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A mechanistic investigation of thrombotic microangiopathy associated with IV abuse of Opana ER.

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Abstract

Since 2012, a number of case reports have described the occurrence of thrombotic microangiopathy (TMA) following IV abuse of extended-release oxymorphone hydrochloride (Opana ER), an oral opioid for long-term treatment of chronic pain. Here, we present unique clinical features of 3 patients and investigate IV exposure to the tablet's inert ingredients as a possible causal mechanism. Guinea pigs were used as an animal model to understand the hematopathologic and nephrotoxic potential of the inert ingredient mixture (termed here as PEO+) which primarily contains high-molecular-weight polyethylene oxide (HMW PEO). Microangiopathic hemolytic anemia, thrombocytopenia, and acute kidney injury were found in a group of 3 patients following recent injection of adulterated extended-release oxymorphone tablets. Varying degrees of cardiac involvement and retinal ischemia occurred, with TMA evident on kidney biopsy. A TMA-like state also developed in guinea pigs IV administered PEO+. Acute tubular and glomerular renal injury was accompanied by nonheme iron deposition and hypoxia-inducible factor-1α upregulation in the renal cortex. Similar outcomes were observed following dosing with HMW PEO alone. IV exposure to the inert ingredients in reformulated extended-release oxymorphone can elicit TMA. Although prescription opioid abuse shows geographic variation, all physicians should be highly inquisitive of IV drug abuse when presented with cases of TMA.

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