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Kawasaki disease and the subsequent risk of ADHD: A nationwide population-based cohort study

Ho-Chang Kuo[1], Wei-Chiao Chang[2], Chun-Yi Lin[1] and Wei-Pin Chang[3]
[1]Chang Gung University, Taiwan
[2]Taipei Medical University, Taiwan
[3]Yuanpei University, Taiwan

Objective: The association between Kawasaki Disease and Attention Deficit Hyperactivity Disorder (ADHD) has rarely been studied in Asian populations. We investigated the hypothesis that Kawasaki Disease may increase the risk of ADHD in Taiwan using a nationwide Taiwanese population-based claims database.

Materials & Methods: Our study cohort consisted of patients who received a diagnosis of Kawasaki Disease in 1997~2005 (N=781). For a comparison cohort, five age- and gender-matched control patients for every patient in the study cohort were selected using random sampling (N=3905). All subjects were tracked for 5 years from the date of cohort entry to identify whether or not they had developed ADHD. Cox proportional hazard regressions were performed to evaluate 5-year ADHD-free survival rates.

Results: The main finding of this study was that patients with Kawasaki Disease seem to be at an increased risk of developing ADHD. Of the total patients, 83 patients developed ADHD during the 5-year follow-up period, among whom 21 were Kawasaki Disease patients and 62 were in the comparison cohort. The adjusted hazard ratios (AHR) of ADHD in patients with Kawasaki Disease was higher (AHR: 1.69; 95% confidence interval: 1.03-2.77; P<.05) than that of the controls during the 5-year follow-up. Our study also investigated whether Kawasaki Disease is gender-dependent risk factor for ADHD. We found that male patient with Kawasaki Disease have increased risk of developing ADHD (AHRs: 1.68 95% CI=1.01-2.83; P< .05).

Conclusion: The findings of our population-based study suggest that patients with Kawasaki Disease may have an increased risk of ADHD. These health associations should be taken into consideration, and effective psychological treatment plans should be designed for Kawasaki Disease patients.

erickuo48@yahoo.com.tw

The role of microglia activation in the development of sepsis-induced long-term cognitive impairment

Joao Luciano de Quevedo
The University of Texas Health Science Center at Houston, USA

Oxidative stress and inflammation is likely to be a major step in the development of sepsis-associated encephalopathy (SAE) and long-term cognitive impairment. To date, it is not known whether brain inflammation and oxidative damage are a direct consequence of systemic inflammation or whether these events are driven by brain resident cells, such as microglia. Therefore, the aim of this study is to evaluate the effect of minocycline on behavioral and neuroinflammatory parameters in rats submitted to sepsis. Male Wistar rats were subjected to sepsis by cecal ligation and puncture (CLP). The animals were divided into sham-operated (Sham+control), sham-operated plus minocycline (sham+MIN), CLP (CLP+control) and CLP plus minocycline (CLP+MIN) (100 µg/kg, administered as a single intracerebroventricular (ICV) injection). Some animals were killed 24 h after surgery to assess the breakdown of the blood brain barrier, cytokine levels, oxidative damage to lipids (TBARS) and proteins in the hippocampus. Some animals were allowed to recover for 10 days when step-down inhibitory avoidance and open-field tasks were performed. Treatment with minocycline prevented an increase in markers of oxidative damage and inflammation in the hippocampus after sepsis. This was associated with an improvement in long-term cognitive performance. In conclusion, we demonstrated that the inhibition of the microglia by an ICV injection of minocycline was able to decrease acute brain oxidative damage and inflammation as well as long-term cognitive impairment in sepsis survivors.

Joao.L.DeQuevedo@uth.tmc.edu