

4 Vignettes in Critical Care

Abstract: The article contains scenarios from critical care with discussion about the diagnosis and management of these problems with useful references.

SCENARIO 1

A 24 year old lady was admitted with a history of fever for 5 days following a normal vaginal delivery. She came to casualty and was found to be conscious with a temperature of 40°C (103°F); respiratory rate of 40/min; blood pressure of 80/60 mm Hg and a pulse rate of 120/min. Examination of the cardiovascular and respiratory systems were normal. Abdominal examination revealed mild lower abdominal tenderness. She was admitted and relevant tests including cultures were sent. The first doses of antibiotics were given. A central line was inserted and the CVP was 8cm H₂O and her blood pressure was now 90/50mm Hg. Ultrasound abdomen and pelvis showed that the uterus was empty but there was some fluid in the pelvis. The other investigation results were as follows:

- Hb 8g %; WBC TC 20,000/cumm; DC N95 L5; Platelets 80,000/cumm;
- Prothrombin time INR 1.2; aPTT 30 seconds
- Serum Na 140 mmol /L ; K 5.5 mmol/L; Cl 95 mmol/L; Ca 7mg% P 3 mg%; creatinine 2 mg%;
- ABG: (on 100% or Face mask): PO₂ 60 mm Hg; PaCO₂ 30 mm Hg; pH 7.15; SaO₂ 90%; HCO₃ 10 mmol/L;
- Base Excess = - 20 mmol/L; Lactate = 3mmol/L;
- Liver Function Test: Bilirubin 2 mg%, direct 1mg% SGPT 34 SGOT 40 (both normal); Alk Phos 150 (mild increase)
- An attempt to aspirate fluid from the pelvic collection was not successful.
- An ABG from the CVC sample showed ScvO₂ 60%.

COMMENTS FOR SCENARIO 1

An acute onset of fever with tachypnea, tachycardia and low blood pressure must raise the possibility of a Systemic Inflammatory Response Syndrome (SIRS). Although all SIRS is not due to infection, an infective etiology is high on the list. If SIRS is due to infection, the diagnosis is sepsis (or an allied syndrome).

The management of septic shock should follow the sepsis guidelines as much as feasible. The components of these guidelines are:

1. Early Source Control – appropriate intravenous antibiotic – the first dose to be given within one hour of presentation and drainage of any localized infected collection.
2. Early Goal Directed Therapy (EGDT).
3. Initial choice of inotrope is between adrenaline and noradrenaline - (cost consideration is important. A recently published study has shown them to be equivalent in terms of effect on outcome.
4. Low dose steroid therapy to be considered.
5. *General supportive therapy:* DVT prophylaxis, GI stress ulcer prophylaxis, blood glucose control.
6. *Support of failing organ systems as appropriate:* Mechanical ventilation, dialysis.
7. *Other specific therapies:* Activated protein C, IV Immunoglobulin as indicated.

The arterial blood gas analysis shows a metabolic acidosis. The analysis can be done using the classical method or the Stewart method. The Stewart method and the classical method are equivalent to understand pure respiratory disorders but the Stewart method gives more insight into the metabolic component of the derangement. The ABG evaluation also shows that her respiratory compensation for the metabolic acidosis is inadequate and this implies that her respiratory muscles are fatiguing and that she may need ventilatory support.

SCENARIO 2

30 year old farmer was admitted with a history of fever for 5 days and cough for 2 days. He has experienced increasing dyspnea for 1 day and was found on examination in casualty to be conscious with a temperature of 38.5°C (102°F); respiratory rate of 45/min; blood pressure of 110/70mm Hg, and an SpO₂ of 80%. He had a skin lesion (Fig. 1).

Cardiovascular examination was normal; Respiratory examination revealed occasional scattered crackles. The abdomen was normal. He was intubated after the ABG showed (on 100% Oxygen): PaO₂ = 45mmHg; PaCO₂ 52mmHg; pH 7.15; HCO₃ 17mmol/L; BE = - 2mmol/L. The other investigations were: Hb10g% TC 30,000 DC N80 L15 M5; Platelets 90,000; Serum Na 138mmol/L; K 4.5mmol/L; Creatinine 1.0mg%.

Chest X-Ray was taken (Fig. 2).

