patients reported the development of a tender lump, which, being part of a systemic disease, was not confused with a carcinoma. One of the patients, however, was subjected to simple mastectomy.[1]

Hard, fixed masses in the breast suggest carcinoma. Fixation to the skin and surrounding tissues with retraction of the nipple of recent onset are all accepted features of the condition, and our patient had axillary lymphadenopathy, further substantiating the clinical diagnosis. In retrospect the generalised symptoms and unusually high erythrocyte sedimentation rate in the absence of any definite cause for this—for example, obvious secondary deposits in bone or bone marrow disease—might have suggested an underlying disease process, perhaps of rheumatic nature, but biopsy of the breast lesions would still have been required.

While the common clinical presentation of a fixed breast mass is usually due to carcinoma, this case and others reported[1-4] emphasise the importance of thorough investigations for those patients in whom additional symptoms suggesting a systemic disorder are present. Thus unnecessary mastectomy will be avoided.

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Hepatitis A in homosexuals

In the United Kingdom hepatitis B is a common cause of acute jaundice among homosexuals.[1] In the last six months we have noticed an increase in the contribution by hepatitis A to the overall numbers of outpatients with acute hepatitis or a history of recent hepatitis attending the department of genitourinary medicine at this hospital.

Methods and results

The development of sensitive radioimmunoassays to detect IgM anti-hepatitis A antibody[2] makes possible an accurate serological diagnosis of recent infection by hepatitis A virus. Over the two periods July-December 1979 and July-December 1980 we examined records and results of serological tests for all men who presented to the department with a history of acute hepatitis within the preceeding three months. Hepatitis B surface antigen (HBsAg) was detected by a sensitive solid-phase radioimmunoassay.[3] Non-B sera (HBsAg negative) were tested for IgM anti-hepatitis A antibody.

Hepatitis B was the most common cause of jaundice (table) in 1979. In contrast, over the same period in 1980 the incidence of non-B hepatitis exceeded by nearly threefold the incidence of hepatitis B. Of the 26 patients with non-B hepatitis, 24 had hepatitis A. Half of these patients were jaundiced when seen in the clinic. The illnesses were spread over the six-month period and were therefore not associated with a common source.

Incidence of hepatitis among patients with jaundice attending over the period July-December

	Hepatitis B	Non-B hepatitis	IgM anti-HAV positive
1979	4*	2	Not tested†
1980	9*	26	24

*One serum specimen tested elsewhere.
†Test not available in 1979.

Three of the 24 patients with hepatitis A had recently travelled abroad to areas where hepatitis A virus is prevalent. One of these three was a heterosexual HBsAg carrier who gave no history of a sexually transmitted illness. Twenty-three patients were homosexual (including one bisexual). They had a history of sexually transmitted diseases, 14 (61%) having suffered from gonorrhoea and 12 (52%) from syphilis. Nine (39%) had previously acquired gonorrhoea, syphilis, and hepatitis B, and only five (22%) had no history of any of these infections.

Comment

Transmission of hepatitis A is usually considered to be faecal-oral. Among homosexuals in the United States of America oral-anal contact predisposed to hepatitis A,[4] and such practices may have facilitated the infections reported here. In our experience other practices such as inserting a hand or even a fist into the partner's rectum, although not uncommon in the United States of America, are seldom admitted to in the United Kingdom. Gross faecal contamination of the environment must result from this behaviour.

There has been a steady decline in the incidence of infectious hepatitis, almost certainly hepatitis A, over the past 10 years in the United Kingdom. The slight increase in hepatitis A in 1980 (N D Noah, personal communication) is insufficient to account for the pronounced increase seen in our patients. It is possible that wider acceptance of the behaviour described above has led to the present outbreak of hepatitis A among homosexuals and that hepatitis A should now be added to the growing list of infections associated with homosexual practices in the United Kingdom.

