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| **Name:** |
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| **Group:** |
| 2B-2 |
| **Basic Science Question:** |
| What is the Inflammatory pathway from a Necrotic nerve? |
| **Report:** |
| **Pulp microcirculation:** Pulp microcirculation is important for blood flow in the pulp. Careful regulation of pulp blood flow is critically important and alterations in pulp micro-circulation may be the first to occur with the onset of pulp inflammation.**Pulp nerves**: The dental pulp contains both sensory and autonomic nerves to fulfill its vasomotor and defensive Functions. It would be expected that sensory nerves participate in the inflammatory process by an increased release of the neuropeptides. **Diseases of pulp:** Irritants can be classified as being short-term, long-term or due to trauma. Each type of irritant or injury will have a different effect on the pulp – in general, the effects will be acute inflammation, chronic inflammation or necrosis. . Long-term irritation will cause chronic inflammation of the pulp and, if left for long enough, pulp necrosis. This will then be followed by infection of the pulp space since bacteria will have a pathway by which they can enter the tooth**Disease Progression:** The two key components in pulp inflammation/ necrosis are the microcirculation and the sensory nerve activity. Injury to the pulp may activate the intradental sensory nerves to release neuropeptides, which causes alteration of microcirculatory haemodynamics. The response of sensory nerves to stimuli depends upon the severity of the pulp injury and the stages of inflammation. Within the first few minutes of injury, destruction and disruption of nerve fibres in the injured dentine and pulp occurs, followed by hypersensitivity of the surviving nerve fibres and the release of neuropeptides into the pulp. Inflammatory mediators, such as bradykinin and the prostaglandin E2, may also cause the neurosecretion of calcitonin-gene-related-peptides. These neuropeptides cause vasodilatation and increased vascular permeability, and in turn - the neurogenic inflammation. The tissue becomes oedematous because filtration of serum proteins and fluid from the vessels. Because of the environment of the pulp, the increase in both interstitial fluid volume and blood volume leads to an increase in the tissue pressure, which in turn causes compression of the thin-walled venules, resulting in a decrease in blood flow and an increase in flow resistance in the venules. The flow stasis causes an aggregation of red blood cells and an elevation of blood viscosity. It also produces tissue hypoxia, which suppress cellular metabolism in the affected area of the pulp. This results in tissue necrosis. An increase in carbon dioxide and a decrease in pH levels alter the local micro-environment, and may lead to vasodilatation in the adjacent area and the gradual spread of inflammation. And lastly, Neutrophils in the area degenerate and release intracellular lysosomal enzymes to digest the surrounding tissue, forming necrotic tissue. (1) |
| **References:** |
| Yu, C., and PV Abbott. “An Overview of the Dental Pulp: Its Functions and Responses to Injury.” *Wiley Online Library*, John Wiley & Sons, Ltd, 12 Mar. 2008, onlinelibrary.wiley.com/doi/pdf/10.1111/j.1834-7819.2007.tb00525.x. (1)Zanini, Marjorie, et al. “Pulp Inflammation Diagnosis from Clinical to Inflammatory Mediators: A Systematic Review.” *Journal of Endodontics*, Elsevier, 17 May 2017. (2)Rechenberg, Dan-Krister, et al. “Biological Markers for Pulpal Inflammation: A Systematic Review.” *PLOS ONE*, edited by Irina Kerkis, vol. 11, no. 11, 2016, p. e0167289. *Crossref*, doi:10.1371/journal.pone.0167289. (3)  |