[16] Buhler, F. R., *et al.*, *New England Journal of Medicine*, 1972, **287**, 1209.
[17] Hansson, L., and Zweifler, A. J., *Acta Medica Scandinavica*, 1974, **195**, 397.
[18] Stokes, G. S., Weber, M. A., and Thornell, I. R., *British Medical Journal*, 1974, **1**, 60.
[19] George, C. F., *Clinical Science and Molecular Medicine*, in press.
[20] Amery, A., Billiet, L., and Fagard, R., *New England Journal of Medicine*, 1974, **290**, 284.
[21] Aberg, H., *International Journal of Clinical Pharmacology*, 1974, **9**, 98.
[22] Karlberg, B. E., in preparation.
[23] Hansson, L., and Aurell, M., in preparation.
[24] Tarazi, R. C., and Dustan, H. P., *American Journal of Cardiology*, 1972, **29**, 633.
[25] Hansson, L., *et al.*, *Acta Medica Scandinavica*, 1974, **196**, 27.
[26] Prichard, B. N. C., and Gillam, P. M. S., *American Journal of Cardiology*, 1966, **18**, 387.
[27] Hansson, L., *Acta Medica Scandinavica*, 1973, Suppl. 550.
[28] Dollery, C. T., *et al.*, *British Journal of Pharmacology*, 1973, **48**, 343.

SHORT REPORTS

Psittacosis and Disseminated Intravascular Coagulation

Psittacosis most commonly presents as mild pneumonia or pyrexia of undertermined origin. The following patient had severe pneumonia, myocarditis, pericardial effusion, and disseminated intravascular coagulation—a hitherto unreported complication.

Case Report

A 36-year-old man had felt ill for 10 days, and had dyspnoea, fever, and slight cough with yellow sputum. He was confused, grey, and unwell, being dehydrated with a temperature of 39·7°C, pulse of 140/minute, and blood pressure of 90/70 mm Hg. He was dyspnoeic at rest and had signs of inflammation in the left lung. A chest *x*-ray film showed extensive opacities in the left lower lobe.

During the next 36 hours he deteriorated. An arterial blood sample showed pH 7·39, Po_2 20 mm Hg, and Pco_2 36 mm Hg. He was intubated and treated with positive pressure ventilation with an oxygen concentration (FIO_2) of 100%. After one and a half hours of ventilation, his arterial blood gases were pH 7·30, Po_2 25 mm Hg, and Pco_2 33 mm Hg. He was given intravenous chloramphenicol, fucidin, tetracycline, and intramuscular streptomycin. On the sixth day he developed surgical emphysema on the right side of his neck, though there was no evidence of a pneumothorax. Chest *x*-ray films showed that his heart shadow was becoming larger and more globular in shape, indicative of pericardial effusion. There was never evidence of cardiac failure. Despite a blood transfusion on the fourth day, his haemoglobulin had dropped to 9·0 g/100 ml on the sixth and his platelet count was 47 000/mm³. Chloramphenicol was discontinued. On the seventh day the platelet count was only 40 000/mm³. The only clinical evidence of bleeding was skin bruising at the site of venous and arterial punctures. Full coagulation studies were performed (see table), and led to a diagnosis of disseminated intravascular coagulation; heparin (500 units intravenously hourly) was started and continued until the 15th day. Streptomycin and tetracycline were discontinued and doxycycline begun.

Coagulation Studies

Days after Admission	Platelets /mm³	Prothrombin Time (Seconds)	Kaolin-Cephalin Time (Seconds)	Fibrinogen Titre	F.D.P. µg/ml
5	Reduced	0	0	0	0
6	47 000	0	0	0	0
7	40 000	3 mins.	6 mins.	No clot	320
8	79 000	23	45	1:2	320
9	47 000	22	37	1:8	320
10	43 000	28	59	1:8	320
15	140 000	15	36	1:32	40
27	172 000	17	48	0	40

F.D.P. = Fibrinogen degradation products.

