

A Rescue Therapy in a Case of Severe Anaphylaxis in a Brittle Asthmatic with Obstructive Sleep Apnoea and Restrictive Lung Disease Overlap in a Critical Care Setting

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Abstract

A 42-year-old man, known brittle asthmatic with restrictive lung and obstructive sleep apnoea admitted with severe anaphylaxis to an intensive care unit following an admission for elective lymph node biopsy for a suspected recurrence of malignant melanoma. His anaphylaxis, underlying co-morbidities and lung disease posed a significant challenge to his ventilation and subsequent weaning from invasive ventilation.

Keywords: Anaphylaxis; Brittle Asthmatic; Obstructive Sleep Apnoea

Abbreviations

ICU: Intensive Care Unit; PEEP: Positive End Expiratory Pressure; RAAS: Richmond Agitation-Sedation Scale; BMI: Basal Metabolic Index

Introduction

A 42-year-old man admitted electively for an excision lymph node biopsy of a suspected melanoma in the groin in February 2017. His past medical history included a brittle asthma with regular bronchodilators and leukotriene receptor antagonist, Montelukast. He underwent an excision biopsy of a malignant melanoma from his right knee in December 2016. He was treated with mirtazapine for his depression. He was not known to have any drug allergies. His regular medications included beclomethasone Dipropionate 200 inhaler, Mirtazapine 30 mg, Prednisolone 5 mg, salbutamol 100 micrograms per metered Inhaler and zopiclone 3.75 mg once a day.

Pre-operative anaesthetic review was done on the morning for elective block dissection of groin lymph nodes at 7.30am. Whilst in theatre complex, Induction and intubation were uneventful in Anaesthetic room. Once transferred onto operating table, whilst being prepped, four minutes after intravenous Co-amoxiclav (amoxicillin and clavulanic acid) administration, he couldn't be ventilated and developed severe hypoxia.

Management

Anesthetist re-intubated patient to exclude an obstruction to endotracheal tube (Grade 1 view). As patient failed to improve, anaphylaxis treatment was initiated. He was administered intravenous adrenaline increments boluses followed by an adrenaline infusion. Chlorpheniramine (antihistaminic), intravenous steroids and fluids were administered. A portable chest X-ray confirmed well positioned endotracheal tube.

His tidal volumes started to improve. However, he remained adrenaline dependent. His initial serum tryptase levels were requested along with routine investigations. His serum lactate levels increased to 9. (Figure 1) He was transferred to intensive care unit (ICU) for supportive therapy. He clinically had significant restrictive lung disease and obstructive sleep apnoea secondary to his high BMI.

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Flow Sheet	13/02/2017	18:00	20:00	22:00	14/02/2017	00:00	02:00	04:00	06:00	08:00	10:00
Days in ICU		1	1	1	2	2	2	2	2	2	2
Heart Rate		100	83	78	73	67	69	70	85	72	
ABP		100/64/76	120/66/84	100/57/71	102/63/76	113/64/80	113/63/80	133/73/93	126/75/92	125/73/90	
Cardiac Rhythm		NSR	NSR	NSR	NSR	NSR	NSR	NSR	NSR	NSR	
Temp1 Central Site											
Temp2 Other Site		35.2/Tym...	35.6/Tym...	35.8/Tym...	35.8/Tym...			36.3/Tym...	36.2/Tym...	36.2/Tym...	
Cardiac Ectopics											
CVP			17		20			22		13	
SpO2		98	100	96	100	100	100	99	100	100	
Respiratory Rate (ECG)		0	15	20	15	7	0	3	13	12	
Oxygen											
FiO2 Delivered											
Insp O2			40	40	28	28	28	28	28	85	30
Ventilator Mode			Mode SPN...	Mode PC...	Mode PC...	Mode PC...	Mode PC...	Mode PC...	Mode PC...	Mode PC...	Mode PC...
H+ - ABG		57	70	47	44	51	47	79	49	49	
pH - ABG		7.243	7.153	7.326	7.355	7.291	7.332	7.105	7.308		
PaO2 - ABG		23.12	15.45	18.14	15.01	11.68	14.61	28.99	15.67		
PCO2 - ABG		5.76	8.76	5.71	6.04	6.93	5.93	11.14	6.18		
BE(B) - ABG		-8.7	-6.9	-4	-1	-2.4	-2.8	-5.2	-3.6		
Hb - ABG		114	116	111	112	110	107	111	107		
Lactate - ABG [0.5-2.0]		9.01	6.26	4.44	2.58	2.54	2.11	1.62	2.08		
Capillary Refill Time								< 2 secs			
Glucose (StatStrip)			7.7		6.6	5.5		7.3	8.0	6.5	7.5
Acute Pain Score			No pain	No pain	No pain	No pain	No pain	No pain	No pain	No pain	No pain
BMI / MUST: Weekly Asses...		0 weekly...									
Weight - Kg (Daily)			120	120	120	120	120	120	120	120	
Weight - Kg (Ideal)											
Ideal Body Wt kg. (calculati...			66	66	66	66	66	66	66	66	
Urine: Catheter			120	105	95(320)	90	155	100	225	55(625)	
SOFA Score			4			4		4		4	

