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Researchers discover underlying mechanisms of skin hardening syndromes

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Researchers from Boston University School of Medicine (BUSM) have discovered new details about the underlying mechanisms of skin hardening syndromes. The team connected pharmacological properties of the Novartis Pharma AG drug called balicatib to the skin disorder for the first time after investigating adverse reactions suffered by patients participating in a clinical trial for the treatment of osteoporosis. These findings appear online in the *Journal of the American Academy of Dermatology.*

Balicatib was developed recently as an osteoporosis drug that can inhibit CathepsinK (catK), an enzyme involved with bone degradation. In a recent trial however, several patients on balicatib experienced hardening of the skin, most frequently around the neck, chest and abdomen. After examining the cases and relating them to recent reports of cathepsin K expression in the skin and the role of cathepsin K in degrading collagen and elastin, the investigators determined that the changes were a direct effect of the drug.

This case study adds a new class of medication to the short list of agents that induce skin hardening syndromes. It also proves that catK affects the skin as well as bones, and marks the first time that skin hardening can be convincingly linked to the pharmacologic properties of a drug.

"This observation emphasizes the importance of intracellular collagen degradation in the skin, a pathway so far vastly underappreciated," said Thomas Ruenger, MD, PhD, a professor and vice-chair of dermatology

at BUSM. "This observation also sheds new light on our understanding of the mechanisms involved in morphea, or skin hardening. Failed collagen degradation has so far not been thought to cause morphea."

The researchers believe these findings have far-reaching implications for osteoporosis patients and those suffering from skin hardening syndromes.

Provided by Boston University Medical Center