COMMENTS FOR SCENARIO 2

Acute Respiratory Distress Syndrome (ARDS) is a common reason for ICU admission. It may be primary (due to a process in the lung) or secondary (due to an extrapulmonary process). The criteria are well defined and can even include the pulse oximetry saturation (using the S/F ratio instead of the P/F ratio) (recently published study).

Ventilatory management must incorporate:

1. Lung protective ventilation strategy.
2. Fluid administration can be conservative or liberal.
3. Beta agonists may be useful.
4. The use of steroids is still not well defined in terms of altering outcome.
5. Prone positioning improves gas exchange but has not been shown to alter outcome.

Scrub typhus is an important reversible cause of ARDS in some areas. The eschar is a characteristic sign of scrub typhus. Antibiotics of choice are doxycycline, chloramphenicol, rifampicin, azithromycin. It is a curable infection - hence an early accurate diagnosis is essential.

SCENARIO 3

50 year old man with chronic obstructive airways disease presented with increased dyspnea for a day. On examination, he was tachypneic with a temperature of 38.3°C (101°F); respiratory rate of 50/min; pulse rate of 130/min, high bounding pulse. His blood pressure was 110/50, and his SpO₂ was 85%. Cardiovascular examination was normal and respiratory examination revealed widespread rhonchi. Abdomen was normal. He had a mild asterixis.

- ABG on 100% oxygen: PaO₂ 150 mm Hg; PaCO₂ 70 mmHg; pH 7.20, HCO₃ 28 mmol/L; BE - 1mmol/L
- He was placed on non-invasive ventilatory support (NIV) with
- FiO₂: 0.5; PEEP 5 cmH₂O; Pressure support 15 cmH₂O.
- After 2 hours, his ABG was as follows:
 - Scenario 3a: PaO₂ 66 mm Hg; PaCO₂ 88 mmHg; pH 7.15, HCO₃ 28 mmol/L; BE - 1 mmol/L.
 - Scenario 3b: PaO₂ 66mm Hg; PaCO₂ 50 mmHg; pH 7.30, HCO₃ 24 mmol/L; BE - 1 mmol/L.

COMMENTS FOR SCENARIO 3

Non-Invasive Ventilation (NIV) is an important modality to treat the acute respiratory failure of those with COPD. It may be given by modern models of standard ventilators (older models did not have this option) or by dedicated BiPAP/ CPAP machines. Those offered NIV must be carefully chosen and monitored for progress. An unconscious patient should not be given NIV.

Scenario 3a shows deterioration in spite of NIV – rising PaCO₂ with a falling pH. Significant lack of improvement must lead to a change of strategy – either changing the settings for NIV or initiation of invasive ventilatory support (endotracheal intubation and ventilation). Weaning from invasive ventilation for patients with COPD is likely to be prolonged.

In scenario 3b, there is significant improvement – the respiratory acidosis is resolving and the patient does not need invasive ventilator support.

SCENARIO 4

56 years old man, who consumes alcohol regularly, presents with a history of left sided upper and lower limb weakness for 10 days and progressive deterioration in sensorium for 7 days. He stopped all medication 10 days ago. On examination, he was drowsy with a temperature of 36.5°C (98°F). Estimated weight was 50kg. His respiratory rate was 14/min; blood pressure was 90/70; pulse was 115/min. He has no pedal edema. Examination of his chest was normal. Abdominal examination revealed mild ascites with no organomegaly. Neurological examination showed a GCS of 10/15, with a mild flap. He had a left hemiparesis. A central line was inserted and showed a CVP of 4cm H₂O.

Investigations

- Hb 9g% MCV 110; TC 12000, DC N70 L25 M5; Platelets 110,000
- PT INR 1.5; PTT 35seconds
- Na 115mmol/L; K 2.5mmol/L; Ca 6mg%; Ionized Ca 2.3mg%; P 3.7mg%; Cl 95mmol/L
- Blood glucose 50mg% ; S Creatinine 1.4mg% Blood Urea 40mg%
- *Liver Function Test*: Bil T= 1mg% Direct 0.5mg%; SGOT 35 SGPT 30 (both normal). Alkaline Phosphatase 120 (normal) Protein T = 6g% Alb 2.5g%
- *ABG on room air*: PaO₂ 100mmHg ; PaCO₂ 30mm Hg; pH 7.60; HCO₃ 30mmol/L; BE + 4mmol/L
- Urine Na 88mmol/L; Plasma Osm 250mOsm/kg; Urine Osm: Random 1100mOsm/kg
- *Ascitic Fluid*: Cells: 65cells N10 L80; Protein 0.8g% Alb 0.5g%
- CT Brain shows right frontoparietal infarct with mild cerebral edema.
- He was given intravenous glucose bolus (20ml of 50% glucose) and started on a 10% glucose infusion. He was also given 5g potassium chloride over 4 hours and 2 liters of normal saline over 24 hours .
- Repeat electrolytes showed serum Na of 124 mmol/L and serum K of 2.8mmol/L.

