

## ABSTRAK

**Latar belakang:** Luka bakar akibat asam format diklasifikasikan sebagai cedera kimia yang bersifat progresif melalui mekanisme kerusakan jaringan yang masih belum banyak diteliti. Beberapa laporan kasus menggambarkan konsekuensi serius seperti nekrosis yang mendalam dan cacat permanen. Namun hingga kini tidak ada studi komprehensif yang mengintegrasikan pendekatan histopatologi dan molekuler untuk menjelaskan mekanisme dimaksud. Penelitian ini bertujuan untuk membuktikan kerusakan jaringan kulit akibat paparan asam format melakukan kajian histopatologi, dan menganalisis stres oksidatif, serta ekspresi molekul jaras inflamasi dan nekrosis.

**Metode:** Penelitian eksperimental *in vivo* menggunakan model babi (*Sus scrofa domestica*) yang dipaparkan asam format selama 3 menit. Sampel kulit dibagi menjadi 3 kelompok meliputi kulit normal (kontrol negatif), paparan zat besi panas (kontrol positif), dan paparan asam format (P), observasi dilakukan 30 menit, 1 jam, 3 jam, dan 6 jam pascapaparan. Evaluasi dengan analisis histopatologi di bawah mikroskop cahaya menggunakan pewarnaan H&E, pengukuran kadar 8-isoprostane sebagai indikator stres oksidatif melalui metode ELISA, ekspresi NF- $\kappa$ B dan HMGB1 sebagai indikator jaras inflamasi dan nekrosis di tingkat hulu (awal respons) melalui pewarnaan imunohistokimia.

**Hasil:** Kajian histopatologi menunjukkan kerusakan struktur kulit signifikan pada area paparan. Kadar 8-isoprostane meningkat secara signifikan, mencerminkan stres oksidatif yang bersifat akut. Peningkatan ekspresi NF- $\kappa$ B dan HMGB1, mengindikasikan adanya aktivasi respons inflamasi dan jaras nekrosis.

**Kesimpulan:** Paparan asam format selama 3 menit, menunjukkan kerusakan jaringan kulit progresif melalui stres oksidatif dan jaras inflamasi diikuti aktivasi jaras nekrosis yang signifikan.

**Kata Kunci:** Luka bakar kimia, Asam format, Histopatologi, 8-isoprostane, NF- $\kappa$ B, HMGB1.

## ABSTRACT

**Background:** Chemical burns due to formic acid are classified as progressive injuries. However, the mechanisms of tissue damage need to be elucidated. A few cases reported profound impact, such as deep necrosis and permanent disabilities. Nevertheless, no comprehensive studies have integrated histopathological and molecular aspects. This study aimed to determine the impact on skin following formic acid exposure through histopathological features, analyzing oxidative stress, and the early proinflammatory mediators and necrosis signals.

**Method:** This experimental study used *Sus scrofa domesticus* as a model. The focus of interest was divided into three groups: normal skin (negative control), exposure to a hot iron plate (positive control), and formic acid (FA). The specimens were taken in 30 minutes, 1 hour, 3 hours, and 6 hours for investigation. They were subjected to 1) histopathological studies using a light microscope with H&E staining, 2) the level of 8-isoprostane as an indicator of oxidative stress with the ELISA method, level of NF- $\kappa$ B as an indicator of inflammatory response, and HMGB1 as an indicator of necrotic signal through immunohistochemical studies.

**Results:** The study findings show significant skin damage histologically in the exposed area. The levels of 8-isoprostane, representing oxidative stress, increased significantly; NF- $\kappa$ B representing activation of inflammatory response and early activation of HMGB1, indicating a necrosis signal.

**Conclusion:** Exposure to formic acid for 3 minutes shows a progressive response of skin tissue damage through significant oxidative stress and inflammatory pathways and signals of necrosis in pig skin.

**Keywords:** Formic acid burns, histopathology, 8-isoprostane, NF- $\kappa$ B, HMGB1.