Redness bruising and swelling biology essay

Science, Biology

Faculty of Science and Technology

NAME: Tim Davis

STUDENT ID: 211334694

UNIT CODE: SLE323

ASSIGNMENT/PRAC No.: 1

ASSIGNMENT/PRAC NAME: Case Studies A&B

DUE DATE: 17/5/13

Plagiarism and collusion

Plagiarism occurs when a student passes off as the student's own work, or copies without acknowledgment as to its authorship, the work of any other person. Collusion occurs when a student obtains the agreement of another person for a fraudulent purpose with the intent of obtaining an advantage in submitting an assignment or other work

Declaration

I certify that the attached work is entirely my own (or where submitted to meet the requirements of an approved group assignment is the work of the group), except where work quoted or paraphrased is acknowledged in the text. I also certify that it has not been submitted for assessment in any other unit or course. I agree that Deakin University may make and retain copies of this work for the purposes of marking and review, and may submit this work to an external plagiarism-detection service who may retain a copy for future plagiarism detection but will not release it or use it for any other purpose.

DATE:

An assignment will not be accepted for assessment if the declaration appearing above has not been duly completed by the author.

Part A – WOUND HEALING

Question 1

The visual clues that show inflammation are redness, bruising and swelling in the area of the surgery. Characteristic symptoms of inflammation are redness and increased local temperature due to increased vessel permeability and therefore increased blood flow to the effected area (hyperaemia), bruising resulting from the break down of excessive blood cells in the area, swelling (oedema) to dilute harmful substances at the injured site and to deliver nutrients to damaged cells, increased number and activity of phagocytes, and pain caused by inflammatory chemicals such as bradykinin (Hargreaves et al., 1988, Vane, 1976), from toxins released by the microbe, fluid pressure build up on the nerves, and nerve damage. The purpose of these symptoms are to limit the spread of pathogens and inevitably destroy them, to remove the damaged tissue debris and repair the tissue damage. In regards to the purpose of localized pain, this will encourage loss of function in order to not cause further harm to the affected area. The three stages of inflammation are the vascular response, visible in seconds after the injury, the migration of white blood cells to tissue also known as the cellular response (within an hour from injury), and tissue repair (Martini et al., 2008). The vascular response is characterized by blood vessel dilation and increased permeability of capillaries caused by leukotrienes and histamine for the passage of larger substances eg. Antibodies and clotting

factors to the damaged areas. In acute inflammation, increased venule and capillary permeability leads to increased formation of interstitial fluids and allowing small amounts of plasma proteins to leak into the interstitium. These proteins allow water to be held osmotically, serving to dilute the toxins. The subsequent oedema presses on nerve endings causing beneficial loss of function to the area due to the pain. The exudate also contains fibrinogen that may be converted into insoluble fibrin that will aid in the clotting process, defensive antibodies and complement proteins to aid phagocytosis and kill foreign or infected cells. Other chemical mediators such as bradykinin cause pain to the localized area and are amplified with prostaglandins released from mast cell activation. The cellular response is distinguished by the margination aided by turbulence and pavementing of white blood cells to the endothelial walls to create an inner lining. The white blood cells then pass through the endothelial cells via diapedesis and in response to chemical signals or chemotactic substances, emigrate to the injured or diseased area. The most common and first type of white blood cells involved in the cellular response are the polymorphonuclear neutrophils attracted by cytokines in turn, express and release cytokines attracting other cell types, followed by monocytes and eosinophils some hours later. Neutrophils initially help fight infection by phagocytosis, degranulation and Neutrophil extracellular traps (NETs). Monocytes become macrophages once in the tissue and engulf degraded neutrophils and microbes. Pus, often shown in infected wounds are dead phagocytes and tissues. The tissue repair phase has three phases: inflammatory phase (vascular response and cellular response), proliferation phase and remodeling phase. The proliferation phase

