Zoster in Childhood after Inapparent Varicella

Sir,

Zoster infection is rare in healthy children and if it occurs it is usually associated with a history of chickenpox. We report here a case of a 7-month-old, otherwise healthy boy who displayed typical skin lesions of zoster in trigeminus I. At no time was chickenpox clinically apparent, although exposure to the varicella zoster virus at the age of 3.5 months had been noted.

Varicella occurs before the age of 14 years in 95% of all children (1). Zoster usually occurs in patients over 50 years old and is rare in childhood. Risk factors for zoster in infants include immunocompromised states and/or primary varicella zoster virus (VZV) infection *in utero* or before 12 months of age (2). Zoster can also occur when children are infected with chickenpox after 1 year of age (3).

CASE REPORT

A 7-month-old boy in good general condition, 70 cm tall and 7.5 kg in weight, was admitted to our paediatric dermatology ward because of the development of skin lesions 2 days previously. The first physical examination found an erythema on his left eyelids, forehead and upper part of his cheeks. A few hours later, grouped vesicles occurred on the erythematous base around the left eye, and 48 h later they took over the upper left half of the face and forehead and the erythema faded (Fig. 1). Enlarged lymph nodes in the neck and under the left mandibula were found. We diagnosed a VZV infection of the trigeminus I dermatoma. There was no abnormality of mucus and no conjunctivitis. The remaining examination was unremarkable. All routine blood parameters were within the normal range, including differential blood count.

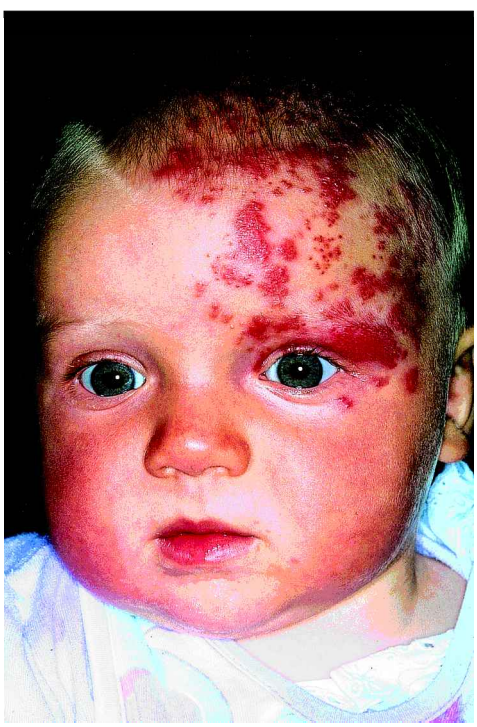

Fig. 1. A 7-month-old boy with typical skin lesions of zoster in trigeminus I.

The history revealed that, 3 months previously, the boy's 3 siblings had had chickenpox. At that time the baby was 3.5 months old. Although he had permanent contact with the other children in the family, no skin lesions were apparent. The serological investigations at the time of hospitalization were:

Mother: VZV-IgG positive, VZV-IgM negative;
Patient: VZV-IgG positive, VZV-IgM borderline, VZV-IgA positive, HIV I and II negative;

The boy received acyclovir (10 mg/kg per day) perorally 3 times a day for 5 days. Under this treatment complete resolution of the lesions occurred.

DISCUSSION

Zoster is generally accepted as the reactivation of a latent infection of VZV from the sensory ganglia (4). We supposed that our patient, at the age of 3.5 months, had had an inapparent varicella infection at the time when his siblings had been suffering from chickenpox. The reason for the non-appearance of infection was that the baby was, at that time, still protected by maternal antibodies. However, this provided only incomplete protection against reinfection, so that as soon as the mother's antibodies faded, the VZV was reactivated and, because partial protection existed, emerged not as chickenpox but as the clinical manifestation of zoster. The serological findings in the mother showed that she had previously been in contact with the varicella virus, probably when she had had chickenpox as a child.

The serological features of the baby show that there had already been contact with the virus, to produce the IgG antibodies; however, the borderline elevated IgM indicates that the immune system is still working to achieve complete immunization. The protecting maternal antibodies usually fade at the age of 6–7 months. Therefore, children usually build up their own immunization with the first varicella contact, which was, in this case, partially inhibited owing to the residual but incomplete protection by the maternal antibodies. If there had been no protecting maternal antibodies at first contact, one would expect the IgG to be positive and complete, and no IgM or IgA would be detectable in the plasma.

