

Sodium Hypochlorite Inhalation: Mechanism for Extrapulmonary Vascular Injury

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Learning Objective:

- Unintentional chlorine gas inhalation can induce an Acute Coronary Syndrome.
- The extrapulmonary vasculature is susceptible to the acute injury and the ticking time bomb of a hypercoagulable state.

Case Description:

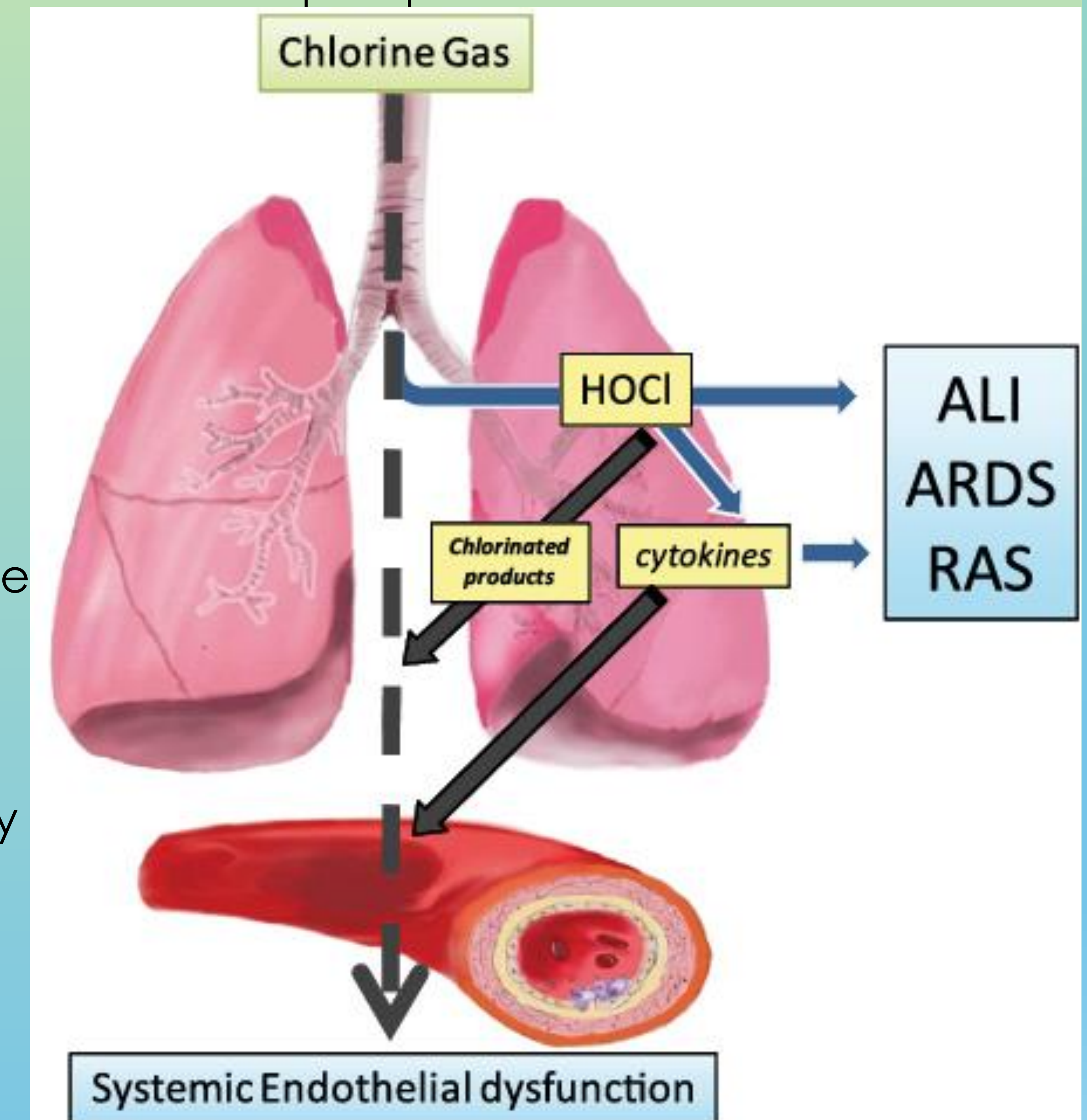
- Patient is a 68 year old female (designated as an American Society of Anesthesiology Class 1 patient) with no past medical history, no known coronary artery disease and no medication.
- Patient is presented on the floor in a hotel room, flat on her back complaining of unrelenting chest pain, extreme weakness, nausea, vomiting and extreme diaphoresis.
- Palpation of the left radial arterial pulse revealed a regular rhythm which becomes weak and thready.
- EMS monitor reveals ST elevation in multiple leads.
- Cardiac Catheterization: Complete and single occlusion of the right coronary artery consistent with thrombus formation no plaque observed.
- Right coronary stent deployed and patient suffered ventricular fibrillation, intubation, CPR and shock 3x, and aortic balloon pump inserted.
- Echocardiogram follow-up reveals: EF = 65%, good left ventricular function with no hypokinesis.
- Patient survives to walk out of hospital.

Discussion:

- Chlorine gas is known to cause systemic endothelial dysfunction.
- Endothelial injury from accidental inhalation of chlorine gas in both toxic and subtoxic exposures can result in delayed harmful effects.
- A hypercoagulable state indolently develops to manifest as thrombus in the extrapulmonary vasculature.
- The injury to the epithelium causes dysfunction to the nitric oxide vascular homeostasis mechanism.
- The vascular endothelium is now predisposed to a proinflammatory and procoagulant state ripe for thrombus formation.
- The “Von Willebrand factor” is critical in arterial thrombogenesis and has been shown to be elevated in chlorine gas inhalation reflecting extensive vascular endothelial dysfunction.

Conclusion:

- This case reflects an acquired thrombogenic inflammatory response through chlorine gas exposure.
- Delayed consequences on the extrapulmonary vasculature endothelial can still occur even if there is no history of heart disease.



References: (1) Suzuki S. et al Case Report Fatal Pulmonary Arterial Thrombosis Associated With Chlorine Gas Poisoning. Clin Appl Thrombosis/Hemostasis. 2001, 7 (4):356-3582; (2) Samal A. et al Potential for Chlorine Gas Induced Injury in the Extrapulmonary Vasculature. Pro Am Thorac Soc. 2010, Vol 7,290-293; and (3) Honavar J. et al Chlorine Gas Exposure Causes Systemic Endothelial Dysfunction by Inhibiting Endothelial Nitric Oxide Synthase-Dependent Signaling. Am J Respir Cell Mol Biol 2011, Vol 45, 419-425.