European Journal of Molecular & Clinical Medicine

ISSN 2515-8260 Volume 02, Issue 04, 2014

Simian varicella virus is present in skin tissue of rhesus macaques after experimental reactivation,,

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Abstract

Varicella zoster virus (VZV) causes varicella (chickenpox), establishes latency in ganglia and reactivates decades later to produce zoster in the elderly. Clinical, pathological, immunological and virological features of simian varicella virus (SVV) infection of primates parallel human VZV infection. Primary SVV infection of primates, cause varicella, after which virus becomes latent in ganglionic neurons and reactivates upon social and environmental stress. Five rhesus macaques were infected intrabronchially with 4.0x105 pfu of SVV. Two weeks later, the monkeys developed varicella rash. Twenty months later four of the monkeys were treated once with a 50 mg/kg of anti-CD4 antibody. All 5 monkeys developed zoster rash, 7- 55 days after the treatment. Punch biopsies of the skin rash were analyzed for the presence of SVV antigens by immunohistochemistry and immunofluorescence. SVV ORF 63 protein and glycoproteins gH and L were detected in sweat glands in skin from all 5 monkeys.

Keywords:

Presence of SVV in the β; 3; tubilin ;positive nerve endings in affected skin suggested possible route of skin infection during zoster