A 023

Nitric Oxide – a mediator of clinical disease in P. vivax infections

Nitric Oxide (NO) is thought to play an important role both in protection and pathology of malarial disease. The present studies investigate the possible role of NO in acute *P. vivax* infections.

The two study groups were *P.vivax* infected patients (n=17) from malarial non-endemic area (Colombo) and patients from endemic area (Kataragama) (n=9). Age and sex matched clinically healthy individuals (n=9) from each area who were blood film negative served as control groups. Blood was obtained before/during/after a paroxysm and also at random timing during the acute infection. Peripheral blood mononuclear cells (PBMC) were purified using sodium metrizoate on a density gradient basis. These cells were cryo-preserved till the measurement of NO production which was carried out based on the conversion of radio labeled arginine to citrulline by inducible isoform of NO synthase (NOS2) enzyme present in PBMC.

Malaria patients infected with *P. vivax* tended to have high NOS2 activity (147.75 $\pm$38.55 pmol / mg) when compared to healthy controls (136.45 $\pm$ 15.4 pmol /mg); ($p>0.05$). NOS2 activity in non-immune malarial patients a were significantly higher (mean = 174.9 $\pm$ 56.8 pmol /mg) when compared to the levels in endemic patients (mean = 120.6 $\pm$ 25 pmol /mg); ($p<0.05$). Most pronounced NOS2 activity was observed during paroxysms (mean = 251.5 $\pm$ 32.8 pmol /mg) of P. vivax infections in clinically non immune patients when compared with samples collected at random timing during acute stage of infection (164 $\pm$ 21.5); ($p<0.05$).

In conclusion, NO production is associated with clinical disease in *P. vivax* infections. It is likely that cytokines released following schizont rupture trigger the production of NO, the levels of which coincide with clinical paroxysms.