Up to now, there has been no consensus about the treatment of zoster in healthy children. The American Food and Drug Administration (FDA) approved high-dose oral acyclovir for the treatment of acute zoster without a minimum age limit because of minimal toxicity. However, Rothe et al. published a guideline for oral acyclovir therapy in herpes simplex virus and VZV infection in children and during pregnancy (5). If intravenous acyclovir therapy is recognized in immunocompromised children, a systematic therapy of zoster in immunocompetent patients does not seem to be indicated, except for zoster involving the first branch of the trigeminal nerve because of its ocular complications. New medications such as famcyclovir or valacyclovir have not yet been studied in children (6). In our patient, under oral acyclovir therapy, the skin lesions healed completely.

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Claudine Lagarde, Astrid E. Steen, Thomas Bieber and Kay H. Steen*
Klinik und Poliklinik für Dermatologie, University of Bonn, Sigmund-Freud-Str. 25, D-53105 Bonn, Germany
*Address for Correspondence

Subcutaneous Metastasis Following Percutaneous Ethanol Injection Therapy for Hepatocellular Carcinoma

Sir,

Percutaneous ethanol injection therapy (PEIT) was first reported by Sugiura et al. in 1983 (1) and has been widely used for the treatment of hepatocellular carcinomas (HCC) (2, 3). Recently, PEIT has also been performed in the treatment of other tumors (4–6). Histopathologic examinations of tumors after therapy have revealed that PEIT can completely destroy the tumor in most cases (7).

Minor complications of PEIT, such as pain, fever and transient alcohol intoxication, have been reported (2, 3). However, a few reports have described of subcutaneous or pleural needle track seedings of tumor cells after PEIT (8–10). Here we describe two cases of metastatic subcutaneous tumors along the needle track for PEIT.

CASE REPORTS

The characteristics of the two patients are summarized in Table I. Briefly, the first patient, a 72-year-old female, had undergone two sessions of PEIT for three HCCs measuring < 20 mm in diameter, but a subcutaneous nodule was noticed 5 months after performance of PEIT in the epigastric region (Fig. 1*a*). The tumor, which seemed to develop as a result of needle track seeding, was resected. The pathological findings of the tumor revealed a non-capsuled solid lesion in the lower dermis through fatty tissue, and the lesion consisted of mainly α-fetoprotein (AFP)-positive, moderately differentiated HCC by immunohistochemical examination (Figs. 1*b*, *c*).

The second patient, a 69-year-old male, had undergone 10 sessions of PEIT for one HCC measuring 18 × 20 mm in diameter. He noticed a subcutaneous nodule in the epigastric region 2 months after undergoing PEIT. The tumor was resected. The pathological findings revealed a non-capsuled solid lesion in the lower dermis and fatty tissue consisting of moderately differentiated HCC (not shown).

Table I. *Clinical features of two patients with subcutaneous nodules after performing percutaneous ethanol injection therapy (PEIT)*

Age/ gender	HCV	HBs	Child	AFP	PEIT	Interval (months)
72/F	+	–	A	29	Twice	5
69/M	+	–	A	50	10 times	2

HCV = hepatitis C virus; HBs = hepatitis B virus; Child = the stage of liver cirrhosis (A = good, B = moderate, C = poor); AFP: α-fetoprotein.

DISCUSSION

The first case of needle track seeding following PEIT for the treatment of HCC was reported by Saito et al. (11) in 1989, in Japan. Since then, there have only been 32 cases, including ours, reported in the literature. In Japan, where PEIT was initially started, 17 cases have been reported. Of note, 28 cases (87.5%) were reported in the past 5 years (1995–1999) after PEIT became a more common procedure.

The clinical features of HCC seeding have been described as solid tumors in the abdominal wall (8, 10), pleural lesion (9), peritoneal lesion (12), intercostal muscle (13), or subcutaneous tissue (10, 11, 13). The incidence of HCC seeding in the subcutaneous tissue after a fine-needle biopsy is below 0.005% (14). Di Stasi et al. (15) reported that tumoral seeding occurred following PEIT in 0.66% of the patients. Saito et al. (11) described the tumor as a black or brown subcutaneous mass protruding from the liver. The interval for the emergence of the tumor following PEIT ranges from 2 months to 4 years (mean 16 months). Our patients developed brown-colored subcutaneous nodules in the epigastric region. The mechanism of needle track seeding is considered to be complex, and several causes have been discussed. The diameter and type of needle, the number of needle passes, extent of tumor aggressiveness, and the immune status of the patient are all considered (10). However, according to the literature, patients developing seeding show no significant clinical differences from the other non-seeding patients. Many reports emphasize the correct therapeutic application and good technique of PEIT. Similarly, in our two patients, PEIT was carried out correctly by doctors experienced in PEIT. Therefore, needle track seeding can occur indiscriminately and accidentally, although the probability seems small.

The prognosis for needle track seeding is usually not bad, which is the same as that for the other non-seeding patients. The systemic condition of the patient is most important. The