

INTRODUCTION

Varicella zoster virus (VZV), a member of the herpes virus family causes varicella (chickenpox) through primary infection and herpes zoster (shingles) through reactivation of latent virus in the dorsal root ganglia after primary infection. Varicella zoster virus is highly contagious worldwide and is spread by droplet or air born transmission (*Whitley - 2000*).

Varicella, a benign exanthematous disease common in children, also occurs in nonimmune adults, neonates or immunocompromised hosts with the risk of serious morbidity and mortality including secondary complications such as pneumonia and encephalitis (*Preblud, 1981*).

The epidemiology differs with the climate, e.g. in temperate countries, most cases occur in preschool and school aged children (*Fairley, et al 1996*). Therefore most adults are immune due to previous exposure in childhood (*Chant, et al 1998*). But in tropical countries, most adults and adolescents are seronegative, so they are susceptible with the risk of greater morbidity (*Flisser, et al 1997*).

A safe and efficacious live attenuated varicella vaccine has been recently licensed in many countries, but vaccination policy is still a matter of debate (*Krause et al 1995*).

Food and Drug Administration (FDA) approved the use of live attenuated varicella vaccine in March 1995 (*Ventura 1997*),

Also, the Center for Disease Control and prevention (CDC) established guidelines for the immunisation of susceptible individuals with live attenuated varicella vaccine in 1996. (*Ventura 1997*).

The vaccine is expensive, So was suggested by Ventura (1997) that restriction of vaccination to only susceptible individuals would be cost effective.

The study of varicella zoster virus antibodies in unvaccinated adolescents can be used as a guide when implementing a strategy for vaccination against VZV (Ulrich *et al.*, 2001).