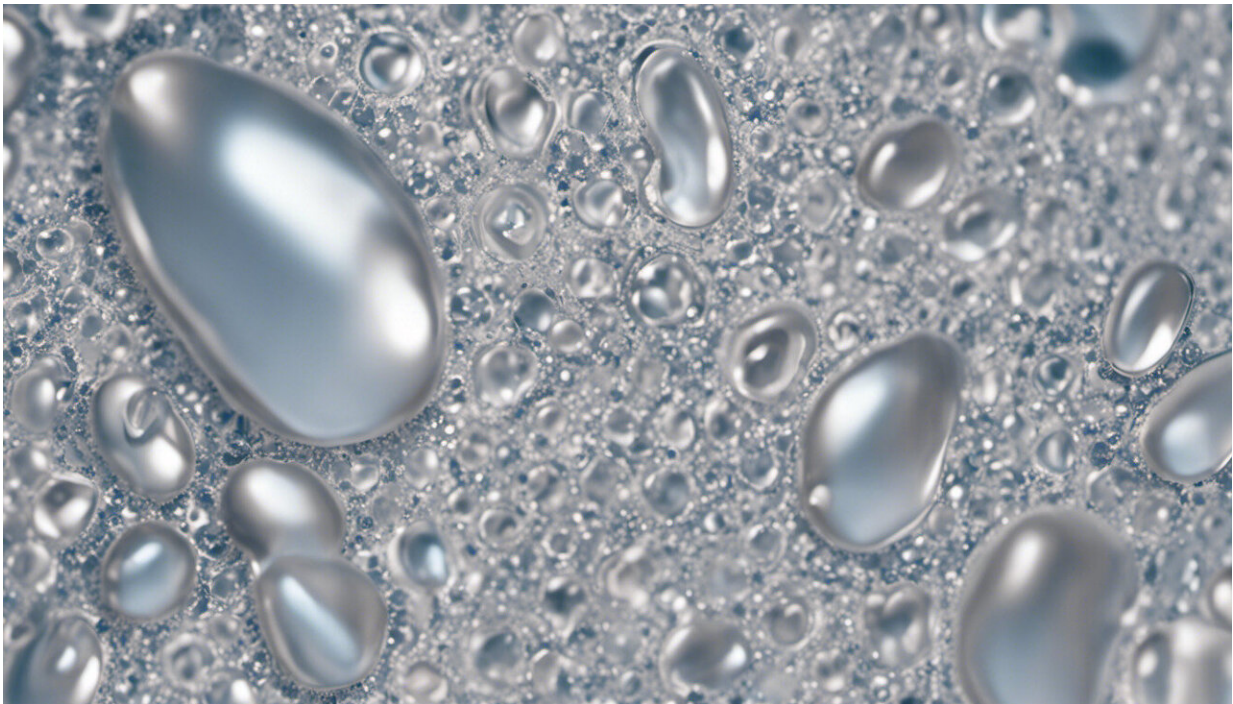


Mechanical basis for abdominal aortic embolism discovered

January 27 2022



Credit: AI-generated image ([disclaimer](#))

Abdominal aortic aneurysm (AAA) is a complex and life-threatening vascular disease with high incidence worldwide. Termed the silent killer, most AAAs are asymptomatic, often going undetected until rupture, and involve a poorly understood set of mechanical and biochemical events. Epidemiologic studies have established associations between AAA and

both vascular inflammation and increased stiffness. That the latter is concomitant with aging explains, in part, why AAA affects almost exclusively those over 65 years of age.

Evidence suggests that abnormal acclimation of vascular smooth muscle cells (VSMC) to biomechanical perturbations, such as increased circumferential stress in hypertension, can stimulate AAA development. However, there is a paucity of knowledge of the molecular drivers of altered mechanobiological behaviors of VSMC. Understanding these might provide promising targetable signals that could repress AAA progression and limit rupture incidents.

Now, researchers at NYU Tandon and NYU Langone have demonstrated mechanobiological changes in VSMC and identified a key ion channel that is involved in the development of AAA. In a new study, published in *Nature Communications*, they describe the means by which VSMC gradually adopt a solid-like state by upregulating cytoskeleton crosslinker, α -actinin2, which powers the mechanosensitive ion channel Piezo1.

"Our team applied biomechanical engineering to study aneurysm pathology," explained Chen. "In contrast to the extensive study of aorta wall properties, we explored how a cell's mechanical sensitivity, or 'mechanosensation' to mechanical stimuli presents an innovative perspective in revealing disease pathogenesis and progression mechanisms."

The researchers measured misshapen VSMC with a novel ultrasound tweezers system and a single-cell RNA sequencing technique. Their findings pointed to Piezo1, which critically regulates VSMC mechanical sensitivity. They also found that inhibition of Piezo1 prevents mice from developing AAA by alleviating pathological vascular remodeling. The findings concluded that deviations of mechanosensation behaviors of

VSMC is detrimental for AAA and identifies Piezo1 as a novel culprit of mechanically fatigued aorta in AAA. This could lead to new mechano-medical approaches to treating this devastating cardiovascular disease.

More information: Weiyi Qian et al, Microskeletal stiffness promotes aortic aneurysm by sustaining pathological vascular smooth muscle cell mechanosensation via Piezo1, *Nature Communications* (2022). [DOI: 10.1038/s41467-021-27874-5](https://doi.org/10.1038/s41467-021-27874-5)

Provided by NYU Tandon School of Engineering

Citation: Mechanical basis for abdominal aortic embolism discovered (2022, January 27)
retrieved 6 May 2024 from
<https://medicalxpress.com/news/2022-01-mechanical-basis-abdominal-aortic-embolism.html>

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