Role of MicroRNA in Cardiac Anesthesia: An Innovative Consequences and New Possibility

The Editor,

“One thing I have learned in a long life: That all our science, measured against reality, is primitive and childlike – and yet it is the most precious thing we have” (“Albert Einstein: Creator and Rebel,” 1972).

Following this statement, we should inspire for work hard and concussive to our scientific work for patients management and translational research. MicroRNAs (miRNA) are basically endogenously expressed through the noncoding short single-stranded RNAs. These non-coding and very short single-stranded RNAs play a significant role in the gene regulation, gene expression, and posttranscriptional modification. Another way these short molecules degrade easily and help to translate the inhibition pattern of target mRNAs. miRNA were rapidly growing and significant contribution in ischemic heart disease (IHD). Again, miRNAs are important candidates for stroke and heart failure. It acts unlike silent killer and promotes to so many other additional diseases. Till date, its molecular mechanism and disease progression in human subject poorly understood. As per the new therapeutics ability concerned these small molecules regulated the very specific target genes and capable targeting to single genes adequately.

Therefore, therapeutic intervention not yet succeeded among so many diseases. Now, miRNA in IHD more less is currently known about miRNAs in cerebral ischemia. Limited knowledge about miRNAs on cerebral ischemia having both changes in expression and identification having potential targets and one of the best biomarkers among cardiac diseases. As of miRNA function in the heart addressed by conditionally inhibiting miRNA maturation in the murine heart revealed that miRNAs play a major role during the development as well. In addition, miRNA expression profiling demonstrated that expression levels of specific miRNAs also responsible in diseased with human hearts. It is directly attached to their involvement in cardiomyopathies. Specifically, miRNAs capable to identify so many distinct roles in both during heart development and pathological conditions. In fact responsible for cardiogenesis, hypertrophic growth response and cardiac conductance as well. In addition, miRNA's in animal models have also been linked to cholesterol metabolism, regulation, and cardiac diseases. Newly emerged miRNA-712 is a potential biomarker and one of the best predictors for athrosclerosis as a mechanosensors of epithelial cells. It responds to the sheer force of disturbed flow (d-flow) easily. Hence, many number of pro-atherogenic genes including matrix metalloproteinase are also upregulated by d-flow. d-flow mediating pro-inflammatory response and pro-angiogenic signals. In ligated carotid arteries of human is mimicking through the effects of d-flow. Accordingly, 24 h, preexisting immature miR-712 formed mature miR-712 suggested that miR-712 is flow-sensitive. In addition, miR-712 is also upregulated in endothelial cells exposed to naturally occurring d-flow in the greater curvature of the aortic arch is quite observable. Pre-mRNA sequence of miR-712 is generated from the murine model of ribosomal RN45s gene helps internal transcribed spacer region 2 (ITS2). Again XRN1 is an exonuclease that degrades the ITS2 region during processing of RN45s reduction. As of XRN1 under d-flow conditions, and leading on the accumulation process. Hence, miR-712 is add an evidence with TIMP3 decreases the expression of tumor necrosis factor-α (TNF-α) a pro-inflammatory regulator during the turbulent flow. As we know TNF-α in turbulent flow measured by the expression of the TNF-α-converting enzyme (TACE) through blood. TNF-α decreased if miR-712 was inhibited or TIMP3 overexpressed. This suggested that miR-712 and TIMP3 regulate TACE activity in turbulent flow conditions and working as an Anti-miR-712 inhibits vascular hyperpermeability. It significantly reduces atherosclerosis lesion development and immune cell infiltration.

Human Homolog MicroRNA-205

The human homolog of miR-712 was found on the RN45s homolog gene, which maintains similar miRNAs to mice. MiR-205 of humans share similar sequences with miR-712 of mice and is conserved across most vertebrates. MiR-205 and miR-712 also share >60% of the cell signaling targets, including TIMP3. miRNAs were differentially expressed between the two groups (upregulated and downregulated in the hierarchical clustering analysis [HCA] group). Other biomolecules, miRNAs were (miR-122, miR-221-5p, miR-31, miR-421-5p, miR-4333, miR-499-3p, miR-542, and let-7d-3p) were significantly dysregulated. Four higher and four lower expression of miR-122 was significantly (5.37-fold) increased. Simultaneously, the HCA group and SO group, indicating that a key role in the HCA-induced mucosal injury. Exposure to HCA caused by intestinal miRNA although deregulations and barrier dysfunction in swine altered the miRNAs related to the protection or destruction of the intestinal barrier may also one causal factor of cardiac complicacy and death.
Now consequences and new possibility in our hand to resolve these diseases through this potential biomarkers. We may save so many lives in our country like India. Moreover, we may also able give new beginning to our future generation through laboratory condition. We also able to develop molecular targeting therapy through the (miRNAs). At present, cardiac anesthetic agents associated with miRNAs profile provides an idea for decision-making for early detection, prediction, early treatment, PAC test, pre, post, follow-up, and management for cardiac surgery. miRNAs help for targeting to develop the molecular medicine as a new possibility in future and innovative consequences knocking on our door. It gives us a special challenge for researchers and clinicians to face up unlike worked out the sum and remembering us the reality.

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Conflicts of interest
There are no conflicts of interest.

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