Figure 1: Demonstrates ICU admission arterial blood gases with high CO₂, lactate 9.01. Patient’s actual weight and ideal weight. Increasing FiO₂ requirement 40% to 28% and increasing to 85% correlating with second surge of severe anaphylaxis reaction.

On ICU, patient was administered loading dose of aminophylline, followed by maintenance dose. Despite optimal treatment, his tidal volumes remained less than 150 ml and was not synchronizing with ventilator. Patient was given a bolus of paralytic agent to aid ventilator dyschrony with good effect.

Patient’s intrinsic PEEP was measured to exclude air trapping. As the intrinsic PEEP was 5, external PEEP was decreased to 1 (Figure 8, 9).

However, patient’s tidal volumes remained fluctuating once paralytic agent effect wore off. His serum tryptase levels were 69.1 at onset of event and peak levels were 92.8 at 8.00am day two of intensive care unit stay respectively. This coincided with second episode of severe anaphylaxis reaction associated with increased FiO₂ requirement and change in lung compliance (Figure 2). His other baseline investigation demonstrated an elevated procalcitonin (Figure 3).

However, once patient sedation was weaned to RAAS -2 to 0 scores, he started to struggle with his ventilation and failed to synchronise with ventilator.

Inspiration vs Expiration ratio were increased to improve shifting of carbon dioxide from 1: 1.9 to 1: 5.2. Suddenly, his tidal volumes dropped to less than 100 ml (Figure 2). Ketamine was started at 8.59 hours on day 2, as patient had worsening bronchospasm despite being on optimal anaphylaxis therapy, aminophylline infusion and regular bronchodilators (Figure 4-6).

Once ketamine infusion was started, his vasopressors were weaned off. He was extubated after five hours on ketamine infusion (Figure 7). Once extubated, his ketamine infusion was discontinued. His tidal volumes improved to > 600 ml (Figure 8,9). His aminophylline infusion was weaned over next 24 hours.

