Aim: to determine the effects of delayed fluid resuscitation on the lung oxidative stress and antioxidant vitamin levels in a rat model of controlled hemorrhagic shock. Material and Methods: Wistar male rats were randomized to three groups as follows: (1) control (n=5), (2) volume-controlled hemorrhagic shock (HS) for 30 minutes and resuscitated 30 minutes (early resuscitation, HS-ER) (n=6), and (3) 90 minutes (delayed resuscitation, HS-DR) (n=6) after hemorrhage. After 24 hours, bronchoalveolar lavage fluid (BALF) was obtained. The right lung was used for biochemical analysis and the left one for histopathological analysis. The slides were subsequently graded by a pathologist using a modified histologic score without prior knowledge of treatment groups (0= no injury; 1=scant, 2= moderate, 3= severe). The lipid peroxidation (MDA), reduced glutathion (GSH), glutathion peroxidase (GSH-Px) and Vitamins A, C and E values were also measured in the BALF and lung homogenate. Results: Interstitial septum thickening, alveolar/interstitial PNL infiltration, and total lung score were higher in hemorrhagic shock groups than control group (p=0.004, p=0.006, p=0.002 and p=0.002, respectively). HS-DR group induced more prominent leukocyte accumulation and lung injury than in HS-ER group (p=0.008 and p=0.026). Lung tissue GSH-Px and vitamin E levels are increased in hemorrhagic groups compared to control group (p=0.027 and p=0.001, respectively). We found a significant increase in GSH-Px activity but not any differences in MDA, GSH, Vit-A and Vit-C in BALF of the groups (p=0.007). Conclusion: We observed that fluid resuscitation in hemorrhagic shock induce antioxidant effect by increasing GSH-Px and vitamin E values. In addition this antioxidant activities were correlated with the time delayed for resuscitation.