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# Conference Handbook

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**Methods:** 10 patients (mean age: 57 ± 11) provided consents to undergo MRI at 3 to 5 days, 2 weeks, 24 weeks and 1 year after stroke event. DTI scan was performed using MRI 1.5T (Signa HDx; GE, USA) using TR/TE = 17000/101.1 ms and b-value = 1000 s/mm$^2$. Data analysis was carried out using Osirix software (v5.8.1). ROI was drawn to acquire the value of FA and MD in the infarcted regions and corresponding contralateral regions.

**Results:** A total of 160 areas measured. FA values of infarcted regions and corresponding contralateral areas were 0.2556 ± 0.15 vs. 0.3876 ± 0.15 (p = 0.33) on 3 to 5 days post stroke, 0.1996 ± 0.9 vs. 0.4203 ± 0.12 (p = 0.001) 2 weeks post stroke, 0.2292 ± 0.15 vs. 0.4147 ± 0.13 (p = 0.014) 24 weeks post stroke and 0.2648 ± 0.20 vs. 0.3920 ± 0.14 (p = 0.078) after one year of stroke. MD values were 60.72 ± 23.65 vs. 93.14 ± 24.4 (× $10^{-5}$ mm$^2$/s) (p = 0.001), 99.09 ± 37.99 vs. 81.46 ± 8.34 (× $10^{-5}$ mm$^2$/s) (p = 0.178), 137.85 ± 46.34 vs. 90.26 ± 18.33 (× $10^{-5}$ mm$^2$/s) (p = 0.17) and 134.48 ± 41 vs. 89.82 ± 13 (× $10^{-5}$ mm$^2$/s) (p = 0.055) respectively.

**Discussion:** There was significant difference in FA value in normal tissue compared to infarcted area at 3 to 5 days, 2 weeks and 24 weeks post stroke. Furthermore, there is also significant different in MD value at 3 to 5 days and 12 weeks post stroke.

**Conclusion:** Measurement of FA and MD may provide additional information about cellular changes in stroke over time.

## Characterization of Vascular Injury and Endothelial Dysfunction in microRNA-34a Knockout Mice with Alcoholic Steatohepatitis

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**Background and Rationale:** Endothelial dysfunction in liver sinusoidal endothelial cells (LSECs) decreases the production of vasodilators, such as nitric oxide (NO) and favors vasoconstriction and vascular injury. We aimed to characterize microRNA regulated sinusoidal endothelial dysfunction (SED) and vascular injury in the mice model of alcoholic liver injury.

**Methods:** Alcoholic liver injury (ALI) was induced in mice by intragastric overfeeding of alcohol (~27 g/kg/day) for eight weeks. The in vivo SED effects were also evaluated in toll like receptor 4 (TLR-4) knockout mice or the morpholino antisense oligomer against miR-34a (miR-34a Morpho/AS) treated mice with ALI.

**Results:** Using intragastric ethanol feeding mouse model of alcoholic liver injury, results showed the significant increase in serum ALT and hepatic miR-34a, severe steatosis, pericellular fibrosis, and intensified nitrosative stress induced by a 32-fold induction of nitric oxide synthase (NOS2). SED markers ICAM-1, VEGFR-2, and E-selectin were significantly up-regulated in the progressive phases of ALI. Lack of miR-34a in vivo, reversed the serum ALT level, and restored the levels of Sirt1 coupled with decreased NOS2 mRNA expression as well as SED dysfunction markers. Depletion of miR-34a in vivo also decreased overall vessel formation. Interestingly, in mice lacking TLR-4 with ALI, significantly reduced miR-34a levels, increased Sirt1 repression and decreased NOS2 mRNA expression were observed. In isolated sinusoidal endothelial cells by laser capture microdissection (LCM) from ALI mice, enhanced expression of miR-34a was observed and SED was discovered. Finally miR-34a morpholino treatment in ALI mice also showed reduced NOS2 mRNA level and reversed SED.

**Conclusion:** The discovery that the activation of miR-34a plays a significant role in the process of alcoholic liver injury through sinusoidal endothelial dysfunction for an exciting field in which the epigenomic microRNAs of hepatic endothelial cells may be manipulated with potential therapeutic benefits.

## Incidence and Time Trends in Prevalence of Stroke and Vascular Risk Factors in an Urban Sri Lankan Population: A Population – Based Cohort Study

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**Background and Rationale:** Prevalence of stroke is increasing in developing countries. However, population data on time trends in prevalence of stroke in Sri Lanka not.

**Methods:** We screened a population-based cohort 35–64 year selected by stratified random sampling from an urban health administrative area in 2007 evaluated them again in 2014. Identified possible stroke patients were independently reviewed by a Neurologist and a Physician with regard to diagnosis and vascular risk factors. Incidence and time trends in prevalence of stroke and vascular risk factors in 50–65-year age group were compared between 2007 and 2014.

**Results:** Of 2985 baseline study population in 2007 (females 54.5%, mean age 52.4 ± 7.8 years), 2204 attended follow-up in 2014. Of them, 45 have had a stroke/TIA, (female 51.1%, mean age 52.9 ± 5.4 years). 25 (55.6%) of them were strokes within the 7 year follow up (annual incidence-1.66 per 1000 population). Prevalence of stroke in 50–65-year age group was of the 45 stroke patients reviewed in 2014, 28 (62.2%) had. Of 27 were definite strokes, 15 probable strokes and 3 TIAs.

**Conclusion:** revalence has increased over time in urban Sri Lanka lie between developed and developing counties.