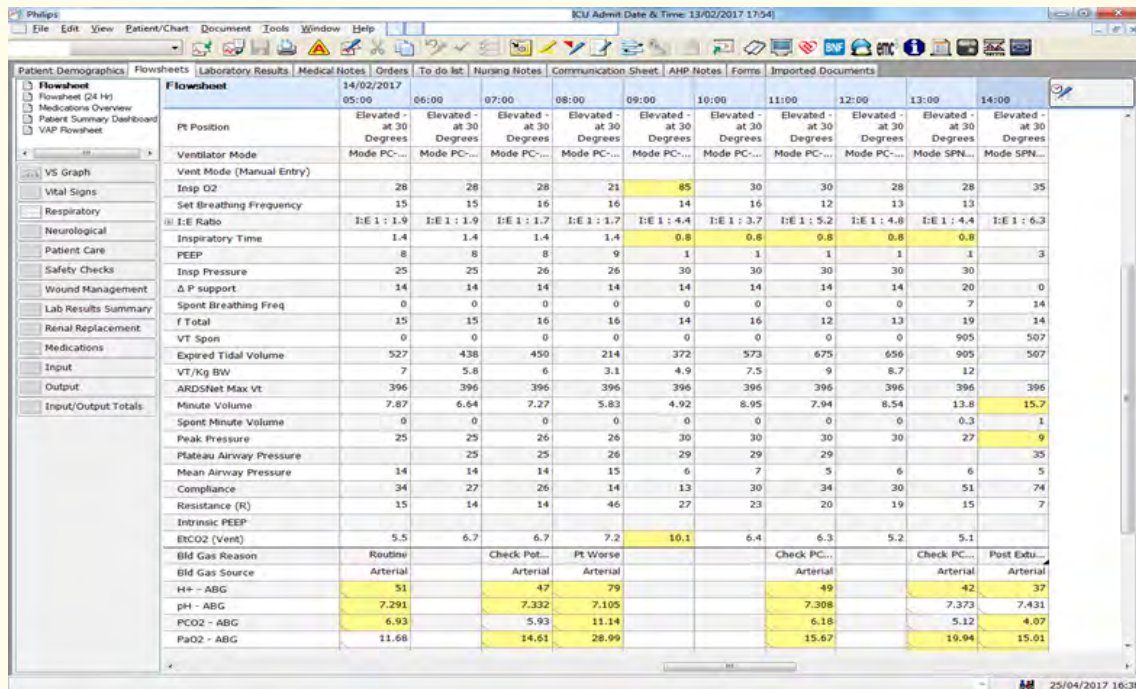


Figure 2: Demonstrates I: E ratio reversal to correct pCO₂ leading to respiratory acidosis component due to bronchospasm during second episode of severe anaphylaxis reaction.

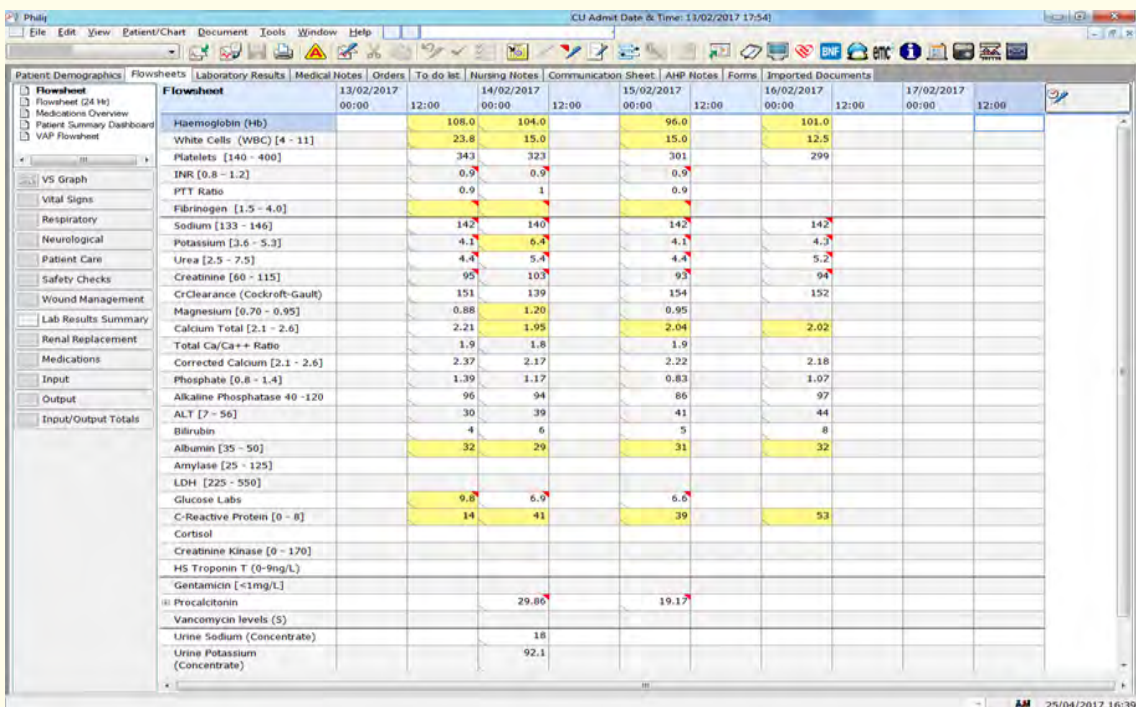


Figure 3: Demonstrates flowsheet of routine investigations of this patient including procalcitonin during his ICU admission following anaphylaxis.

