Phytophotodermatitis associated with parsnip picking

L Lutchman, V Inyang, D Hodgkinson

Abstract

Phytophotodermatitis to certain plant groups is a well recognised entity. The combination of sunlight exposure and contact with plants of the umbelliferae family leads to the development of painful, erythematous, and bullous lesions and later to cutaneous hyperpigmentation. Agricultural workers and many clinicians often fail to make this link when patients present with these lesions. An incident involving 11 patients is presented to highlight this problem.

(J Accid Emerg Med 1999;16:453-454)

Keywords: phytophotodermatitis; parsnip picking

Case report

In June 1998, 11 patients (seven male) presented to the accident and emergency

department on the same day complaining of a painful skin eruption affecting the forearm and hands. One patient also complained of similar lesions on both legs.

All 11 individuals had, for the week before presentation, been involved in hand picking parsnips at a local farm. Although there had been rain for the first few days, the sun came out towards the end of the week. Within 48 hours of the sun exposure, these patients began to develop a painful erythematous rash that eventually became bullous in appearance. The rash was initially attributed to possible chemical pesticide or fertiliser exposure.

Upon presentation, these patients showed a variable degree of skin involvement but one male patient had severe bullous eruptions of the fingers (see fig 1) that restricted finger movement and necessitated admission to hospital. He was treated with limb elevation and flomazine dressings. The other patients were treated with non-adherent dressings and followed up in the outpatient clinic.

Over the subsequent 48 hours all patients showed significant resolution of the lesions. The patient who had been admitted to hospital was discharged with a considerable reduction in hand swelling and improved range of movement.

Discussion

For more than 3000 years it has been recognised that combined exposure to sunlight and certain plants leads to an acute skin reaction and eventually to hyperpigmentation in some cases.¹ The effect was used for the treatment of vitiligo in ancient Egypt.

In 1942 Klaber coined the term phytophotodermatitis to describe the phenomenon. Further work has revealed that the implicated plants, which include parsley, celery and parsnips, contain photosensitising furocoumarins. Phytophotosensitivity to giant hogweed was recognised in 1970 as posing a public health hazard,² and there are other reports in the literature on the photosensitivity associated with parsnip³ and the potential occupational risk to farmers.

As a result of this case, the National Farmers Union was contacted and appropriate educational material was disseminated to agricultural employers and workers. Nevertheless, such exposure continues to occur and health care workers also need to be reminded of the presentation of phytophotodermatitis.

Treatment is symptomatic with non-adherent dressings and analgesia. In more severe cases with significant vesiculation and bullous eruption, popping the bullae under

Accident and
Emergency
Department, Ipswich
Hospital, Ipswich,
Suffolk IP4 5PD
L Lutchman
V Inyang
D Hodgkinson

Correspondence to:
Mr Lutchman.

Accepted 29 May 1999

Figure 1 Severe hand involvement in a parsnip picker. (The digital loss was pre-existing.)

solution of potassium permanganate (drying agent) then covering with a topical steroid cream, for example Betnovate RD (betamethasone) cream often brings about rapid resolution of the condition.

Patients should be advised that hyperpigmentation may persist for several months but is best left untreated. Affected skin may remain

photosensitive for several months and sunscreens may be needed.

Conflict of interest: none.

Funding: none.

- 1 Lovell C. *Plants and the skin*. Oxford: Blackwell, 1993.
- 2 Drever JC, Hunter J. Giant hogweed dermatitis. *Scott Med J* 1970;15:315-19.
- 3 Aberer W. Occupational dermatitis from organically grown parsnip. *Contact Dermatitis* 1992;26:62.

EMERGENCY CASEBOOKS

Shock and ipsilateral pulmonary oedema after tube thoracostomy for spontaneous pneumothorax

Anup V Desai, Paul R Phipps, David J Barnes

A 17 year old male smoker, previously well, was referred to hospital for management of a spontaneous right pneumothorax. After one week of right pleuritic chest pain he had developed mild dyspnoea and his chest film demonstrated a complete right pneumothorax with mediastinal shift (fig 1). Physical examination revealed normal vital signs other than tachypnoea of 20 breaths per minute, and signs consistent with the radiological findings. An intercostal chest tube was inserted and placed on low suction, with prompt resolution of the pneumothorax. However, over the next four hours clinical deterioration occurred,

Figure 1 Complete right pneumothorax on presentation.

with tachycardia, hypotension, respiratory distress, and hypoxaemia (oxygen saturation of 90% on 50% inspired oxygen). The patient was agitated, pale and clammy with inspiratory crackles noted in the right lung. The packed cell volume was raised at 0.63. A repeat chest film (fig 2) showed right mid and lower zone alveolar opacification. A diagnosis of ipsilateral re-expansion pulmonary oedema (RPO) was made. After transfer to the intensive care unit, the patient was treated with supplemental oxygen and continuous positive airway pressure via a full facemask, intravenous fluids, and morphine. Within 24 hours there was normalisation of his chest film, gas exchange, and packed cell volume (0.38). Two days after discharge from the intensive care unit, however, there was a recurrent pneumothorax requiring thoracoscopic pleurodesis and stapling of an apical bleb. The patient was discharged well on the third postoperative day.

RPO after aspiration of a pneumothorax was first reported in 1959.¹ The incidence is between 0.9% and 14%² with many cases being asymptomatic with rapid resolution of radiographic infiltrates. Associated hypotension and shock is less common and is likely to be a major contributor to the fatalities recorded in association with the syndrome.³ The incidence is increased in patients under 40 years of age and is related to the duration of lung collapse, its severity, and the rate of re-expansion.² However, RPO and shock can occur regardless of the duration of lung collapse and in the absence of suction. The onset is usually within hours and almost always involves the ipsilateral lung.³ The mechanisms involved include increased vascular permeability and protein leak-

Department of
Respiratory Medicine,
Royal Prince Alfred
Hospital, Sydney,
Australia
A V Desai
P R Phipps
D J Barnes

Correspondence to:
Dr D J Barnes, Suite 413,
RPAH Medical Centre, 100
Carillon Avenue, Newtown,
2042 Sydney, Australia
(e-mail:
dbarnes:ns.wbigpond.net.au).