SHORT REPORTS

Lassa fever: retrospective diagnosis of two patients seen in Great Britain in 1971

Lassa fever was first described by Frame *et al*[1] in 1970, but it was not until three outbreaks had occurred in Jos, Liberia, and Sierra Leone that the disease attracted general attention,[2-4] especially as the mortality of these outbreaks averaged 45%. We describe two patients admitted under our care in 1971 who recovered. The diagnosis was confirmed retrospectively by serological tests.

Case 1

This patient, a nursing sister, had worked in Sierra Leone for nearly four years. On the morning of 10 June 1971 she became ill at Serabu Hospital with general aches and pains, fever, and rigors and treated herself for malaria without improvement. Her temperature persisted and varied between 38° and 40°C, and she had severe anorexia, nausea, and vomiting. Despite courses of chloramphenicol and ampicillin the symptoms persisted and she was transferred to Freetown, where pseudomonal septicaemia was diagnosed and she was given a course of prednisone and cloxacillin. Her condition improved and she was transferred to the Tropical Diseases Unit in Liverpool for further investigation. She arrived by air from Sierra Leone via Las Palmas, with an overnight stop at the airport hospital in London on 8 July.

On admission she had been feverish for two days (38°C), had a severe right-sided iridocyclitis, and felt weak. Findings on physical examination were essentially normal, except for slight tenderness in the right hypochondrium. She was managed as a case of pyrexia of unknown origin, isolated in a side ward, and barrier-nursed. Nothing abnormal was found on investigation, except for hookworm and stronglyoides, for which she was treated. Her iridocyclitis subsided after a course of subconjunctival hydrocortisone, her condition continued to improve, and she was discharged on 23 July with a diagnosis of ? pseudomonal septicaemia/? arbovirus infection.

A team from the Communicable Diseases Centre, Atlanta, visiting Sierra Leone in September 1972, tested her serum and found a complement fixation titre to Lassa fever of 1/16, indicating that her illness in June 1971 was undoubtedly Lassa fever. In 1975 the titre had fallen to 1/4.

Case 2

This patient, a doctor, had been working in Segbwema Hospital, Sierra Leone. On 21 September 1971 he developed flu-like symptoms and despite a course of tetracycline by mouth continued to feel ill, his temperature rising to 38°-39°C over the next three days. On 24 September he left Sierra Leone on a scheduled flight "feeling grim," and on arrival in the UK next day proceeded to his home in Kirton, Lincs. On the 27th he still complained of malaise, headache, anorexia, and generalised joint pains. On examination he looked ill, was feverish, and had a sallow complexion. Results of physical examination were essentially negative, and blood was sent for investigations. No malaria parasites were seen, and the only relevant findings were a white cell count of $2{\cdot}6\times10^9$/l (2600/mm^3), a platelet count of 148×10^9/l (148 000/mm^3), and an erythrocyte sedimentation rate of 55 mm in the first hour. On 1 October he developed pain under the right costal margin and also in the right shoulder, made worse by deep inspiration, and as his fever persisted he was admitted to London Road Hospital, Boston, Lincs, the same day. Findings on physical examination remained essentially negative, except for signs of weight loss, and *x*-ray pictures of chest and abdomen as well as results of agglutination tests for typhoid, paratyphoid, and brucella were normal.

The day after admission his temperature fell; no specific treatment was given, and he was discharged on 4 October diagnosed as having had a virus infection. A fluorescent antibody test to Lassa fever was done in 1972 and gave a titre of 1/8, indicating recent Lassa fever. He has since been used as a plasma donor for critically ill Lassa fever patients at Segbwema Hospital.

Comment

By the time our first patient came to Liverpool she had been ill for 28 days, and it is more than likely that she was no longer excreting virus, although Lassa virus has been isolated from the urine 32 days after onset of the disease.[5] In Serabu, at a stage of "maximal infectivity," she was nursed in the convent for three weeks and had many and frequent visitors. She was not barrier-nursed. None of the personnel looking after her developed the disease, and all were negative for serological evidence of subclinical infection.

Our second patient, on the other hand, arrived on a scheduled flight from Sierra Leone within four days of the onset of symptoms; none of his fellow passengers, family, or laboratory or medical personnel looking after him at home and in hospital developed Lassa fever, although only the "normal" care for a probable virus infection was taken. The medical practitioner in charge of the case had no antibodies to Lassa fever when tested in 1976.