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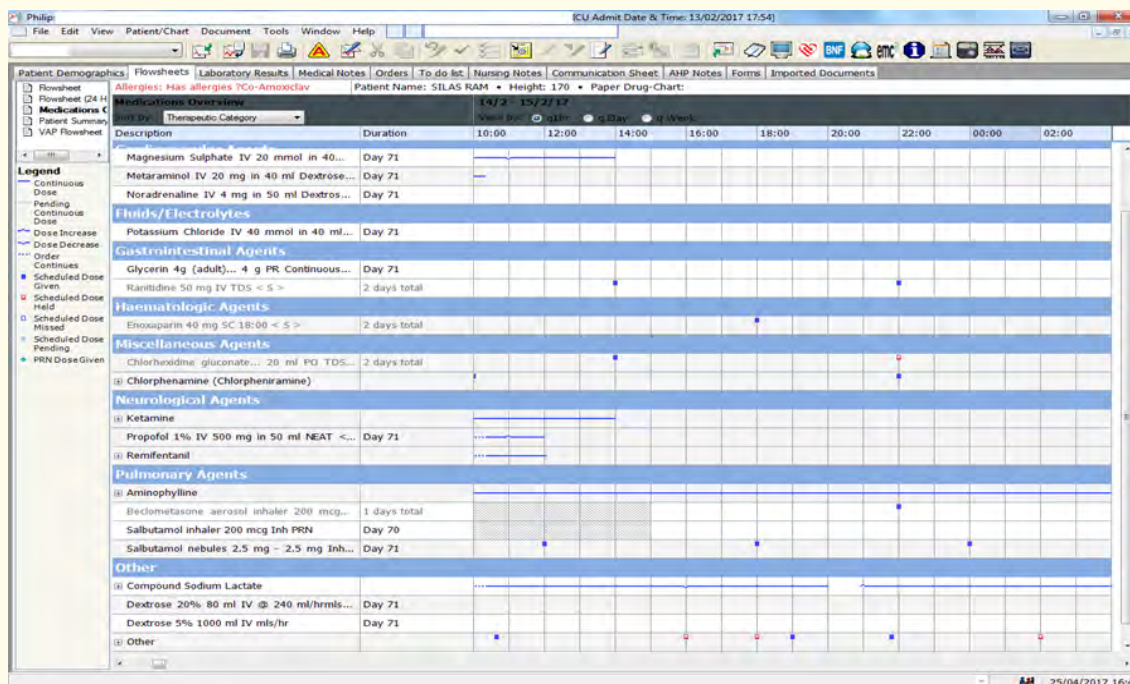


Figure 4: Demonstrates flowsheet of drug infusions of this patient including ketamine, aminophylline, Propofol and Remifentanyl on day 2 of his ICU admission following anaphylaxis. Note: Metaraminol infusion was successfully weaned off after one hour of starting ketamine infusion.

