Necrosis - a disruptive form of cell death.

Nathan Copling*

Faculty of Dentistry, McGill University, 2001 McGill College Avenue, Montreal, QC, H3A 1G1, Canada

Introduction

Cell death is a fundamental biological process that occurs throughout an organism's life cycle. One of the most disruptive forms of cell death is necrosis. Unlike apoptosis, a controlled and programmed cell death that is essential for maintaining tissue homeostasis, necrosis represents a chaotic and unregulated demise of cells. This form of cell death can have severe consequences for the surrounding tissues and has been linked to various pathological conditions. Necrosis is a result of cell injury or damage caused by a myriad of factors, such as infections, toxins, trauma, lack of oxygen (hypoxia), or exposure to extreme temperatures. Unlike apoptosis, necrosis does not follow a predefined and organized pathway. Instead, it occurs abruptly and leads to the rupture of the cell membrane, releasing intracellular content into the surrounding tissue. This uncontrolled release triggers an inflammatory response, further exacerbating the damage [1].

Morphologically, necrosis can be identified by certain characteristic features. Under a microscope, necrotic cells appear swollen, with disrupted organelles and a denser cytoplasm. The nucleus often shows changes, such as pyknosis (condensation), karyorrhexis (fragmentation), or karyolysis (dissolution). In some cases, necrosis may lead to the formation of a coagulative necrosis, where the tissue maintains its overall structure but with altered texture and appearance. This form is commonly seen in ischemic conditions, where the lack of oxygen leads to denaturation of proteins, resulting in a coagulation-like appearance of the affected tissue. Often observed in infections, this type of necrosis occurs when enzymes released by immune cells lead to the rapid breakdown of cellular components, creating a liquid, pus-like substance [2].

Characteristic of certain granulomatous infections, this type is named for the cheese-like appearance of the necrotic tissue. Gangrenous Necrosis: Usually occurs in extremities and results from a combination of coagulative and liquefactive necrosis, often seen in conditions like severe frostbite or arterial occlusions. Fat Necrosis: Typically found in adipose (fat) tissue, this type occurs when lipase enzymes break down triglycerides, leading to the formation of fatty acids that complex with calcium, forming chalky deposits. Necrosis can be detrimental to the organism, as it can disrupt tissue integrity, impair organ function, and initiate an inflammatory response. The release of intracellular contents, such as damage-associated molecular patterns (DAMPs), can activate immune cells, further exacerbating tissue damage and inflammation. If necrosis is widespread or affects vital organs, it can have life-threatening consequences [3].

Detecting necrosis is crucial for understanding the progression of various diseases. Diagnostic techniques such as biopsies and medical imaging are used to identify necrotic regions within tissues. In clinical practice, efforts are focused on managing the underlying cause of necrosis, promoting tissue repair, and reducing inflammation. During necrosis, various cellular events contribute to the ultimate demise of the cell. As the cell undergoes stress or injury, the mitochondria, often referred to as the powerhouse of the cell, can be severely affected. Mitochondrial dysfunction can lead to the generation of reactive oxygen species (ROS) and the collapse of the electrochemical gradient across the mitochondrial membrane, which ultimately disrupts energy production. The increase in intracellular calcium is another crucial event in necrosis. Elevated calcium levels trigger the activation of various enzymes, such as proteases and phospholipases, which can break down cellular structures and contribute to cell damage [4].

Necrosis elicits a potent inflammatory response in the affected tissues. The release of DAMPs, such as high mobility group box 1 (HMGB1) and heat shock proteins (HSPs), can activate pattern recognition receptors (PRRs) on neighboring immune cells. This, in turn, triggers the production of pro-inflammatory cytokines and chemokines, leading to the recruitment and activation of immune cells like neutrophils and macrophages. Necrosis plays a significant role in the pathogenesis of various diseases. In the context of ischemic events, such as myocardial infarction (heart attack) or stroke, necrosis in affected tissues can lead to permanent damage and loss of function. Additionally, necrosis is involved in the progression of autoimmune diseases, neurodegenerative disorders, and certain types of cancer [5].

Conclusion

Necrosis represents a disruptive form of cell death that can have severe consequences for the affected tissues and the overall health of an organism. Understanding the mechanisms and identifying the types of necrosis is crucial for diagnosing and managing various pathological conditions. Further research in this field may pave the way for developing targeted therapies and interventions to mitigate the detrimental effects of necrosis on the human body.

***Correspondence to:** Nathan Copling , Faculty of Dentistry, McGill University, 2001 McGill College Avenue, Montreal, QC, H3A 1G1, Canada. E-mail: nathancopling@gmail.com

***Received:** 27-Jun-2023, Manuscript No. JMOT-23-109768; **Editor assigned:** 28-Jun-2023, PreQC No. JMOT-23-109768 (PQ); **Reviewed:** 17-Jul2023, QC No. JMOT-23-109768; **Revised:** 19-Jul-2023, Manuscript No. JMOT-23-109768 (R); **Published:** 29-Jul-2023, DOI: 10.9768/jmot-8.4.155*

***Citation:** Copling N. Necrosis - a disruptive form of cell death. J Med Oncl Ther. 2023;8(4):155*

References

1. Peter ME. Apoptosis meets necrosis. Nature. 2011;471(7338):310-2.
2. Old LJ. Tumor necrosis factor. Scientific American. 1988;258(5):59-75.
3. Waters JP, Pober JS, Bradley JR. Tumour necrosis factor and cancer. J Pathol. 2013;230(3):241-8.
4. Edinger AL, Thompson CB. Death by design: apoptosis, necrosis and autophagy. Curr Opin Cell Biol 2004;16(6):663-9.
5. Tang P, Hung MC, Klostergaard J. Human pro-tumor necrosis factor is a homotrimer. Biochemistry. 1996;35(25):8216-25.

Citation: *Copling N. Necrosis - a disruptive form of cell death. J Med Oncl Ther. 2023;8(4):155*