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Prevalence of rheumatic heart disease among primary school students in mid-eastern Ethiopia

Hailu Abera Mulatu
Addis Ababa University, Ethiopia

Objective: To determine the prevalence of rheumatic heart disease (RHD) among primary school students in Shashemene town of Oromia regional state of Ethiopia.

Design: A cross sectional study

Setting: The study was carried out among selected primary pupils in three public and six private schools in Shashemene town, in mid-eastern Ethiopia.

Subjects: Using a multistage sampling technique, pupils were selected from public and private primary schools in Shashemene town. They were clinically screened for evidence of RHD by auscultating for significant murmurs. The students with significant murmurs then had echocardiographic evaluation to confirm the presence of RHD.

Main outcomes: Students with significant murmurs and students with echocardiographically confirmed RHD.

Results: Of the 1764 students recruited, 900 (51.02%) were females while 864 (48.98%) were males. The mean age of the students was 8.86±2.14 years. 1065(60.37%) and 699(39.63%) respectively were recruited from public and private schools. Of the 1764 students, six (0.34 %) had significant murmur. Only one of the six had RHD, giving a prevalence of 0.57/1000 students. The student with RHD was a male, from public school and in the low socioeconomic class.

Conclusion: The prevalence of RHD in this study is low compared to similar studies conducted previously in the country. The true prevalence may be underestimated since higher prevalence is obtained from echocardiographic based screening compared to clinical screening.

dhailu2001@gmail.com

MITOL blocks mitochondrial dysfunction and cardiac cell death

Peifeng Li, Jing Wang, Lynn Htet Htet Aung and **Jinling Li**
University of Illinois at Chicago, USA

Myocardial infarction (MI) is one of the leading causes of death in the United States. Several studies have shown that apoptosis is the major process of cardiomyoctye loss during MI. Cardiomyocytes are enriched with mitochondria. The balance between mitochondrial fission and fusion regulates the mitochondrial morphology and function. Excessive mitochondrial fission triggers the cells to undergo apoptosis. Therefore, controlling mitochondrial fission could reduce the cardiac cell loss during MI. Recently, the mitochondrial ubiquitin ligase MITOL, localized in the mitochondrial outer membrane, was reported to play an important role in the regulation of mitochondrial dynamic and apoptosis. However, its underlying mechanism remains uncertain. To understand the involvement of MITOL in cardiac cell apoptosis, HL-1 cardiomyocytes were treated with H2O2 or doxorubicin and MITOL expression was evaluated with immunoblotting. We found that MITOL was up-regulated and the cell underwent apoptosis under treatment. We noted that over-expression of MITOL decreased the percentage of cells with mitochondrial fission and apoptosis (analyzed by TUNEL staining) by reducing the Drp1 accumulation in mitochondria and caspase-3 and caspase-9 activation. Conversely, upon knockdown of MITOL, a higher percentage of cells underwent mitochondrial fission and apoptosis. These findings suggest that MITOL plays a protective role against cardiac apoptosis, and may serve as a potential therapeutic target in apoptosis-related cardiac diseases.

peifli@uic.edu