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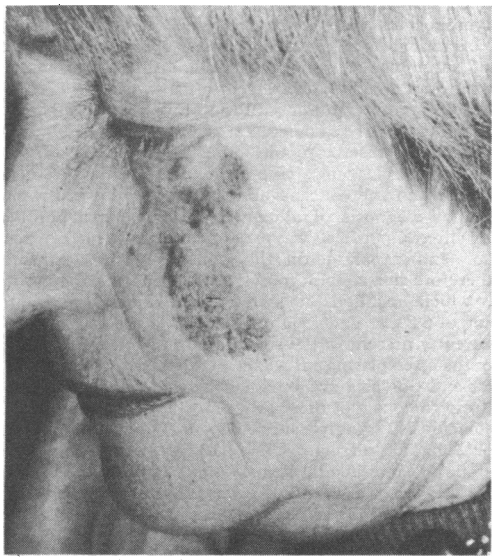
Proprietary depigmenting cream used wrongly for lentigo maligna

Claims that topically applied hydroquinone can reduce melanin pigmentation have a sound basis,[1] and creams containing this chemical have been available over the counter in the USA for more than 10 years and in the United Kingdom for at least two. A recent report has warned of the danger of the mistaken use of such creams in superficial malignant melanomas.[2] We describe a patient whose use of such a depigmenting cream delayed the surgical excision of a lentigo maligna for a year, allowing it to grow to a considerable size.

Case report

A 57-year-old white woman presented with a four-year history of a pigmented lesion on the left cheek. She had been brought up and spent much of her adult life in South Africa, and her skin had always burned easily in the sun. A basal-cell carcinoma had been excised from her face when she was aged 37. The lesion on her cheek had grown slowly for about two years and thereafter more quickly. She had tried various proprietary creams with little success, and for this reason had changed to a cream containing 2% of hydroquinone, available over the counter. She had persisted with this for about a year as it had undoubtedly caused some depigmentation, particularly in the centre of the lesion (figure). The lesion had continued to enlarge, however, eventually reaching 4·5 cm in diameter. A skin biopsy specimen confirmed the clinical diagnosis of lentigo maligna, and she was referred to the department of plastic surgery for excision and grafting.



Effect of depigmenting cream on lentigo maligna, with loss of pigment in centre of lesion.

Comment

A monograph issued by the United States Food and Drug Administration in 1978[3] concluded that products containing between 1·5 and 2·0% concentrations of hydroquinone were safe for sale over the counter, subject to warning about avoiding the sun, skin and eye irritation, and their use in children. These products were considered to be effective when used topically for several rather vaguely defined conditions including "age spots" and "liver spots." It is here that the difficulty arose with our patient.

A lentigo maligna (melanotic freckle of Hutchinson) usually appears on the face of an elderly person as a flat brown stain that gradually becomes larger. Lay people may be excused for confusing this with an age spot. The treatment of choice for lentigo maligna, however, except perhaps in the very old, is surgical excision, since up to a third of cases may change into a frankly invasive melanoma.[4] Delay in treating a growing lesion makes surgery more difficult and must also increase the chance of this malignant transformation occurring. The recommended warning, to stop hydroquinone applications if no depigmentation is detected after two months, had no effect on our patient as depigmentation definitely occurred in the centre of the lesion, confirming the ability of hydroquinone to affect abnormal as well as normal melanocytes.[5]

Our patient did not develop an invasive malignant melanoma, but her lentigo maligna escaped excision for an extra year while she used the hydroquinone preparation. We believe that general practitioners and dermatologists should be aware of this possibility as topical depigmentating agents are now generally available over the counter in this country. If further problems are reported with these agents the question of making them available only on medical prescription should be reopened.

