

## Pathophysiology Scenario: Diabetes

Answer to question1.

Based on Roberto's case, it could be stated that diabetes affected him to a greater extent. From his haematology and biochemical tests it could be stated that Roberto might have developed the condition of diabetic ketoacidosis, which could also be supported by the presence of ketone bodies in urine. The ABG test enables to measure blood pH, level of carbon dioxide (CO<sub>2</sub>) and oxygen (O<sub>2</sub>) present within the blood stream. It helps to understand how well the lungs are working to transport O<sub>2</sub> into blood and eliminate CO<sub>2</sub> from the blood circulation (Noble, 2009). pH was 6.9 that indicates acidosis. A PCO<sub>2</sub> level below 35mmHg causes alkalosis and indicates increased ventilation or the condition of tachypnoea. The HCO<sub>3</sub><sup>-</sup> level demonstrates acidosis in metabolic component of ABGs. Acute cause of alteration in pH might include vomiting that resulting in metabolic alkalosis. Roberto is in somewhat compensated metabolic acidosis along with minor hypoxemia.

Answer to question2.

Roberto appears to have poor blood glucose control. As a result of this, he may develop diabetic nephropathy in the future. Hyperglycaemia is a major factor accountable for structural change at the renal stage. Hyperglycaemia stimulates damage to the kidneys by activating protein kinase C that increases the production of glycosylation and related products along with the synthesis of diacylglycerol. It also affects glomerular filtration and increase shear stress. Such changes contribute to excessive stimulation of renal cells that secrete more amount of TGF-beta1, which upregulates GLUT-1 and induces glucose uptake and transport (Kanwar et al., 2011). TGF-beta1 also increases deposition of extracellular matrix protein like laminin, fibronectin at glomerular level and thereby, thickens the glomerular basement. Simultaneously, poor enzymatic degradation of the extracellular matrix gives rise to excessive accumulation. Roberto's condition shows that he has HCO<sub>3</sub><sup>-</sup> level below the normal range, which in turn indicates abnormal functioning of the kidneys in regulating body's buffer system (Scheda, 2005). Roberto's breathing pattern was rapid that exhales too much CO<sub>2</sub>, reduced his PaCO<sub>2</sub> and increased the pH of arterial blood. His body tried to compensate for the alkalosis by causing the kidneys to eliminate more bicarbonate that made the arterial blood more acidic.

Answer to question3.

The amount of HbA1c is equivalent to the amount of glucose present in the blood. It identifies the number of glycosylated red blood cells (RBCs). HbA1c level less than 6.5% (48mmol/mol) is recommended as the end point for detecting diabetes (RACGP, 2014). It can be used to detect diabetes without an OGTT because it provides an indication of prolonged glycaemia and a slow progression to problems over the entire 120 days lifecycle of the RBCs. The red blood cells are viable for 8-12weeks and measuring HbA1c gives an average idea about the blood glucose level since last 8-12weeks (Practitioners, 2016). In this test individuals need not to drink anything and not simply inform how a system processes glucose like in OGTT.

#### References

Kanwar, Y., Sun, L., Xie, P., Liu, F. and Chen, S. (2011). A Glimpse of Various Pathogenetic Mechanisms of Diabetic Nephropathy. *Annu. Rev. Pathol. Mech. Dis.*, 6(1), pp.395-423.

Noble, K. (2009). THE ABCs of Arterial Blood Gasses. *Journal of PeriAnesthesia Nursing*, 24(6), pp.401-405.

Practitioners, T. (2016). *RACGP - 3.4 Diagnosis of diabetes*. [online] Racgp.org.au. Available at: <http://www.racgp.org.au/your-practice/guidelines/diabetes/3-screening,-risk-assessment,-case-finding-and-diagnosis/34-diagnosis-of-diabetes/> [Accessed 6 Oct. 2016].

Schena, F. (2005). Pathogenetic Mechanisms of Diabetic Nephropathy. *Journal of the American Society of Nephrology*, 16(3\_suppl\_1), pp.S30-S33.