QUIZ 8th July 2020 (answers below)

1. **What are the differential diagnoses for left upper lobe consolidation?**

2. **What are risk factors for acquiring TB?**

3. **Does a positive tuberculin skin test indicate TB infection?**

4. **Who gets screened for latent TB in Australia?**

5. **Describe and interpret the following blood gas analysis.**

ABL827 Emergency
PATIENT REPORT

Syringe - S 250uL

Sample # 70624

Identifications

Patient ID	00000
Patient Last Name	ACUTE9
Patient First Name	UNKNOWN
Sex	Female
Sample type	Venous
T	37.0 °C
FO_2(I)	21.0 %
PEEP	cmH2O
Pressure Support	cmH2O
SIMV	Rate
Liter Flow	L/min
Note	
Operator	
Accession No.	

Blood Gas Values			
↓ pH	**7.091**		[7.350 - 7.450]
↑ pCO_2	**61.4**	mmHg	[32.0 - 45.0]
↓ pO_2	**31.2**	mmHg	[75.0 - 105]
Oximetry Values			
↓ *c*tHb	**86**	g/L	[115 - 165]
↓ sO_2	**48.3**	%	[95.0 - 99.0]
*F*COHb	**0.5**	%	[0.0 - 1.5]
↓ *F*MetHb	**0.0**	%	[0.0 - 1.5]
Electrolyte Values			
↑ cNa^+	**148**	mmol/L	[137 - 146]
↓ cK^+	**3.4**	mmol/L	[3.5 - 5.0]
↕ cCa^{2+}	**< 0.20**	mmol/L	[-]
↓ cCl^-	**91**	mmol/L	[98 - 106]
Metabolite Values			
↑ *c*Glu	**15.6**	mmol/L	[3.0 - 7.8]
↑ *c*Lac	**6.5**	mmol/L	[0.0 - 2.2]
*c*Crea	**84**	µmol/L	[40 - 90]
Calculated Values			
ABE_C	**-11.4**	mmol/L	[-]
$cHCO_3^-(P)_C$	**17.8**	mmol/L	[-]

QUIZ answers 8th July 2020

1. **What are the differential diagnoses for left upper lobe consolidation?**

Consolidation is when the alveolar air spaces are filled with exudate, transudate, blood, cells or other material.

Acute unilateral consolidation can be from:

Pus – bacterial, fungal, viral, atypical, aspiration
Fluid – unilateral pulmonary oedema
Blood – contusion, haemorrhage
Emboli – fat, PE, amniotic fluid
Cells – bronchoalveolar carcinoma

With lymphadenopathy it also includes

Post obstructive causes – lung Ca, Lung, mets, Lymphoma, Leukaemia
Infection – post primary TB, fungal, atypical – EBV, Mycoplasma

When chronic it also includes

Neoplastic – post obstructive, lymphoma, adenoCa
Infective – TB, Fungal, Incomplete treatment of infection
Inflammatory – sarcoid, eosinophillic, cryptogenic, granulomatosis
Radiation penumonitis

2. **What are risk factors for acquiring TB?**

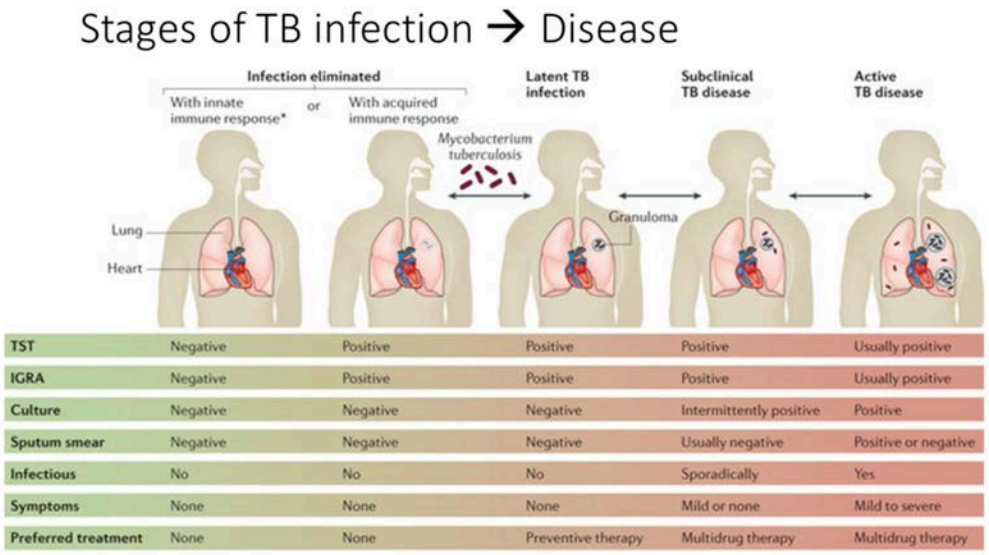
Host factors – Smoking, diabetes, Chronic kidney disease, immunosuppression