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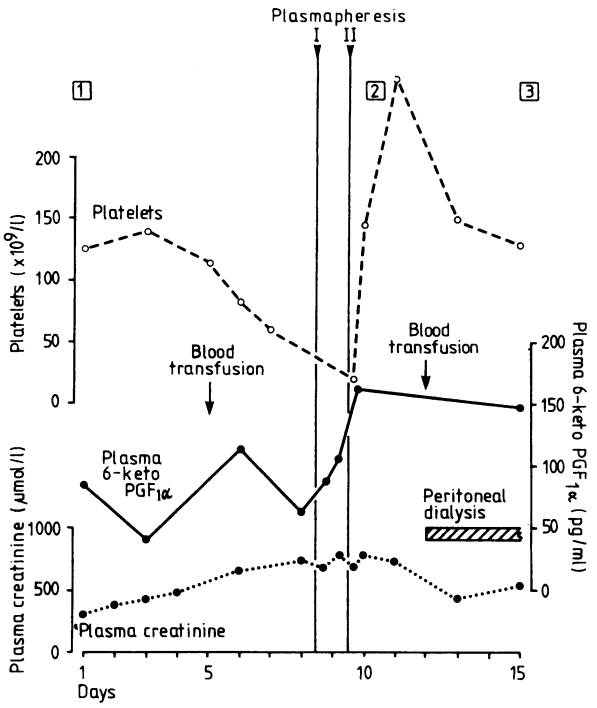
Plasmapheresis in the haemolytic-uraemic syndrome in children

Recent hypotheses concerning the pathogenesis of the haemolytic-uraemic syndrome and thrombotic thrombocytopenic purpura include the absence of a plasma factor needed for prostacyclin (PGI_2) production by vascular endothelium,[1] the presence of a circulating inhibitor to PGI_2 production,[2] or both. The finding of PGI_2 deficiency in the haemolytic-uraemic syndrome in adults,[3] supports such concepts. The possible therapeutic value of plasmapheresis, using normal plasma for the exchange, has been suggested either to replace a missing plasma factor or to remove an inhibitor. Our findings after plasmapheresis in the childhood haemolytic-uraemic syndrome are relevant to this debate.

Case report

A 3½-year-old boy with no relevant history presented with cough, anorexia, and vomiting. On admission he was clinically anaemic and dehydrated with widespread purpura. Haemoglobin concentration was 6·2 g/dl, platelet count 60×10^9/l (60 000/mm^3), and serum fibrin degradation products 80 mg/l. There was red-cell fragmentation on the blood film and a serum creatinine concentration of 200 μmol/l (2·26 mg/100 ml). The haemolytic-uraemic syndrome was diagnosed, and he was treated with aspirin (20 mg/kg/day) and dipyrimadole (5 mg/kg/day). After rehydration his urinary output was satisfactory and remained so throughout his first admission. His condition gradually improved with conservative management alone, and on discharge 10 weeks later he was clinically well and there was no evidence of persisting haemolysis. He had continuing microscopic haematuria, however, proteinuria of 1·5 g/24 h, and evidence of impaired glomerular filtration (^{51}Cr-EDTA clearance of 42 ml/min/1·73 m^2).

Readmission 10 days later was precipitated by recurrent vomiting. Investigations showed evidence of recurrence of the disease with oliguria (figure). Percutaneous renal biopsy was carried out, and histological examination showed an occlusive endarteritis affecting the afferent arterioles, supporting the clinical diagnosis. Continuous-flow plasmapheresis (Aminco) was performed on two consecutive days. One litre was exchanged on both occasions, using fresh frozen plasma as replacement solution, after which the platelet count rose. Studies of PGI_2 metabolism over this period showed



Haematological values suggesting recurrence of disease with oliguria.
□ = Test of ability of patient's plasma to stimulate PGI_2 release from exhausted rat aortic rings: 1, reduced activity (day 1); 2, approximately normal activity (day 9); 3, intermediate activity (day 15).

Conversion: SI to traditional units—Creatinine: 1 μmol/l ≈ 0·0113 mg/100 ml. Platelets: 1×10^9/l = 1000/1mm^3.