occurs two to three days after the injury and can occur via primary or secondary intention. The formation of scar tissue involves proliferating connective tissue from surrounding areas extending into the damaged tissue. Primary intention is healing of a wound where the wound edges re-approximate and generally results in a small line of scar tissue. An example of primary intention healing is a sutured incision made by a doctor. Healing by secondary intent occurs in larger wound areas and burns. In this case, the wound edges cannot re-approximate and is left open to fill with granulation tissue. Normally, healing by second intention is slower than by primary intention and results in a large amount of scar tissue being formed. During primary intention, the wound healing occurs by re-epithelialization, fibroblast migration, and collagen-producing epithelial cells repairing the surface. This is most commonly shown as a scab. The scab lifts off when the epithelial cells have completely covered the open wound. Fibroblasts are the most common type of connective tissue in animals and proliferates at the site of inflammation and synthesizing collagen. Key features present in secondary intent healing include: macrophages releasing angiogenesis factor and fibroblast-activating factors to aid in the repair of the large wound, the re-epithelialization including the inward migration of fibroblasts, the beginning of collagen synthesis and secretion and the formation of fragile granulation tissue. However problems can occur when excessive granulation tissue is produced hindering the re-epithelialization. This is known as proud flesh. The remodeling phase occurs three weeks after the injuring lasting for up to six months or longer. This phase includes the completion of the contraction, differentiation and remodeling of the granulation tissue with simultaneous

synthesis of collagen and collagenase, re-orients the scar to increase the strength of the wound, and the disappearance of the new blood vessels from the scar tissue

Question 2

The dark granulation tissue are signs of necrosis possibly due to a lack of blood supplying oxygen to the localized area (ischemia)(Scher et al., 1986). This information shows us that granulation tissue including angiogenesis are present (Eming et al., 2007).(I) The cell death of acute lymphoblastic leukemia (ALL) patients was preceded by molecular and cellular changes characteristic of apoptosis (Manabe et al., 1992, Meydan et al., 1996). ALL is characterized by an overproduction of blast cells (the precursors to immune cell lines)(Robertson et al., 1993). Blast cells can crowd the bone marrow, preventing it from making normal blood cells and they can also spill out into the blood stream and circulate around the body (Ferrando et al., 2002). Blast cells can also spread to the surface of the bone or into the joint from the marrow cavity can cause symptoms of Bone pain and joint pain. Due to the blast cell’s immaturity, they are unable to function properly to prevent or fight infection. Apoptosis is triggered by the P53 gene when a cell is damaged beyond repair, or is genetically altered (particular in ALL patients, the pathophysiology is associated with exposure to radiation or mutagenic chemicals). Apoptosis is also known as programmed cell death and affects isolated cells ie. Blast cells, rather than cell masses. Studies have demonstrated the role that chemotherapeutic drugs play in cell-intrinsic pathways for inducing apoptosis either by inducing expression of death receptor ligands or inducing release of cytochrome c from the mitochondria

(Kaufmann and Earnshaw, 2000).(II) The cell death that is occurring in gangrene as a result of smoking is coagulative necrosis (Khan, 2006). Coagulative necrosis is a type of cell death that is typically caused by ischemia (Majno and Joris, 1995). The lack of oxygen saturation in the body (hypoxia) as a side-effect of long-term smoking can cause cell death in a localized areas where blood perfusion is low, for example, the lower extremities. In the lower extremities of long-term smokers, vessels can fail to deliver enough oxygen, but also other important nutrients needed for continuous cell function. The lack of oxygen and nutrients to the cells leads to coagulative necrosis (Majno, 1964).

