Four Cases of Vesicular Lesions in Adults Caused by Enterovirus Infections

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Echovirus types 4 and 33 and coxsackievirus type B1 were recovered from vesicular lesions in four adults. Patient 1 had cutaneous localized vesicles, patient 2 had a recurrent cutaneous vesicle, and patients 3 and 4 had mucosal vesicles. Three of the patients were suspected of having herpesvirus lesions. One of the patients was a human immunodeficiency virus type 1-seropositive man, and the enterovirus infection was the first clinical manifestation. Our results underline the importance of virological diagnosis before treatment with acyclovir, especially for immunocompromised patients.

Various skin rashes have been reported in acute infections caused by enteroviruses (2). The rashes are usually transient and associated with fever and other symptoms. They can mimic rubella, and diagnostic differentiation is necessary for pregnant women (10).

However, lesions are rarely vesicular, except in hand, foot, and mouth disease, which is characterized by vesicular rash on the extremities, caused by coxsackievirus type A; and in herpangina (6, 10).

We report four unusual cases of vesicular lesions in adults with isolation of echovirus type 4, echovirus type 33 (two cases), and coxsackievirus type B1 isolated on cell culture from vesicle fluid.

Case reports. Case 1. In July 1983, a 25-year-old male on a camping holiday had acute diarrhea with fever and a vesicular rash on the left thumb. Vesicle fluid was inoculated on human fibroblasts, and coxsackievirus type B1 was isolated and identified by a seroneutralization test.

Case 2. In July 1987, a 38-year-old male was admitted for a herpeslike vesicle on the cheek bones. Five years previously, the patient had similar lesions on the cheeks and forehead suggestive of herpetic primary infection, but no virological investigation was performed and no specific treatment was given. The lesions persisted for 2 years and thereafter resolved spontaneously. After this apparent cure, the vesicular lesions reappeared, associated with fever. Echovirus type 33 was isolated from vesicle fluid. After a month, the infection disappeared without treatment.

Case 3. In May 1987, a 29-year-old female presented with vesicles on rectal prolapse after sexual relations, without fever or diarrhea. Echovirus type 4 was identified in vesicular fluid.

Case 4. In May 1987, a 40-year-old male homosexual seropositive for human immunodeficiency virus type 1 was admitted to the infectious diseases ward with a herpeslike stomatopharyngitis with onset 1 week before hospitalization. Clinical examination showed fever (39°C) and angina but no diarrhea. Treatment with acyclovir made no improvement. On histological examination, buccal mucosa showed aphthoid lesions with superficial ulcerations covered by a pyogenic membrane. Virological samples of vesicular lesions were inoculated before treatment on human fibroblasts (MRC5) to isolate herpesvirus, but, surprisingly, echovirus type 33 was identified instead of the expected herpesvirus.

In these four cases, the viruses were identified on human fibroblasts (MRC5; Bio-Mérieux, Marcy l'Etoile, France). Type determination was performed by a seroneutralization test with Melnick pool sera and was confirmed by the National Health Laboratory (M. Aymard; Medicine Faculty, Lyon, France).

Discussion. In these four cases of vesicular lesions in adults caused by enteroviruses, all the vesicles mimicked herpesvirus infections. To avoid inappropriate treatment with acyclovir, a laboratory diagnosis should be made, especially between May and October, when the incidence of enterovirus infections is the highest. Case 1 occurred at the beginning of an outbreak of coxsackievirus type B1. We reported previously (9) that in 1983 there were two outbreaks of coxsackievirus type B1 and echovirus type 33. In 1987, the incidence of enterovirus infections exhibited a definite peak in July (data not shown).

In one of our patients (case 4), treatment with acyclovir began immediately after virological sampling had no effect. The case of this seropositive patient is particularly interesting because he had no symptoms of acquired immunodeficiency syndrome-related complex or acquired immunodeficiency syndrome. The results of laboratory examinations showed only decreased lymphocytes (CD4+). The vesicular lesions were the first clinical manifestations of the disease.

In case 2, it is possible that chronic infection was caused by an enterovirus. It is unfortunate that no virological sample of the vesicle was taken at the first eruption. Chronic infection has been shown in vitro with echovirus type 6 and in vivo in patients with postviral fatigue syndrome with the use of an enterovirus-group-specific monoclonal antibody directed against the VP1 polypeptide (11).

It has been observed that many types of enteroviruses can cause enanthems and exanthems but rarely vesicles. Diagnosis is easy when acute diarrhea, febrile illness, or upper respiratory tract infections are associated with vesicular lesions, especially in childhood, but is more difficult in adults. Enteroviruses have rarely been isolated from vesicle fluid. Enterovirus type 71 has been isolated from vesicular fluid in a typical hand, foot, and mouth disease (1). Deseda-Tous et al. reported two cases of echovirus type 11 recovered from vesicular lesions in adults (3). All these lesions

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mimicked herpesvirus lesions. In some cases, poxviruses can be incriminated (8).

Thus, virological laboratory examinations are very important in the diagnosis of this type of vesicular lesion, especially when the location of the lesions is uncommon or when they are found in immunocompromised patients (4, 5).

The inoculation on cell cultures of vesicle fluid is easy to perform, and the isolation of a virus in this particular site is conclusive proof of its responsibility for infection. In certain cases, electron microscopy can rapidly differentiate enteroviruses from herpesviruses and poxviruses.

The present wide use of acyclovir should not put an end to virological investigations. The increase in human immunodeficiency virus infections may be responsible for the occurrence of atypical mucocutaneous syndromes (7).

LITERATURE CITED