COMMENTS FOR SCENARIO 4

This patient has multiple problems. Serum sodium is a ratio between total body sodium and total body water. Although Na is distributed predominantly in ECF, correction must consider total body water as sodium exerts changes in all body water compartments through its osmotic action. He has hyponatremia with a high urine spot Na, low plasma osmolality and high urine osmolality. Hyponatremia can be due to depletion or dilutional states. The dilutional hyponatremias can be due to SIADH (due to free water retention) or due to an excess total body water and sodium – with water being retained in excess of sodium as in edematous states (cirrhosis, nephrotic syndrome, CCF). The high urine sodium is consistent with a depletion state of renal origin (extra renal sodium depletion would result in a low urine sodium as the body tries to conserve sodium). A low urine sodium would also be seen in relative intravascular volume depletion as in hypoalbuminemia). In a SIADH, the urine sodium would be high (as the ADH stimulates free water absorption) but there would be no volume depletion (as seen in this patient by the low blood pressure and low central venous pressure). Diuretic use would also

cause natriuresis but this person has stopped all medication. The cause for the hyponatremia in this person is likely to be renal sodium wasting – the possibility of a cerebral salt wasting needs to be considered. He was given 2 liters of normal saline – a total of 308 mmol of sodium (154 x 2). This results in a rise of serum Na by about 9mmol/L. It is important to limit the rise of serum sodium during correction to < 12mmol/L per 24 hours in order to avoid the neurological complication of central pontine and extra pontine myelinolysis. This is important if the hyponatremia is chronic (> 48 hours). Acute hyponatremia can be corrected more rapidly. He also has low potassium and calcium levels. The low potassium level needs correction but the rate of potassium administration must be monitored as the relationship between body stores and serum levels of potassium is not linear. At a serum level of 4mmol/L, hyperkalemia can occur rapidly if potassium infusion is continued at the same rate as to correct it from a level of 2mmol/L to 3 mmol /L (Fig. 3).

The low calcium level is not just because of the low albumin – the ionized calcium is also low.

Magnesium deficiency results in impaired parathyroid hormone secretion (resulting in hypocalcemia) and increased renal tubular loss of potassium. Suspect magnesium deficiency in a person with resistant hypokalemia or with combined hypocalcemia and hypokalemia. Magnesium deficiency is common in alcoholics and needs to be corrected as it is difficult to correct the hypokalemia just by repletion when there is concomitant magnesium depletion.

In this person, it is also important to rule out an infection – the ascitic fluid shows a high SAAG (indicating portal hypertension). However, the profile of ascitic fluid analysis is unlike an infection.

The patient also has a high MCV – liver disease is a cause of macrocytosis but in a patient with chronic alcohol intake, a folic acid deficiency needs to be considered as a possible cause of the high MCV.

The altered sensorium in this person needs to be considered to be due to one or more of many factors: cerebrovascular accident with cerebral edema, hyponatremia, hepatic coma or associated sepsis.

SUGGESTED READING

These are not comprehensive but chosen on the basis of being at least one of the following: State of the Art article, available online, Indian context, significant management option.

SEPSIS, SHOCK, ACID BASE ANALYSIS

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6. Online Reference : Ventilator Bundle: <http://www.ihl.org/IHI/Topics/CriticalCare/>

NON INVASIVE VENTILATION

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ELECTROLYTE ABNORMALITIES OF THE CRITICALLY ILL:

1. Online References: Critical Care & Resuscitation: downloadable pdf files: <http://www.jficm.anzca.edu.au/publications/journals/index.htm>.
 - a. September 1999: Potassium correction
 - b. September 2000: Speed of sodium correction
 - c. June 2002: Phosphorus metabolism in ICU patients
 - d. December 2002: Calcium, Phosphorus, Magnesium metabolism in the critically ill.
 - e. September 2005: Saline, Osmoles, Albumin.

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