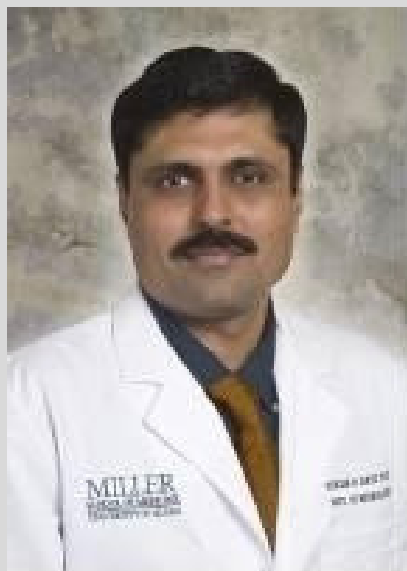


Wallace H. Coulter Foundation Lecture Series

Intracerebral Hemorrhage and Therapeutic Targets



Kunjan Dave

Research Assistant Professor of
Neurology, University of Miami
Miller School of Medicine

Lecture: Friday, March 9, 2018
9:00AM-10:00AM
Room EC 2300
10555 West Flagler Street
Miami, FL 33174

Biography

Dr. Kunjan R. Dave is a Research Assistant Professor of Neurology, University of Miami Miller School of Medicine. Dr. Dave received his Ph.D in Biochemistry in 2000 from the M. S. University of Baroda, India. During his PhD training he worked on several research projects including secondary complications of diabetes, Alzheimer's disease and drug toxicity among others. Dr. Dave briefly worked at the Zandu Pharmaceutical Works, Mumbai, India, as a Biochemist, where he participated in a drug development program. Dr. Dave then joined the Department of Neurology, University of Miami as a post-doctoral fellow with Dr. Miguel A. Perez-Pinzon. Dr. Dave has performed research essential for the understanding cerebral ischemia pathophysiology and Amyotrophic Lateral Sclerosis. The goal of Dr. Dave's research is to study potential signaling pathways responsible for neuronal death in neurodegenerative diseases, especially cerebral ischemia. Investigation of intracellular signaling pathways may lead to the development of novel therapies for patients with neurodegenerative diseases and stroke. His research also includes evaluating novel strategies to lower damage following intracerebral hemorrhage.

Abstract

The spontaneous intracerebral hemorrhage (ICH) accounts for 10-15% of all strokes affecting about 2 million people worldwide. The sICH is the deadliest stroke subtype. One month mortality is about 40% and most of the survivors remain disabled. Major mechanisms of primary injury include hematoma expansion, acute hypertensive response, increased intracranial pressure, mechanical disruption of brain cells. While major mechanisms of secondary injury include activation of pro-inflammatory pathways, disruption of the blood-brain barrier, formation of brain edema, release on blood products during hematoma resolution. Although multiple mechanisms of injury and therapeutic strategies have been identified to treat sICH in preclinical studies, none of the therapeutic strategies has translated in clinical settings. Because of no proven therapy, clinicians are not able to offer more than supportive care for this deadlines stroke subtype. Continued cerebral bleeding leading to hematoma expansion is highest in the first 3 hours after symptom onset and may continue in 40% of patients between 3 and 24 hours after the onset. The prevention of hematoma expansion in sICH has been an attractive therapeutic target. Red blood cell microparticles have the potential to be used as a therapeutic agent in sICH since they promote clotting at sites of active bleeding, in part, by interaction with platelets and by amplifying activation of the contact pathway. This talk will discuss strategy to lower hematoma growth in sICH using red blood cell microparticles.