# Association of Angiotensin Converting Enzyme Insertion/Deletion Polymorphism with Vaso-occlusive Complications of Sickle Cell Anaemia

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**Background:** Angiotensin I-converting enzyme (ACE), dipeptidyl peptidase, is a membrane-bound enzyme, which is present in endothelial and epithelial cells of various tissues, and innards including lungs and kidneys. ACE converts angiotensin I to II, a very potent vasoconstrictor agent . Angiotensin is a hormone as well as a locally produced cellular factor, directly affecting vascular endothelial cells and smooth muscles. Furthermore, it has been demonstrated that receptors of angiotensin II are found in the atherosclerotic vessel walls. It is pointed out that angiotensin II can promote vasoconstriction, inflammation, and thrombosis in the vascular endothelium and vessel walls. Besides being a potent vasoconstrictor, angiotensin II is a proatherogenic agent, which elevates plasminogen activator inhibitor-1 levels, which results in a decrease in the fibrinolytic activityhe ACE I/D polymorphism is an insertion/deletion of an ALU-repeat sequence of 287 base pairs (bp) in intron 16 of the ACE gene, located at 17q23. This results in three genotypes: II, ID, and DD.] Previous studies have reported that plasma levels of angiotensin II are closely associated with ACE insertion/deletion (I/D) polymorphism and that the serum level of ACE is likely to increase 2-fold in the presence of ACE D/D polymorphism, consequently increasing the levels of plasma angiotensin II.