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The acute pulmonary and prothrombotic effects of cerium oxide nanoparticles following intratracheal instillation in mice

Abstract *

Cerium oxide nanoparticles (CeO₂ NPs), used as a diesel fuel catalyst, are emitted into the ambient air resulting in human exposure by inhalation. Recent studies reported the development of lung toxicity after exposure to CeO₂ NPs. However, little is known about the thrombotic effects of CeO₂ NPs. Here, we investigated, in mice, the acute (24 h) effect of intratracheal instillation of either CeO₂ NPs (0.1 or 0.5 mg/kg) or saline (control) on pulmonary and systemic inflammation and oxidative stress and thrombosis. CeO₂ NPs induced an increase of neutrophils into the bronchoalveolar lavage (BAL) fluid with elevation of tumor necrosis factor α (TNF α) and a decrease in the activity of catalase. Lung sections of mice exposed to CeO₂ NPs showed a dose-dependent infiltration of macrophages and neutrophils. The plasma levels of C-reactive protein and TNF α were increased whereas the activities of catalase and total antioxidant were decreased. Interestingly, CeO₂ NPs dose-dependently enhanced thrombosis in pial arterioles and venules. Moreover, the plasma concentrations of fibrinogen and plasminogen activator inhibitor-1 were elevated by CeO₂ NPs. The direct addition of CeO₂ NPs to mouse whole blood *in vitro* neither caused platelet aggregation nor affected prothrombin time or partial thromboplastin time, suggesting that the thrombotic events observed *in vivo* may have resulted from systemic inflammation and/or oxidative stress induced by CeO₂ NPs. We conclude that acute pulmonary exposure to CeO₂ NPs induces pulmonary and systemic inflammation and oxidative stress, and promotes thrombosis *in vivo*.

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