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Resumo	Men with sickle cell disease (SCD) frequently experience priapism, defined as prolonged, painful erections occurring without sexual arousal or desire. This urological emergency can lead to penile fibrosis and permanent erectile dysfunction if not treated adequately. Due to its complex pathophysiology, there is currently no effective preventative treatment for this condition. Recent studies have highlighted the dysfunction of the nitric oxide (NO) and cyclic guanosine monophosphate (cGMP) pathway in erectile tissues as a critical mechanism in developing priapism in SCD. Additionally, further research indicates that intravascular hemolysis promotes increased smooth muscle relaxation in the corpus cavernosum and that excess heme may significantly contribute to priapism in SCD. Pharmacological treatments should ideally target the pathophysiological basis of the disease. Agents that reduce excess free heme in the plasma have emerged as potential therapeutic candidates. This review explores the molecular mechanisms underlying the excess of heme in SCD and its contribution to developing priapism. We discuss pharmacological approaches targeting the excess free heme in the plasma, highlighting it as a potential therapeutic target for future interventions in managing priapism.
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