We thank Dr W Gibson Barrie for allowing us to use the hospital records in case 2.

1 Frame, J D, *et al*, *American Journal of Tropical Medicine and Hygiene*, 1970, **19**, 670.
2 White, H A, *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 1972, **66**, 390.
3 Monath, T P, *et al*, *American Journal of Tropical Medicine and Hygiene*, 1973, **22**, 773.
4 Fraser, D W, *et al*, *American Journal of Tropical Medicine and Hygiene*, 1974, **23**, 1131.
5 Monath, T P, *et al*, *American Journal of Tropical Medicine and Hygiene*, 1974, **23**, 1140.

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Cryosurgery for axillary hyperhidrosis

Excessive axillary eccrine secretion causes ruined clothing and extreme social embarrassment. Conservative treatment is ineffective in most severe cases. Cervicodorsal sympathectomy has been employed but is too radical for axillary hyperhidrosis alone.[1] Local treatment by excision of the worst affected areas is surprisingly recent,[2] and gives satisfactory reduction of axillary secretion[3]—as it did in a personal series of five cases. Nevertheless, the resultant scar from excision of up to a 5-cm width of skin is apt to hypertrophy or keloid, stretching or contracture.

Here we report the results of surface nitrous oxide cryoprobe applications to the affected skin in four young women with axillary hyperhidrosis. We thought it likely that two minutes of freezing would cause permanent damage to the sweat glands, which are at the level of the mid-dermis.

Patients, methods, and results

Treatment was carried out in a well-ventilated room in the outpatient department. The first axilla was allowed to heal before treating the second side. All the hypersecretory area was marked out. In our experience the glistening of minute beads of sweat caused by a light shining obliquely on to the skin has allowed more precise demarcation of the hyperhidrotic area than to the use of dyes such as starch/iodine or quinazarin. Up to 20 ml of lignocaine, 1% solution, was used for local anaesthesia. The cryoprobe tip used for the first two patients was elliptical and measured 13×9 mm. Eleven applications were required for each axilla and this treatment took about 45 minutes. A circular 18-mm diameter probe tip used subsequently required only seven applications and treatment time was reduced to about 30 minutes. After treatment normal activities were resumed and a dressing was advised only if discharge was troublesome.

All four patients had had persistent severe staining of their clothing in both cold and hot weather; conservative treatment including frequent washing and aluminium chlorhydrate deodorants had failed. Their ages in order of presentation (duration of symptoms in parentheses) were 26 (11 years), 31 (6 years), 16 (3 months) and 14 years (4 years).

In the first two patients, where the smaller cryoprobe tip was used, slight excessive secretion with minimal staining of clothing persisted for 4-5 months, and this arose from the gaps between sites of application. A further treatment was given to all four axillae, and one required a third application. Three of these four axillae have been observed for a year since the last treatment and one for nine months. All have less than normal amount of sweating, and this has not tended to increase. No full-thickness skin loss nor subcutaneous cellulitis arose as a result of the treatment. The two patients on whom the larger cryoprobe tip was used had excellent results with only one treatment to each axilla (follow-up in one 14 and in the other 10 months). At two application sites in one of these patients (and at two in a furher patient under treatment) full-thickness skin loss about 10-15 mm in diameter occurred but soon healed. Late sequelae included depigmentation and almost complete depilation (a welcome side effect), but there was no noticeable change in skin contour or texture, and no sebaceous cysts have arisen.

Discussion

The results to date of cryosurgery for axillary hyperhidrosis have been encouraging. The 18-mm diameter cryoprobe tip seems to be much more effective than the smaller one used at first, though it did cause an occasional small area of necrosis.

The two patients treated with the larger cryoprobe tip required less medical and nursing time, had less local infection, and had less disturbance to their usual routine compared with excision. In our experience the final cosmetic result was better after cryosurgery than after excision.

[1] *Lancet*, 1974, **2**, 91.
[2] Skoog, T, and Thyresson, N, *Acta Chirurgica Scandinavica*, 1962, **124**, 531.
[3] Ellis, H, *British Journal of Hospital Medicine*, 1972, **7**, 641.

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Cigarette smoking and cancer of bladder and lung

Cigarette smoking is an aetiological factor in malignant disease in the lung, bladder, kidney, and larynx.[1] Hence some patients might be expected to develop more than one of these tumours. This paper records a series of eight such cases.

