

RESULTS

The results are given in Table I, which also shows the steroid medication given to some of the patients prior to collection of the samples. PGE-like activity was recovered in the blister fluid from all the patients. Patients not yet treated with steroids showed higher PGE values than those treated. The highest value, 3000 pg/0.1 ml of fluid, occurred in a case of bullous pemphigoid not yet treated with steroids. The lowest value, 75 pg/0.1 ml of fluid, was encountered in another case with the same diagnosis treated with 70 mg of prednisolone daily for 2 weeks. In a case of cicatricial pemphigoid, the PGE content of the bullae dropped during treatment with prednisolone and azathioprine from 1500 to 156 pg/0.1 ml of fluid.

COMMENTS

The study demonstrates considerable PGE-like activity in the blister fluid from blistering dermatoses of apparently immunological origin as well as from burn blisters and pressure bullae due to intoxication. In a recent *in vitro* study, hydrocortisone and methylprednisolone were ineffective as inhibitors of PGE₂-synthesis in isolated human epidermal cells (2). In the present study, prednisolone treatment, 80 to 25 mg daily, seemed to have an inhibitory effect on the PG-synthesis, although measurable amounts of PGE were still present. The demonstration of PGs of the E group in the blister fluid may be more important than the demonstration of the F group, since the former is much more active than the latter when injected into human skin (8).

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Acute Tendovaginitis after Percutaneous Steroid Injection with Dermojet®

Hans Hammar

Department of Dermatology, Karolinska sjukhuset, Stockholm, Sweden

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The mode of intralesional administration of steroids has been facilitated by devices utilizing high pressure injection of a crystal suspension of the steroid aided by a jet stream (1, 2). Dermojet® and Port-O-Jet® are two types of such devices on the market. Complications connected with this treatment are few (1). The present case report describes an uncommon side-effect following the percutaneous injection of triamcinolone acetonide (Kenacort-T, Squibb, 5 mg/ml) into a plaque of lichenified atopic dermatitis on the dorsal aspect of the hand.

CASE HISTORY

A 34-year-old woman, a member of an atopic family, had had atopic dermatitis since infancy and allergic rhinitis and asthma since early childhood. In recent years her main complaint has been a disseminated atopic dermatitis. She also has lichenified plaques on the backs of the hands and on the buttocks.

During February 1976 the patient received an intensified local treatment with steroid ointments and systemic steroids (Celestona bifas, Schering Corp.). On March 17 the lichenoid plaques on the buttocks and on the back of her right hand were treated with Kenacort-T suspension (Squibb 5 mg/ml) with percutaneous injection utilizing Dermojet®. This device was used according to the directions of the manufacturer and the apparatus was in good technical condition.

The patient was given several shots in the buttock lesions without any immediate or delayed complaints. At the same session 6 shots were given to her right hand plaque. The lesion, 3×6 cm was localized in the middle of the dorsal aspect of the hand about 4 cm proximal to the 3rd and 4th metacarpophalangeal joints. She felt radiating pain up in the forearm and down into the fingers immediately after the shots and she refused to take shots on the contralateral hand where she had a similar plaque. The next day the back of the right hand was swollen and tender and she could not move the index, middle and ring fingers more than 30° in the metacarpophalangeal joints. The area was pale with a non-pitting edema but without crepitation on movement of the fingers. She reported radiating pain along the paths of the tendons passing the area. She had no adenitis and the temperature was normal. On suspicion of a phlegmonous infection in the swollen area she was treated with phenoxymethylpenicillin 1.6 g (Calciopen K, Leo) and cloxacillin 0.5 g (Ekvacillin, Astra) four times a day. In spite of the treatment, the symptoms became aggravated and the patient was admitted to hospital on March 23. The back of the right hand was swollen and pale, but without any erythema. On top of the edematous area there was a lichenified scaling plaque without erosions. The pale skin lesion did not differ in temperature from its surroundings. The patient was afebrile and without any swollen or tender regional lymph nodes.

Laboratory examinations. Routine blood and urine tests were normal, including differential white blood cell counts; the erythrocyte sedimentation rate was 33 mm/h. Antistreptolysin and antistaphylococcal titres were normal, 100 and 1.0 respectively. Bacterial cultures from the hand obtained

March 23 gave growth of *Staphylococcus aureus*. On March 29 an incision was made in the tendon sheath. The clear fluid obtained was sterile.

Subsequent course. The patient was treated with large doses of penicillin during the first 3 days after admission to the hospital: benzyl-penicillin 10 million units and cloxacillin 4 g per day i.v., subsequently followed by oral penicillins. Pain and edema decreased slowly during the first week. The finger flexibility was not improved. An incision was made and clear fluid drained from the tendon sheath. Finger movement improved immediately and on discharge from the hospital after 2 weeks there was only 1 cm diastasis between the palms and the flexured finger tips. There was no tactile pain at the lesion and the incisional ulcer was closed.

Comments. This patient was treated with antibiotics on the assumption that a phlegmonous infection had been introduced by the Dermojet® treatment. Although no cultures were taken from the instrument at the time of injection, it seems improbable, in retrospect, that any germs were introduced by an infected apparatus, since no reaction was noted at the other sites treated. It cannot be excluded that bacteria on the skin surface could have followed the jet stream into the skin. It was conspicuous, however, that pain was felt immediately and the objective signs did not include erythema, lymphangitis, adenitis or fever as signs of a bacterial infection. By exclusion, therefore, it is most probable that the crystals were injected within the tendon sheath giving rise to an aseptic inflammation. This type of reaction has rarely been observed by Dr J. Bleeker (personal communication) as a complication accompanying Port-O-Jet treatment. This communication is intended to point out the possible drawback of this injection procedure in areas of skin overlying tendons.

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