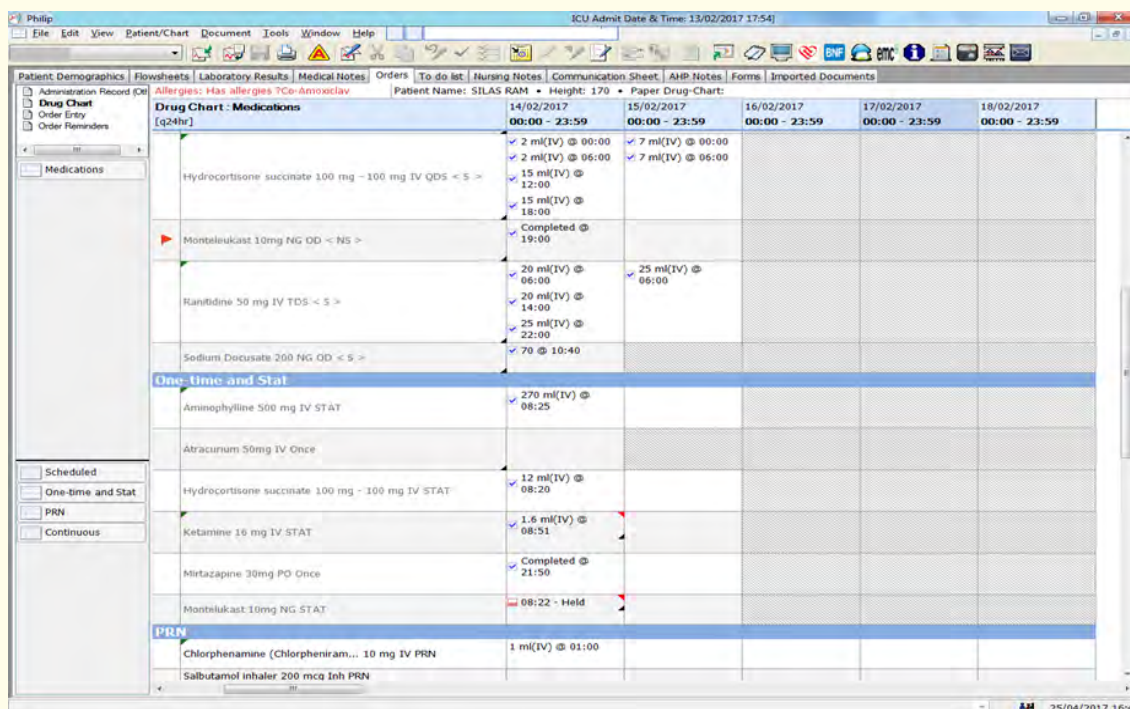


Figure 5: Demonstrates electronic prescription sheet reflecting a stat dose time of ketamine and rest of his medications on day 2 of ICU.

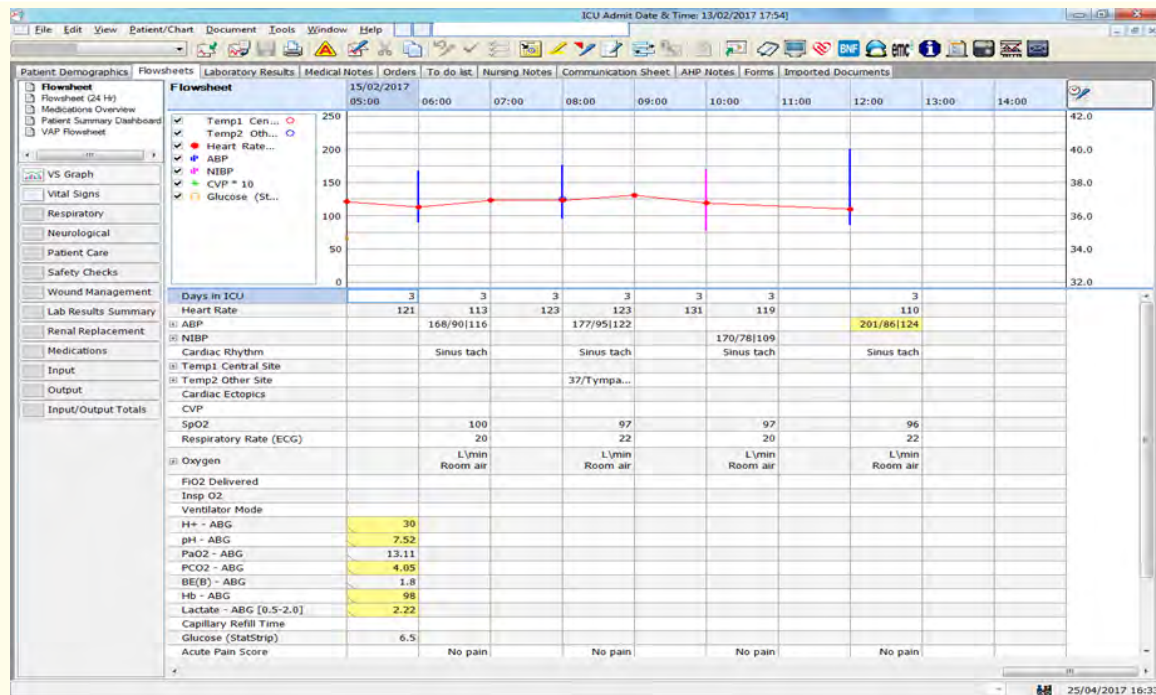


Figure 6: Demonstrates routine observations and arterial blood gas following his extubating, on day 3 of ICU before discharge to respiratory ward.

