Histopathological Response of Breast Cancer Tissue to Cold Atmospheric Plasma (CAP) Exposure

Inas Abd Al Majed Rasheed, M.B.Ch.B , MSc, PhD* Anas Ahmed Saleh, F.I.C.S.M, C.A.M.B** Ahmed Salim Khazaal, F.I.C.M.S(GS)**

ABSTRACT

Cold Atmospheric Plasma (CAP) has emerged as a promising therapeutic modality due to its ability to induce oxidative stress and apoptosis in malignant tissues with minimal impact on healthy cells(1,2,3,4). This study aimed to evaluate the histopathological and biochemical responses of human breast cancer tissue to CAP exposure, specifically focusing on key oxidative stress and apoptotic markers: malondialdehyde (MDA), glutathione (GSH), and caspase-3. A total of 50 freshly excised breast tissue samples (25 malignant and 25 benign fibroadenomas) were subjected to standardized CAP treatment using a dielectric barrier discharge system. Biochemical analyses were performed on tissue lysates to quantify baseline and post-treatment levels of caspase-3, GSH, and MDA. At baseline, malignant tissues demonstrated significantly elevated levels of caspase-3 and MDA and lower GSH levels compared to benign controls, indicating higher apoptotic activity and oxidative stress. Following CAP exposure, malignant tissues showed further increases in caspase-3 and MDA and a marked decrease in GSH, suggesting an intensified pro-apoptotic and oxidative effect. No post-treatment data were available for benign tissues due to the study design. Linear and multivariate regression analyses revealed that baseline GSH and MDA levels significantly predicted post-CAP biochemical changes, especially the magnitude of GSH depletion and MDA elevation. These findings underscore the potential of CAP as a targeted therapeutic strategy for breast cancer by exploiting redox imbalance and apoptotic vulnerabilities in malignant cells. The distinct biochemical profile in response to CAP treatment supports further exploration of CAP in oncologic applications and highlights its selective impact on cancerous tissue.

Bahrain Med Bull 2026; 48 (1): 2807-2814

* Department of Pathology, College of Medicine
Tikrit University, Iraq.
Email: dr.enas11@tu.edu.iq

** Department of Surgery, College of Medicine
Tikrit University, Iraq.