possibility, and there was usually enough time to aspirate the pericardial fluid. In only one case was there an appreciable amount of blood in the pericardial fluid, tamponade being due to the infusate in the remainder. In the two successes treatment consisted in removal of the catheter and needle aspiration of the fluid. We suggest that when a clinical diagnosis of tamponade is made treatment should be instituted without waiting for confirmation from radiographs, which may in any case be equivocal. Initially an attempt should be made to aspirate fluid through the catheter by attaching a syringe direct to its Luer connector. The aspiration of fluid other than blood supports the diagnosis; and when no more fluid can be withdrawn the catheter should be removed. No more action need be taken if the patient's condition is satisfactory. Continuing clinical deterioration or failure to obtain aspirate from the catheter are indications for urgent pericardial needle aspiration. We further suggest that a needle suitable for pericardial aspiration should be kept in the same room as patients with central venous catheters, that needle aspiration of the pericardial sac should be included in the routine resuscitation programme for cardiac arrest when a central venous catheter is in situ, and that fluid used in the resuscitation programme should not be administered through an existing central venous catheter without pulling it back at least 15 cm.

### References


1 Delfalque, R. J., *Canadian Anaesthetists' Society Journal*, 1971, **18**, 681.
2 McMahon, M. J., *British Medical Journal*, 1973, **3**, 353.
3 Friedman, B. A., and Jurgeleit, H. C., *Journal of the American Medical Association*, 1968, **203**, 1141.
4 Burton, J. J., Venecko, R., and Gross, M., *Obstetrics and Gynecology*, 1968, **32**, 556.
5 Kline, I. K., and Hofman, W. I., *Journal of the American Medical Association*, 1968, **206**, 1974.
6 Thomas, C. S., Carter, J. W., and Lowder, S. C., *Archives of Surgery*, 1969, **98**, 217.
7 Brandt, R. L., *et al.*, *American Journal of Surgery*, 1970, **119**, 311.
8 Fitts, C. T., *et al.*, *Journal of Trauma*, 1970, **10**, 764.
9 Bowe, D. K., *et al.*, *Archives of Surgery*, 1973, **106**, 868.
10 Adar, R., and Mozes, M., *British Medical Journal*, 1971, **3**, 746.
11 Homesley, H. D., and Selenik, J. S., *American Journal of Obstetrics and Gynecology*, 1971, **109**, 1216.
12 Dane, T. E. B., and King, E. G., *British Journal of Surgery*, 1975, **62**, 6.


# SHORT REPORTS

## Minor Epidemic of Trichophyton rubrum

Within two weeks three men in the same geriatric ward had an erythematous rash around the buttocks. In one the eruption had been present for several months, but the other two developed a similar rash within a few days of each other. All three were ideal candidates for a Candida infection as they were debilitated, intermittently incontinent, and had been on broad-spectrum antibiotics; one was a diabetic. They proved, however, to have a dermatophyte infection. They shared the use of a commode seat and as only their three names were written on the elastoplast stuck on the commode back rest, it suggested that the commode was responsible for the spread of infection. In a recent outbreak of dermatophytosis the commode seat was incriminated in the transmission of fungus.[1]

### Case Histories

An 88-year-old diabetic hemiplegic developed an erythematous rash on his buttocks which failed to respond to fluorinated steroid ointment and Castellani's paint. He was intermittently incontinent and had had courses of tetracycline. The non-irritating erythematous scaling rash extended from the buttocks to the perineum, thighs, and sacral region. Mycological examination confirmed the clinical diagnosis of dermatophyte infection of the buttocks and mild scaling in the toe clefts also proved to be fungal in origin.

A mildly demented 83-year-old intermittently incontinent hemiplegic, who had previously been on broad-spectrum antibiotics, developed a non-irritating erythematous scaling rash on buttocks, sacrum, thighs, perineum, and scrotum. Mycological examination confirmed the dermatophyte infection, but no fungus was present in the toe clefts.

A 70-year-old demented, intermittently incontinent hemiplegic with prostatism, who had recently been on broad-spectrum antibiotics, developed an itchy, erythematous scaling rash on the buttocks, perineum, scrotum, thighs, and lower back. The dermatophyte infection was confirmed, but there was no evidence of fungus on the feet.

Mycological culture showed that all three men had *Trichophyton rubrum* infection on the buttocks; the first patient had *T. rubrum* in the toe clefts also, but the other two did not. They all cleared on griseofulvin systemically and local applications.