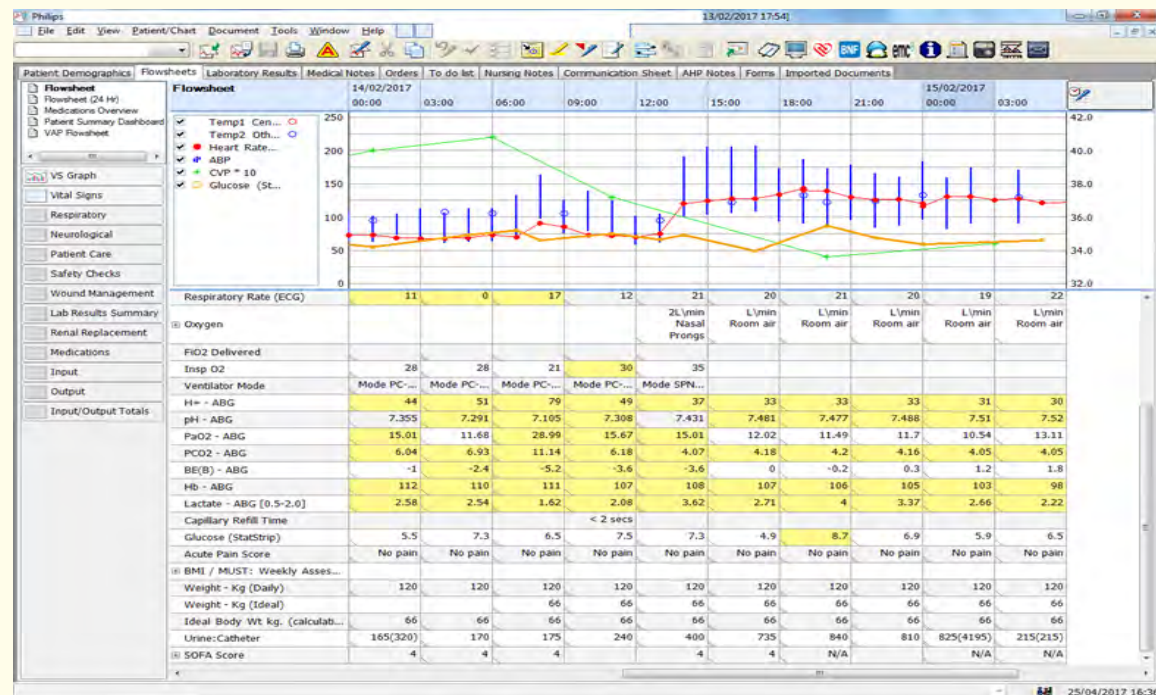


Figure 7: Demonstrates patient weaned off from invasive ventilation whilst on low dose ketamine and aminophylline infusion.

