**Virus (VZV)-specific Cell-mediated Immunity in Facial Palsy:**

**INTRODUCTION**

Both Hunt syndrome and Bell’s palsy show facial palsy. In 1907, James Ramsay Hunt analyzed 60 cases with facial palsy, and proposed zoster oticus, which is now known as Ramsay Hunt syndrome or simply Hunt syndrome. It is now well-recognized that the etiology of Hunt syndrome involves reactivation of varicella-zoster virus (VZV) in the geniculate ganglion.

Bell’s palsy was considered to be idiopathic in the past. However, recent studies indicate that herpes simplex virus type 1 (HSV) plays an important role in Bell’s palsy.

**Methods**

This prospective study was conducted at our tertiary referral hospital between 2010 and 2012. Six Hunt syndrome and 28 Bell’s palsy patients were enrolled. Mononuclear cells were isolated from whole blood and incubated with VZV antigen in culture plates for 40 h. Anti IFN-γ antibody was added and the ELISPOT system counted immunostimulated spots indicating VZV-specific CMI. Numbers of spots and the relationship between the spots and days from the onset of palsy were compared.

**RESULTS**

No difference in the number of spots was observed between Hunt syndrome and Bell’s palsy.

In Hunt syndrome, there was a strong positive relationship between numbers of spots and days from the onset of palsy (r = 0.87). In the early days, spot numbers in Hunt syndrome were much lower, indicating low VZV-specific CMI. In contrast, Bell’s palsy showed no such relationship (r = -0.24).

**Conclusions**

This study indicates that low CMI to VZV may play a role in VZV reactivation in the facial nerve. VZV vaccination may promote VZV-specific CMI, leading to prevention of Hunt syndrome.