Immediate treatment with inhaled hydrogen (H₂) has been demonstrated to attenuate oxidative stress conditions and inflammatory cytokines in serum after resuscitation from cardiac arrest. However, applying inhaled H₂ early is not practical in out of hospital cardiac arrest due to safety issues. In the present study, we investigated the effects of delayed treatment with H₂ on oxidative stress and inflammatory cytokines in a porcine model of cardiac arrest (CA). We hypothesized delayed inhalation of H₂ reduces proinflammatory cytokines but has no effect on oxidative stress after resuscitation in a CA porcine model.

Methods

Animal Model

Twenty male domestic pigs weighing 39±2 kg were studied. Ventricular fibrillation (VF) was induced electrically and CPR was initiated after 10 minutes of untreated VF. Animals were randomized into two groups immediately after successful resuscitation: delayed inhalation of H₂ (DH group) or continuous inhalation of room air (C group). DH group animals were ventilated with 2% H₂/21% oxygen from post-resuscitation 120 minutes (PR120) until PR240 minutes. Serum levels of oxidative product (8-iso-PGF2α) and proinflammatory cytokines (tumor necrosis factor-α [TNF-α], interleukin-6 [IL-6], and high-mobility group box protein 1 [HMGB1]) were measured by ELISA at baseline (BL), PR30, PR180 and PR360.

Results

Figure 1. Changes in serum biomarkers.

Levels of: (A) 8-iso-PGF2α; (B) IL-6; (C) TNF-α; (D) HMGB1. HMGB1, high mobility group box 1; BL, baseline; PR, post-resuscitation; C group, control group; DH group, two hours delayed inhalation of H₂ group.

# p < .05 vs. BL, * p < .05 vs. the C group.

Conclusions

Delayed treatment with H₂ attenuates proinflammatory cytokines without an effect on oxidative stress conditions after resuscitation.