### Discussion

*T. rubrum* infection usually affects the feet[2] and hands; some think that if the trunk is affected it is secondary to infection of limbs. In these patients one man had *T. rubrum* on his feet with secondary spread to the buttocks and groins. Consequently he probably contaminated the commode seat with shreds of skin and was instrumental in transmitting the infection directly on to the buttocks of the other two men who shared the commode. Neves and Zavier,[3] investigating the transmission of tinea cruris in a sanatorium, postulated that non-living objects (such as toilet seats) can act as vehicles, carrying fungi to the skin of others. When dermatophytes reach the skin of susceptible hosts they grow under favourable conditions—for instance, poor hygiene, increased sweating and secretions—and produce lesions. The cases under discussion comply with these criteria and the situation was aggravated by the trauma[4] to the buttocks of excess pressure and products of incontinence. Pressure and incontinence are the most likely causes of an erythematous rash on the buttocks and groins of any long-term inpatient, but psoriasis, eczema, erythrasma, and fungal infections must be considered in the differential diagnosis. Monilia is the most common fungal infection of the perineum, but as these cases illustrate, a dermatophyte infection could quickly escalate into a minor epidemic.


1 *British Medical Journal*, 1975, **1**, 745.
2 Rosenthal, S. A., *et al.*, *Archives of Dermatology*, 1967, **96**, 51.
3 Neves, H., and Xavier, N. C., *British Journal of Dermatology*, 1964, **76**, 429.
4 Baer, R. L., and Rosenthal, S. A., *Journal of the American Medical Association*, 1966, **197**, 1017.



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## Treatment of Hypertriglyceridaemia after Renal Transplantation

Hyperlipoproteinaemia, which is often associated with premature atherosclerosis, occurs in many patients after renal transplantation owing to an accumulation of very-low-density lipoproteins (type IV hyperlipidaemia).[1 2] It is attributed to corticosteroid therapy,[1] which through the increase of basal insulinaemia may stimulate the hepatic secretion of triglycerides.[3] Since it is not apparently related to renal function or transplant age[1] patients with successful transplants are exposed to a complication which per se or in combination with other conditions—arterial hypertension, glucose intolerance, arterial calcification, and hyperuricaemia, for example—predisposes to accelerated atherosclerosis.

A low calorie, low carbohydrate diet reduces the triglycerides to normal in most patients with essential type IV hyperlipidaemia,[4] presumably by affecting the production of very-low-density lipoproteins and reducing the precursors of triglycerides. We therefore recommended such a diet for six adults with normal renal function

*Change in Serum Lipid Levels in Transplant Patients after Dieting for One Month*

| Case No. | Sex | Age (Years) | Time since Transplantation (Months) | Plasma Creatinine (μmol/l) | Plasma Cholesterol (mmol/l) | | Plasma Triglyceride (mmol/l) | | Pre-β-lipoproteins (%) | | Body Weight (kg) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Before | After | Before | After | Before | After | Before | After |
| 1 | F. | 31 | 14 | 106 | 4·0 | 4·1 | 2·3 | 1·5 | 26 | 21 | 63·0 | 62·0 |
| 2 | M. | 30 | 10 | 141 | 4·7 | 5·7 | 3·8 | 2·0 | 31 | 16 | 57·0 | 54·6 |
| 3 | M. | 34 | 15 | 133 | 7·3 | 3·9 | 4·6 | 1·9 | 40 | 22 | 71·0 | 65·8 |
| 4 | M. | 36 | 7 | 133 | 8·0 | 6·3 | 2·5 | 1·8 | 31 | 10 | 74·0 | 73·4 |
| 5 | M. | 42 | 18 | 141 | 7·3 | 6·5 | 5·1 | 1·9 | 45 | 19 | 74·5 | 70·0 |
| 6 | F. | 33 | 13 | 115 | 7·0 | 7·0 | 3·6 | 1·8 | 25 | 19 | 64·5 | 62·5 |
| Mean±S.D. | | 34·3 | 12·3 | 124 | 6·3±1·6 | 5·6±1·3 | 3·7±1·1 | 1·8±0·2 | 33±7 | 17±4 | 67·3±6·9 | 64·7±6·6 |
| Significance | | | | | N.S. | | P<0·01 | | P<0·01 | | N.S. | |

and severe fasting hypertriglyceridaemia 7-18 months (mean 12·3 months) after renal transplantation. All were on a free diet and their body weight was higher than ideal before treatment.

### Diet and Results

The suggested diet was: *breakfast*, milk 200 g; *lunch*, spaghetti or rice 50 g, beef, veal, or poultry 150 g or lean ham 110 g, vegetables (carrots, cabbages, tomatoes, lettuce) 150 g, fruit (apples, pears, peaches, oranges, apricots, mandarines) 150 g, and olive-oil 15 g; and *supper*, spaghetti or rice 50 g, lean cheese 70 g, vegetables 150 g, fruit 150 g, and olive-oil 15 g. Butter, maize oil, coconut oil, margarine, alcohol, fruit juices, sweets, and sugar were forbidden. This diet contained about 7 MJ (1700 kcal), 130 g carbohydrate, 70 g protein, and 100 g fat daily.

After a month on the diet the lipid pattern returned to normal (table). Then, to avoid excessive weight loss in individual patients, an increase of 1·7-2·5 MJ (400-600 kcal) was allowed, with the addition of protein and some fat. After six months on this regimen the lipid pattern remained in the normal range, with little fluctuation in body weight.

