has become an integral part of clinical practice, demonstrating efficacy as a sedative in a wide range of conditions. Recent experimental studies [18] suggest that dexmedetomidine may also have organ-protective effects on the lungs in septic shock. In addition, several studies since 2010 [19] and continuing to the present [20, 21] have reported a significant reduction in vasopressor requirements in patients who received dexmedetomidine sedation from the onset of illness. However, similar to midazolam, prolonged use of dexmedetomidine is associated with the development of tachyphylaxis.

For a long time, inhalation sedation was not widely used in the ICU due to technical challenges. However, in 2012 (and in Europe since the early 2000s), the AnaConDa (The Anaesthetic Conserving Device) was certified in Russia, allowing the safe and effective use of inhalational anesthetics in critical care. This advancement allowed clinical application of the previously established organ-protective properties of sevoflurane [21–25] in patients with septic shock, with the goal of reducing multiple organ dysfunction.

Concerns regarding the hemodynamic effects of sevoflurane in patients with circulatory failure due to septic shock have not been substantiated by numerous studies [22, 23, 26]. The only retrospective study evaluating the effect of this sedation technique on mortality in septic shock clearly demonstrated the superiority of sevoflurane over intravenous sedation, with a 20% reduction in both in-hospital and one-year mortality [27].

Another study [28] demonstrated a significant improvement in 7-day overall survival in mice (83.3%) following administration of 1% sevoflurane for 6 hours after induction of abdominal sepsis compared to untreated mice (16.6% in the control group).

Conclusion

The presented clinical observations indicate that prolonged inhalation sedation in the ICU using the AnaConDa device in patients with sepsis has no adverse effect on the cardiovascular system (clinical case 2) and allows to achieve adequate sedation levels without the development of tachyphylaxis. This approach helps to avoid dose escalation in patients requiring multimodal sedation with intravenous anesthetics (clinical cases 1 and 2).

Clearly, prospective randomized clinical trials comparing various sedation strategies are required to gain a full understanding of all aspects of inhalation sedation in the ICU.

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