Question 3

False. In photo A of Chelsea from the case study, the surgical incisions have been sutured an therefore characteristic of primary intention wound healing. This type of wound healing is allowing Chelsea’s wound to heal from the outside-in because the edges of the skin are clean cut. Healing by secondary intention means that the wound will heal from the inside-out and is often distinguished by an open wound as shown in photos J, K, L and M. As opposed to primary intention, secondary intention allows for the owner or the Veterinarian to monitor the complete healing process (Witte and Barbul, 1997). The answer is false because Chelsea’s open wound in photos J and K (marked by the hash symbol [#]) have shown to be healing by both primary and secondary intention simultaneously as wound healing is not a linear process (Kirsner and Eaglstein, 1993).

Part B – Hypertension

Question 1:

(a)Essential hypertension also known as primary or idiopathic hypertension is a form of hypertension that has no identifiable cause (Folkow, 1982). Hypertension affects one billion people worldwide and is implicated in 7. 1 million deaths each year from ischemic heart disease and stroke (Mein et al., 2004). Essential hypertension is the most common type of hypertension, affecting 95% of hypertensive patients. It has been shown in family and epidemiological studies that essential hypertension results from synergistic characteristics of genetic (Kurtz and Spence, 1993) and environmental lifestyle exposures for example: dietary sodium intake (Kawasaki et al., 1978), alcohol consumption, and body weight (Snieder et al., 2003). Essential hypertension is more often than not a factor associated with ageing, being overweight, insulin resistance, diabetes, and hyperlipidaemia (Messerli et al., 2007).(b)Thiazide diuretics are widely used for the treatment of hypertension (Hughes, 2004). Thiazides lower blood pressure by causing diuresis resulting in a fall in plasma volume and an overall reduction in cardiac output. Thiazide acts in aiding the body in natriuresis through inhibiting the sodium-chloride symporter in the distal convoluted tubule, leading to a retention and excretion of water in the urine. Thus, when the sodium is excreted, the amount of water in the blood stream is reduced. The short-term action of thiazides is based on a decreased preload (initial stretching of the cardiac muscle fibers prior to contraction) and more viscous blood resulting in a decreased cardiac output and less pressure exerted on the artery walls. According to the Frank-Starling law of the heart, decreasing

the cardiac output results in a decrease in blood pressure. Long-term use thiazides can cause a reduction in blood pressure by lowering the total peripheral resistance via vasodilation. Although the mechanisms on how they lower blood pressure in the long term are not fully understood (O'Brien et al., 2007). Sometimes diuretics alone will not effectively treat hypertension but, other medications such as ACE inhibitors or beta blockers can also be prescribed (Yoshimura and Kawai, 2010).

Question 2:

Liquorice is a sweet confectionary flavoured with the extract of roots from the liquorice plant. Uncontrolled eating of liquorice is know to show similar symptoms of excess aldosterone and can cause hypertension. However, hypertension is known to be reversible when the confectionary is withdrawn from the diet. The main sweet tasting compound of liquorice is call glycyrrhizin and is the widely associated cause of liquorice induced hypertension. The Increased hypertension is reflected by the increased activation of renal mineralocorticoid receptors by cortisol (Shibata, 2000). It has been shown that licorice inhibits 11 beta-dehydrogenase, preventing local inactivation of cortisol and allowing cortisol inappropriate access to intrinsically nonspecific renal mineralocorticoid receptors. Deficient 11 beta-dehydrogenase activity provides a novel pathogenetic mechanism for hypertension, and current research suggests that several common forms of hypertension can be explained by the mechanisms that operate in licorice-induced hypertension. Doses as low as 50 grams of liquorice or 100mg of Glycyrrhizin daily for two weeks can raise blood pressure or cause muscle

weakness, chronic fatigue, headaches or swelling, and lower testosterone levels in men.

Question 3:

(a)What is pseudohyperaldosteronism?(2 mark)(b)How did the doctor’s conclude that she had this condition i. e. explain their reasoning? (3 marks)

Question 4:

The old lady was told not to eat liquorice and prescribed potassium tablets. This resulted in the old lady’s potassium levels returningto normal. However, her blood pressure remained high and she was given a -blocker drug. Explain how -blockers work to reduce blood pressure.(6 marks)