Between 40 and 50 patients with carcinoma of the bladder are seen each year by one radiotherapy firm, in consultation with surgical colleagues in the North-west Thames Area. From these patients are drawn cases for clinical trials of the value of hyperbaric oxygen in radiotherapy,[2] where a special effort towards detailed follow-up study is made including a routine chest *x*-ray film every six months. Four of the eight patients reported here (see table) come from the 124 patients included in this group between January 1966 and December 1973. The other four cases were among those not suitable for the trials.

Discussion

Of all the patients included in the hyperbaric oxygen trials, 74% gave a history of smoking over five cigarettes a day for more than five years. All eight patients described here were men who gave a history of heavy cigarette smoking. There appears to be no special pattern of time relationship between the occurrence of the two tumours nor any evidence that the treatment of the first tumour had any influence on the appearance of the second.

So far we have not seen a patient treated for carcinoma of the lung subsequently developing carcinoma of the bladder. As a longer latent interval with regard to cigarette smoking has been suggested for bladder cancer than for lung cancer such cases might be expected.[3] The number of patients surviving after treatment of lung cancer is similar to that after treatment for bladder cancer. This is because the greater frequency of lung cancer is compensated for by the better overall survival in bladder cancer. This can be illustrated by considering the cancer statistics for England and Wales.[4] In the year 1970 registration of male cases totalled 23 797 for carcinoma of trachea, bronchus, and lung and 5197 for carcinoma of the bladder. The percentage of male patients registered in 1962 and 1963 surviving for five years was 4·5% for lung and 27·8% for bladder. We can therefore expect 846 "lung survivors" and 982 "bladder survivors." Despite this equality in numbers at risk the higher incidence of lung cancer would make it probable that only one case of bladder cancer would be detected in patients treated for lung cancer during a period when five cases of lung cancer would be found in those treated for bladder cancer.

The pathological evidence for the existence of separate primary tumours has been carefully examined in each case. The histological appearance of the tumours was distinctly different in six patients; in one case the lung carcinoma was diagnosed by cytological study of sputum but the oat cells seen were clearly different from those in the histological material from the bladder. In one case, however, the diagnosis of lung carcinoma rests upon the naked eye findings at necropsy, as unfortunately no material was taken for histology. One lung tumour was clearly an adenocarcinoma, a type more commonly observed in non-smokers.

The appearance of a solitary tumour in the lung of a patient who has been treated in the past for carcinoma of the bladder may not be a metastasis and may be due to a new primary carcinoma of the lung. This possibility should be considered and the patient treated accordingly if a new tumour is confirmed. Although the prognosis in carcinoma of the bronchus is not good, it remains better than that of metastasis from carcinoma of the bladder provided that adequate treatment is given. In clinical trials of the management of malignant

Details of patients

Case No	Age at presentation	Bladder cancer		Lung cancer			Survival after diagnosis of lung cancer (months)	Evidence for tumour at last follow-up or death		History of cigarette smoking
		Histology	Treatment	Interval to diagnosis (months)	Histology	Treatment of primary tumour		Bladder	Lung	
1	65	Transitional cell cancer grade II	RT	66	Oat-cell	None	Dead (5)	–	+	20/day for 56 years until death
2	59	Transitional cell cancer grade I	RT	54	Large-cell undifferentiated	Surgery	Alive (38)	–	–	10/day for 39 years. Stopped aged 49
3	72	Transitional cell cancer grade III	RT	31	Squamous carcinoma	RT	Dead (26)	–	–	25/day for 60 years. Stopped aged 74
4	57	Transitional cell cancer grade III	RT	37	Oat-cell	None	Dead (0)	–	+	40-60/day for many years until death
5	68	Transitional cell cancer grade III	RT	19	No histology. Necropsy performed.	None	Dead (0)	–	+	Smoked for 54 years until aged 67
6	61	Transitional cell cancer grade II	RT	10	Oat-cell	RT	Dead (5)	–	+	30/day for >20 years until death
7	71	Transitional cell cancer grade I	Cysto-diathermy	0	Oat-cell	RT	Alive (14)	–	?	20/day for >20 years and continues
8	76	Keratinising squamous carcinoma	None	0	Adenocarcinoma	None	†Dead (0)	+	+	Smoked for 61 years until death

RT = radiotherapy. †Necropsy examination showed, in addition, a papillary carcinoma of the parenchyma of the right kidney.