Source factors – Pulmonary disease with cavity and AFB positive, extent of pulmonary disease, duration of exposure (hours, days, weeks), Effective treatment for TB

Ambient factors – humidity, ventilation, UV light, "crowding index"

3. **Does a positive tuberculin skin test indicate TB infection?**

The Tuberculin Skin Test (TST) tests for an immune response to TB, not for actual TB. A person can have eliminated the TB and still have an immune response. If, however, they also have a scar on CXR, then they are regarded as having latent TB. See diagram below.



4. Who gets screened for latent TB in Australia?

WHO recommends that in middle to high income countries with TB incidence less than 100 per 100,000 population, systematic testing and treatment for latent TB should be performed in people living with HIV, adult and child contacts of pulmonary TB cases, patients initiating anti-TNF treatment, patients receiving dialysis, patients preparing for organ or haematological transplantation and patients with silicosis.
Systematic testing and treatment for latent TB should be considered for prisoners, health workers, immigrants from high TB burden countries, homeless persons and illicit drug users.
Systematic testing for latent TB is not recommended in people with diabetes, harmful alcohol use, smokers and underweight people unless they are already included in the above recommendations.

5. Describe and interpret the following blood gas analysis.

pH	*7.091*	*Acidotic*
pCO_2	*61.4mmHg*	*Respiratory acidosis*
		If this were the only abnormality it would cause the HCO_3^- to acutely increase by 2mmol/L from 24 to 26mmol/L. The resultant pH would be 7.25. The pH here is more acidotic than that, so without even looking at the HCO_3^-, we can say that there must be a metabolic acidosis as well.
HCO_3^-	*17.8 mmol/L*	*Metabolic acidosis, also evidenced by BE -11.4mmol/L*
Anion gap	*39.2mmol/L*	*High anion gap metabolic acidosis HAGMA*
Delta ratio	*3.3*	*HAGMA + metabolic alkalosis*

<0.4	*Pure NAGMA*
0.4 – 1.0	*HAGMA + NAGMA*
1.0 – 2.0	*Pure HAGMA*
>2.0	*HAGMA + metabolic alkalosis*

Hb *86g/L = low*
iCa *<0.2mmol/L = low*
Glu *15.6mmol/L = high*
Lactate *6.5mmol/L = high*
Creat *84umol/L = normal*

Causes of HAGMA

Ketones – may be high as glucose is high
Lactate – high
Uraemia – less likely as creatinine is normal
Toxins – unknown

Causes of metabolic alkalosis

Initiation factors

Ketone metabolism – could be as hyperglycaemic
Exogenous bicarb – antacids, bicarb, citrate

Unknown but low Hb could have resulted in blood transfusion resulting in citrate. This would also explain the very low ionised calcium.

GIT loss of acid
Renal – but creatinine is normal

Maintenance factors

Chloride depletion – yes
Hypokalaemia – yes, mild
Decreased GFR – no, creatinine normal
Mineralocorticoids – could be, as hypernatraemia and hypokalaemia

➔ *Respiratory acidosis*
High anion gap metabolic acidosis
Concurrent metabolic alkalosis
Anaemia
Hyperlactataemia
Hyperglycaemia
Hypocalacaemia

➔ *This patient was a major trauma patient who was in hypovolaemic shock with GCS 3 and had received 3 units of blood pre-hospital.*
The respiratory acidosis is from hypoventilation
The HAGMA is from lactate due to shock
The anaemia is from haemorrhage and the metabolic alkalosis and low iCa is from the citrate from blood tranfusion
And the hyperglycaemia is a stress response