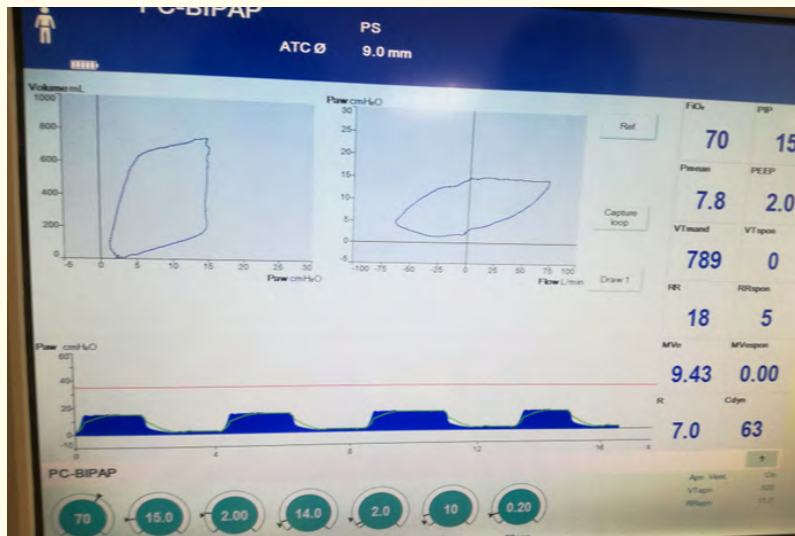


Figure 8: Demonstrates the shape of flow loops at PEEP 2 and volume loops whilst on ketamine and aminophylline infusion.

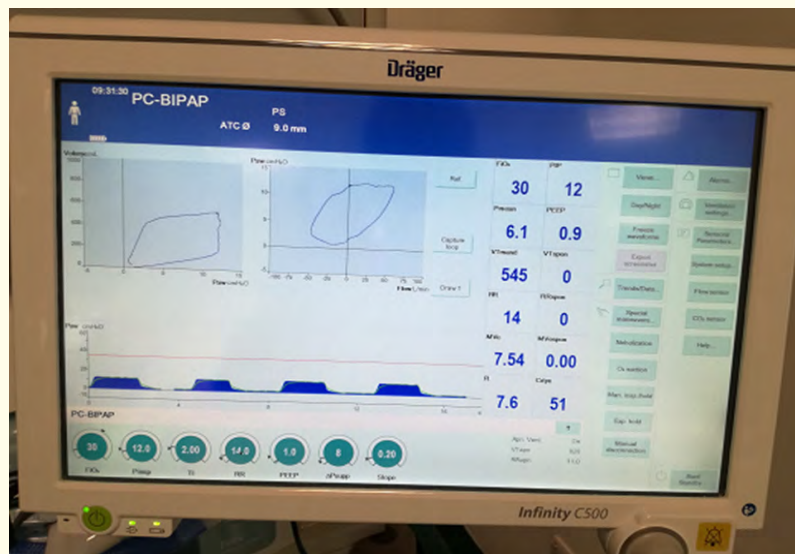


Figure 9: Demonstrates the shape of flow loops at PEEP 1 and volume loops whilst on ketamine and aminophylline infusion.

Following extubation, he was stable for next twenty-four hours (Figure 10). He was discharged to a Respiratory ward. His Spirometry - FEV1: 1.66 (44%), FVC: 2.03 (44%), FEV1/FVC = 81%, PEF: 390 (72%). He was discharged home with a regular follow up by an immunologist. After 3 weeks, he was electively readmitted for a lymph node biopsy, under local and spinal anaesthetic agents.

