Human Wound Site Tissue Contaminated with Actinide Material

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An accident involving insults such as physical trauma can induce several types of wounds, i.e., incisions, punctures, burns, lacerations, and abrasions. Moments after the wound has occurred, disruption and destruction of local tissue morphology and histology and blood vessels initiated a cascade of events that lead ultimately to repair of the injury site. However, the exact nature or synergy of the physiological response that occurs when an acute wound produce by a cut, puncture, abrasion, etc., is accompanied by hot acidic or corrosive solutions that may or may not contain radioactive materials, i.e., Pu, Am or U is largely unknown. It is known however that cell sensitivity to radiation varies extensively throughout the cell cycle. As a consequence, cells alter their transit through the cell cycle after exposure to radiation. Such exposed cells may delay in G1, S, G2 or mitosis and thus affect the normal wound healing process. Consequently, chronic irradiation from extravasated deposits of thorium dioxide has induced delayed wound healing at the site of Thorotrast injections in some patients. However, how the resulting dose and or dose rate from embedded particles of actinide material affect the overall multi-step wound repair process remains poorly understood. Pathological examination of wound site tissue contaminated with actinide material from three USTUR Registrant cases were examined to evaluate the wound site healing process following decades of low-level alpha irradiation in situ. Other than the thorotrastoma that developed at the site of Thorotrast injection, wound site tissue pathology in the other two-Registrant cases was normal. Radiochemical analysis of wound site tissue areas disclosed actinide values comparable to that level found in normal muscle and skin.

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