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Determination of factors that lead to enhanced infection & impaired healing in diabetic wound

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Diabetic foot ulcers are the leading cause of lower extremity amputations in the US and are responsible for more hospitalizations than any other complication of diabetes. The sheer number of diabetic ulcers that progress to amputation underscores the inadequacy of conventional therapies and the need for novel approaches. Bacterial infection and hyper-inflammation are two major co-morbidities associated with impaired healing in diabetic chronic ulcers. However, the mechanism(s) underlying these effects remain poorly understood. Most studies have focused on old chronic diabetic wounds and found these ulcers to be infected and locked in a hyper-inflammatory mode. However, very little is known about the dynamics of inflammatory responses and infection control early after injury in diabetic wound. We have used a well-established wound model for type II diabetes, (db/db mouse and their normal littermates, C57BL/6), to study the early dynamics of bacterial infection control in diabetic and normal wound tissues. Our data demonstrate that diabetic skin harbors a significantly higher number of bacteria prior to injury and is severely defective in preventing bacterial colonization in a manner that is independent of its initial high bacterial loads. Surprisingly, we have found that unlike chronic diabetic ulcers which are known to be in a persistent inflammatory state, the acute phase of inflammation, which is needed to counter invading pathogens, is significantly delayed in diabetic wounds early after injury. This delay in inflammatory responses is partly due to reduced chemokine expression and partly due to impaired chemotactic response in diabetic leukocytes during the acute phase of healing early after injury. Our data indicate that reduced inflammatory responses early after injury in diabetic wounds is just as harmful as the persistent and hyper-inflammatory state that dominates diabetic wounds as they become chronic. Importantly, onetime treatment with a pro-inflammatory chemokine, CCL2, was able to jumpstart inflammatory response and significantly stimulated wound healing. Chemokine based therapy may offer an alternative approach to stimulate wound healing in diabetic ulcers.

Biography

Dr. Sasha Shafikhani has completed his PhD from University of California at Berkeley and postdoctoral studies from University of California at San Francisco. He has published more than 22 papers in reputed journals and has been serving as an editorial board member of several reputed journals. As a cellular microbiologist, Dr. Shafikhani focuses his research on immune dysregulation that renders diabetic wound vulnerable to infection and microbiome shift toward pathogenic bacteria. He also studies the virulence strategies that pathogenic bacteria use to drive a diabetic wound toward non-healing chronic state.

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