### Discussion

Thus even in transplanted, corticodependent patients such a diet can correct hypertriglyceridaemia. This can be of practical importance, since apart from graft rejection and infection cardiovascular disease is the most common cause of death after renal transplantation.[5] Moreover, hyperlipidaemia could contribute to the severity of renal vascular disease in kidney transplant rejection or disfunction.[2]

We conclude that the transplanted, hypertriglyceridaemic patient should be told of the danger of a high calorie, high carbohydrate diet. This might in some cases improve the long-term prognosis.


[1] Casaretto, A., *et al.*, *Lancet*, 1974, **1**, 481.
[2] Edwards, K. D. G., and Charlesworth, J. A., *Lancet*, 1973, **1**, 1192.
[3] Robertson, R. P., *et al.*, *Journal of Clinical Investigation*, 1973, **52**, 1620.
[4] Levy, R. I., Morganroth, J., and Rifkind, B. M., *New England Journal of Medicine*, 1974, **290**, 1295.
[5] Lowrie, E. G., *et al.*, *New England Journal of Medicine*, 1973, **288**, 863.



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# Metastatic Carcinoma Causing Haematemesis

We describe a case of haematemesis due to gastric metastases from bronchial carcinoma. Secondary spread of carcinoma to the stomach is unusual, and haematemesis from this cause has not been reported.

### Case Report

A 59-year-old engineer was admitted with a history of vomiting blood. Nine weeks previously he had developed low back pain, for which he had taken phenylbutazone. He had smoked 15 cigarettes per day. He was obese and shocked. There was slight epigastric tenderness on abdominal palpation but no masses could be felt. Rectal examination demonstrated melaena stool. The chest was clinically clear and the remainder of the physical examination was normal. Haemoglobin was 10·3 g/dl, P.C.V. 25·5%, W.B.C. 15·4 × $10^9$/l, platelet count and prothrombin time normal, blood urea 40·7 mmol/l (245 mg/100 ml), serum electrolytes normal, the *x*-ray film showed a large peripheral left lower zone opacity and the right hemidiaphragm was raised.

Blood transfusion was started and continued for the next 36 hours, while repeated haematemeses occurred. An emergency barium meal showed no abnormality. Bronchopneumonia developed and he died four days after admission.

At necropsy on opening the chest a solitary solid round pale tumour 8 cm wide was found in the peripheral part of the left upper lobe. It was in contact with the chest wall with invasion and pathological fracture of adjacent ribs. Further extrapleural tumour masses were seen in the right hemithorax which had not been conspicuous in the chest *x*-ray film. Below the diaphragm a large tumour had replaced most of the right kidney, with growth filling the main renal vein. Secondary deposits were also found in the other kidney, liver, spleen, para-aortic lymph nodes, and thoracolumbar vertebrae. The stomach contained fresh blood derived from two ulcers on the posterior wall of its body, 1·2 and 0·5 cm wide. They were circular with hard, smooth, rolled edges. The small and large intestines were normal. Histological examination showed all lesions to be derived from an oat-cell carcinoma of the bronchus. The site of bleeding in the gastric secondary deposits was from ulcerated submucosal veins distended with growth.

### Discussion

Carcinoma of the stomach may be primary or secondary. Though bloodborne metastases to the stomach have been reported as incidental findings in several large necropsy series,[1 2] we have not found reports of haematemesis from secondary carcinomatous deposits in the stomach. The most common tumours that spread to the stomach through the blood stream are melanomas and carcinomas of the breast and lung. Tumours of the testis and thyroid also behave in this way.[1] Those lung neoplasms which are likely to spread are undifferentiated or adenocarcinomas rather than squamous cell carcinomas. Our patient had the oat-cell variety. Spiro[3] states that if carcinoma of the lung spreads to the stomach it is usually as a diffuse submucosal growth; rarely, its appearance may simulate lymphoma with large ulcerating lesions. The stomach lesions in our patient had the appearances of umbilicated secondary deposits, which usually occur in secondary deposits from a melanoma.

Clinically the case demonstrates the necessity for a routine chest radiograph in all cases of haematemesis. Faced with the normal barium meal, gastroscopy and biopsy might have been helpful in reaching an earlier diagnosis, though this would not have affected the final outcome. The case was complicated by the history of phenylbutazone intake for low back pain, but there was no evidence of erosive gastritis at necropsy. Vertebral metastases were the cause of the back pain and bilateral renal metastases together with blood in the gastrointestinal tract the cause of the high blood urea.

We would like to thank Dr. D. N. Phear for allowing us to publish this case.


[1] Willis, R. A., *The Spread of Tumours in the Human Body*. London, Butterworth, 1952.
[2] Abrahms, H. L., Spiro, R., and Goldstein, N., *Cancer*, 1950, **3**, 74.
[3] Spiro, H. M., *Clinical Gastroenterology*. Toronto, Macmillan Company, 1970.



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