Background

Oxygen (O2) is undoubtedly essential for human beings and the consequences associated with hypoxemia is devastating. While O2 has been most widely prescribed for therapy in medicine (Bateman NT. 9740573), there is an emerging concern that hyperoxia and hyperoxemia could also embrace potential detrimental systemic effects (Hafner. 26585328), involving numerous injurious pathways including the development of oxidative stress and cellular damage caused by excess reactive oxygen species (Laffey. 12131125). Recently, the potential harmful impact of oxygen has been studied in clinical trials and advocated in specific and non-specific population; cardiopulmonary resuscitation (Kilgannon, 21606393) (Kilgannon, 20516417) (Janz. 22971589), stroke (Padma. 21264137) (Ronning. 10512903), myocardial infarction (Sub. 26002889), traumatic brain injury (Rincon. 23794718), mechanical ventilation (de Jonge. 19077208), and medical-surgical intensive care (Girardis. 27706466).

Considering the suggested injurious mechanisms such as excessive reactive oxygen species production induced by excess oxygen exposure and the systemic effect of oxidative stress (Brueckl. 16357365) (Fessel. 12482927), kidney could be one of the targeted organs (Francis. 28641323) (Hsia. 28347910). Some animal studies have shown the detrimental effect of hyperoxia on renal tissue through protein expression associated with inflammation and imbalance of renal oxygen delivery and demand (Hinkelbein. 26106253) (Pohlmann. 26676131). A human study showed that intraoperative oxidative damage, which could be induced by hyperoxia, independently predicts AKI following cardiac surgery (Billings. 22626819). In addition, hyperoxia increases peripheral valscular resistance (Harten. 12911363) (Harten. 15868523), which could lead hypoperfusion of kidney and cause kidney injury.

Since significant hypoxemia can quickly lead to fatal event and large amount of hemorrhage can lower the ability of oxygen carriage by hemoglobin, early aggressive supplemental oxygen is commonly provided either in response to or for prevention of dangerous reductions in the arterial partial pressure of oxygen for traumatic patients. However, the impact of keeping possible supranormal arterial blood oxygen tensions during stay in emergent department and after admission on kidney function is unclear.

We hypothesize that hyperoxemia increase renal dysfunction in traumatic patients. The aim of this study is 1) to survey the prevalence and the degree of hyperoxemia among patients in an emergency department and an intensive care unit 2) to investigate the association between partial arterial oxygen and the development of acute kidney injury.

※the prevalence and the degree of hyperoxemia among patients in an emergency department is unclear

※FiO2 is difficult to measure.

Primary outcome: AKI (KDIGO) vs. PaO2

Secondary outcomes: Mortality vs. PaO2