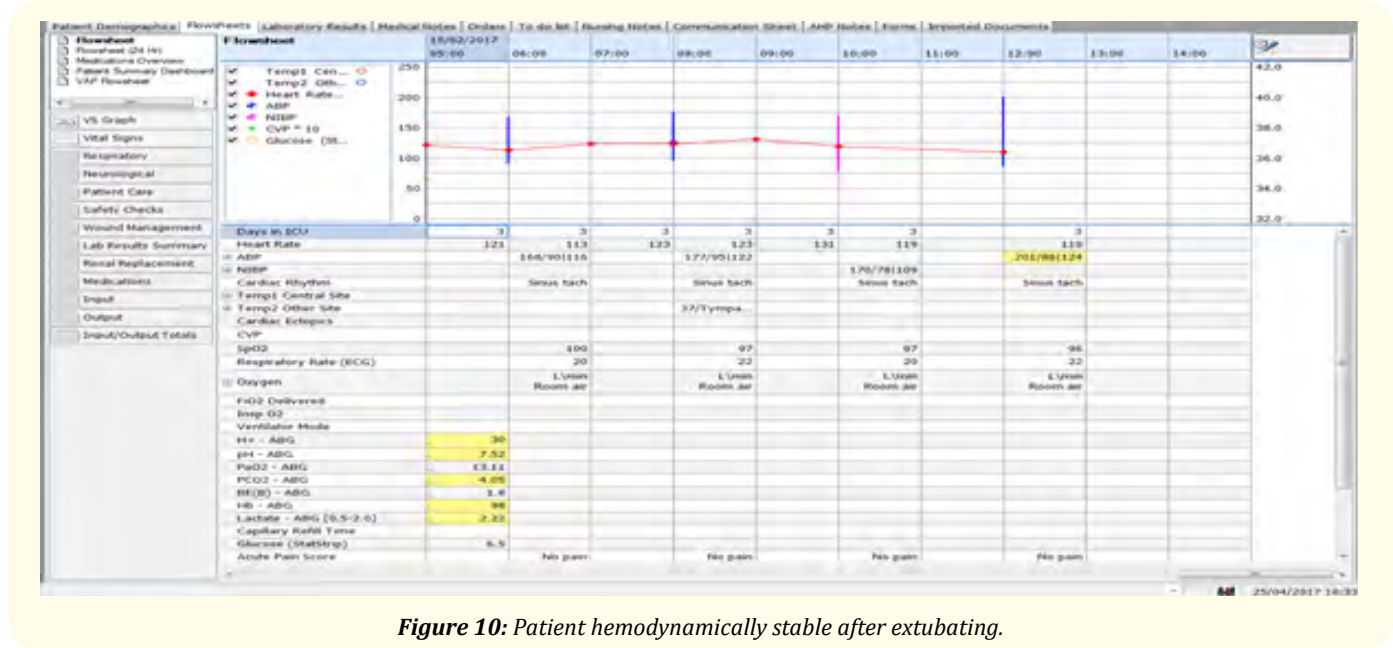


Figure 10: Patient hemodynamically stable after extubating.

Discussion

Anaphylaxis is a sudden onset of severe, multisystem allergic reaction after contact with an allergen [7]. Urticaria or angioedema, hypotension, and bronchospasm constitute classic presentation. However, up to 20% of anaphylactic reactions lacking any cutaneous manifestations or signs of vasomotor instability [1]. Diagnostic criteria were established by a multidisciplinary task force in 2005 to aid in the recognition of atypical presentations of anaphylaxis [1]. In cases of anaphylaxis, laboratory tests might show transient elevation of tryptase and histamine levels, but these are not useful diagnostically in the acute setting [2-4].

20% of patients with rapid onset of severe anaphylactic reactions are at risk of biphasic or rebound anaphylaxis [2]. Most biphasic responses occur during the first 8 hours, but it might be delayed up to 72 hours [5]. There is no consensus on the optimal period of observation for a patient who has been treated for anaphylaxis [6].

Steroids are unlikely to be helpful in the treatment of acute anaphylaxis. They have a delayed onset of 4 to 6 hours. Steroids are thought to play a role in preventing rebound anaphylaxis; however, this has never been proven.

Bronchodilators might be used in patients with refractory wheeze, but they do not relieve bronchial smooth muscle contraction or decrease mucus production.

Ketamine, a water soluble phencyclidine derivative contains an asymmetric carbon atom with two enantiomers: The S (+) isomer and the R (-) isomer [8].

It being highly lipid soluble and undergoes rapid breakdown and redistribution to peripheral tissues. It is metabolized extensively in the liver by N-demethylation and ring hydroxylation pathways [9].

Patients who received ketamine improved clinically, had lower oxygen requirements, and obviated the need for invasive ventilation. Mechanically-ventilated patients for severe bronchospasm showed reduction in peak inspiratory pressures, improved gas exchange, dynamic compliance and minute ventilation, and could be weaned off successfully following introduction of ketamine. It has minimal effects on central respiratory drive and produces airway relaxation by acting on various receptors and inflammatory cascades and bronchial smooth muscles [8].

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Thus, stimulates the cardiovascular system resulting in an increase in heart rate, blood pressure and increase in cardiac output, mediated principally through the sympathetic nervous system [10].

Conclusion

In this case, ketamine was effective in relieving bronchial smooth muscle spasms secondary to second surge in anaphylaxis. Ketamine can be a rescue therapy in severe sudden onset anaphylaxis cases needing admission to intensive care unit.

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