Rapidly increasing incidence of Kaposi’s varicelliform eruption in patients with atopic dermatitis

Sir,

Kaposi’s varicelliform eruption (KVE) is occasionally observed in patients with atopic dermatitis (AD). However, most of them are sporadic cases and no detailed analysis of the monthly incidence of KVE has been reported with accurate diagnosis by PCR. We analyzed the incidence of KVE in our hospital since the year 2006.

All of the patients had typical features of AD. KVE was diagnosed clinically based on the appearance of disseminated vesicles, pustules or erosions on face and various other sites of the body. Skin swabs taken from the lesion according to a previous report, demonstrated the presence of HSV-1 or HSV-2 DNA by PCR. Patients in whom HSV-1 or HSV-2 DNA was not detected by PCR, were excluded. As shown in Table 1, monthly incidence of KVE was less than 20 cases in June 2006 or July 2006. Similar numbers were seen in previous years in our hospital. However, the incidence began to increase in all ages since August 2006 and it reached 138 cases in April 2007. The youngest patient was a 2.5 month-old baby. HSV-1 was detected in most of the cases while HSV-2 was detected in only a few cases.

Children (<16 years of age) were treated with oral acyclovir (20 mg/kg) four times a day and vidarabine ointment for 7-14 days. On the other hand, adults (>17 years of age) were treated with valacyclovir 1000 mg three times a day and vidarabine ointment for 7-10 days, except for 20 patients who were admitted to hospital and treated with intravenous acyclovir 250 mg three times a day for 7-10 days. Antibiotics were administered when patients had secondary bacterial infection. After treatment, skin lesions healed completely in all of the patients.

The reasons for the rapidly increasing incidence of KVE remain to be elucidated. It was reported that a mini-outbreak of KVE occurred in a skin ward. However, the outbreak of herpes infection in a local area was an unlikely explanation as patients came to our hospital from various cities in Japan. Moreover, the incidence of herpes infection in subjects without AD was not found to be increased in our hospital. It was reported that risk factors for KVE were the reduced production of some cytokines (IFN-β or CXCL 10/IP-10) or elevated serum total IgE levels. In fact, serum total IgE levels in KVE patients younger than a year of age (338 ± 22 IU/ml, n = 48) were significantly higher than those in age-matched AD patients without KVE (157 ± 8 IU/ml, n = 48). Large scale analysis will be necessary to survey the worldwide incidence of KVE.

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Table 1: Incidence of Kaposi’s varicelliform eruption in patients with atopic dermatitis

| Year   | 2006 | 2006 | 2006 | 2006 | 2006 | 2006 | 2006 | 2007 | 2007 | 2007 |
|--------|------|------|------|------|------|------|------|------|------|------|
| Total patients | June | July | Aug. | Sept. | Oct. | Nov. | Dec. | Jan. | Feb. | March | April |
| Sex (F/M) | 10/8 | 9/10 | 10/15 | 15/14 | 17/16 | 25/24 | 26/30 | 31/37 | 58/63 | 61/63 | 67/71 |
| Age | 0-1 year | 0 | 1 | 2 | 3 | 3 | 2 | 4 | 4 | 8 | 11 | 11 |
| | 1-16 years | 8 | 9 | 11 | 12 | 14 | 21 | 22 | 25 | 32 | 34 | 38 |
| | <17 years | 10 | 9 | 12 | 14 | 16 | 26 | 30 | 39 | 68 | 79 | 89 |
| | HSV-1/HSV-2* | 16/2 | 17/2 | 20/5 | 23/6 | 26/7 | 40/9 | 45/11 | 57/11 | 88/20 | 103/21 | 115/23 |
| | Fever | 14 | 15 | 18 | 20 | 26 | 35 | 45 | 54 | 72 | 81 | 101 |

Values are numbers of patients; *Number of patients in whom HSV-1 DNA or HSV-2 DNA was detected by PCR.
Isolated facial palsy in varicella

Sir,

Isolated facial palsy as a neurological complication of varicella is reported.[1-5] As such neurological complications caused by varicella are estimated to occur in approximately 0.01-0.03% of infections. Frequent neurologic complications of varicella are cerebellar ataxia and encephalitis while the rare complications are transverse myelitis, aseptic meningitis, Guillain-Barré syndrome, meningo-encephalitis, ventriculitis, optic neuritis, delayed contralateral hemiparesis, peripheral motor neuropathy, cerebral angiitis, Reye’s syndrome, and facial paralysis.[1]

Ten days after varicella (chicken pox), a 26-year-old house wife presented with a left facial palsy without any neurological symptoms. While she was recovering from varicella, she developed inability to close her left eye and deviation of mouth to the right side. There was no clinical history of retroauricular pain, hyperacusis, decreased production of tears, and altered taste. On examination, there were multiple discrete crusted lesions all over the trunk, back, extremities, and healed eroded lesions over the face. Complete neurological examination revealed lower motor neuron type of facial palsy without any other abnormal features [Figure 1]. Other systemic examination revealed no abnormalities. Vital signs are normal. Patient is not hypertensive. Routine investigations were within normal limits. Serological tests for syphilis and human immunodeficiency virus (HIV) infection are nonreactive. Varicella zoster virus IgG (13.2 NTU) and VZV IgM (13.7 NTU) were positive. Computed tomography (CT) scan of brain revealed no abnormality. The patient was treated with acyclovir 800 mg five times/day for 7 days and prednisolone 10 mg three times/day in tapering doses for 3 weeks. The patient was examined after 3 weeks and complete recovery of facial palsy was noticed.

Facial paralysis in varicella can be seen either prior to or after the appearance of exanthem. It may occur 5 days before exanthema appears or during 16-day period after varicella is diagnosed. Facial palsy as a complication of varicella can be a result of preeruptive hematogenous or neurogenous spread of varicella zoster virus[1,3,4] Bilateral facial palsy can also occur rarely during varicella. [3] Though in our case facial nerve palsy developed during varicella infection with elevated V-Z IgG and IgM antibodies, it should be differentiated from closely identical conditions of facial palsy like Bell’s palsy and Ramsay Hunt syndrome.

Table 1: Differential diagnosis of our case

| Cause                  | Distinguishing features                                                                 |
|------------------------|-----------------------------------------------------------------------------------------|
| Classical Bell’s palsy | HSV Type 1, history of exposure to cold, no exanthematous rash, no constitutional symptoms, sudden onset, prolonged course |
| Ramsay Hunt syndrome   | Varicella zosteriform virus, pronounced prodrome of pain, hyperacusis, vesicular eruption over the external pinna and in ear canal or pharynx |
| Our case               | Varicella zosteriform virus, history of varicella, active and healed lesions over the body, elevated VZV antibodies (IgG and IgM) |

Figure 1: Healing chicken pox lesions over the face with facial palsy