By the eighth day his condition and the results of the coagulation studies had improved. On the tenth day he developed multiple ventricular ectopic beats and a short run of ventricular tachycardia. Intravenous lignocaine and oral practolol abolished the ventricular arrhythmias. He then developed difficulty in speaking and was unable to swallow. A tracheostomy was performed and he was ventilated. He maintained his good progress and was discharged 30 days after admission. Antibody titre to psittacosis was 1/16 on the third and seventh day and 1/256 on the thirtieth day. When seen in follow-up one month later the patient was fit and well, apart from slight generalized weakness. He mentioned that he kept tropical fish and that his only contact with birds was in the pet shop from which he bought his fish food.

Discussion

Psittacosis usually produces a mild respiratory infection, though occasionally it may cause severe respiratory failure. The mortality is mainly from complications and approaches 5%.[1] The considerable hypoxia in this patient necessitated mechanical ventilation. Psittacosis may also involve the cardiovascular system,[2 3] and may damage the liver, producing jaundice. Here it resulted in disseminated intravascular coagulation (D.I.C.), which is characterized by the deficiency of fibrinogen and other clotting factors and thrombocytopenia. Several viruses may cause D.I.C., which may be transient, fatal, or pass unnoticed. The platelet count is almost invariably depressed, but, as here, there is no correlation between the absolute level of circulating platelets and the haemorrhagic tendency.

In treating D.I.C. the first step is to treat its cause—for example, septic shock.[4] D.I.C. may be reversed with intravenous heparin; in D.I.C. with infectious mononucleosis a suitable dose may be 12 000 units over 24 hours.[5]

I should like to thank Lord Waverley and Dr. D. G. Price for their help and for permitting me to report details of a patient under their care, and Drs. J. V. Dadswell, W. A. P. Hamilton, and F. Hampson for reviewing the manuscript.

[1] *British Medical Journal*, 1972, **1**, 1.
[2] Dymock, I. W., *et al.*, *British Journal of Clinical Practice*, 1971, **25**, 240.
[3] Ward, C., and Ward, A. M., *Lancet*, 1974, **2**, 734.
[4] Corrigan, J. J., Jordan, C. M., and Bennet, B. B., *American Journal of Diseases of Children*, 1973, **126**, 629.
[5] Dodsworth, H., and Burns, A., *British Medical Journal*, 1971, **4**, 466.

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Severe Peripheral Neuropathy after Mandrax Overdose

Poisoning with methaqualone and diphenhydramine (which are available in this country as Mandrax tablets containing methaqualone 250 mg and diphenhydramine hydrochloride 25 mg) is quite common and the clinical features are well known.[1 2] This case illustrates an unusual manifestation of Mandrax poisoning causing severe peripheral motor and sensory neuropathy; no similar complication has apparently been described.

Case Report

A 73-year-old man complained of numbness, pain, and weakness in the legs and feet. He had been treated for Mandrax overdose six months ago when depressed, having taken 28 tablets in the evening. A few hours later the police found him wandering in the street confused. After gastric lavage at the casualty department he was transferred to the medical department, and treated supportively, recovering consciousness seven days later. The first thing he felt on recovery was that "my legs have gone." Later he developed numbness below the knees, together with burning pain and tingling in the legs and feet, and considerable weakness.

The legs showed definite wasting of the anterior compartments as well as of the calves. There was no dorsiflexion of toes or ankles and there was a bilateral foot drop. Both knee jerks were depressed, the ankle reflexes were present, and there was no plantar response. Vibration sense was lost below the iliac crests and joint position sense was virtually absent in the toes and grossly defective at the ankles. Below both knees sensation to touch, pin prick, hot and cold was lost. The results of extensive laboratory and radiological investigations were normal. An electromyogram showed findings characteristic of a peripheral neuropathy.

Comment

These findings leave no doubt that this patient had peripheral neuropathy and the time relation to the overdose would implicate Mandrax as a causative agent. The antihistamine component of Mandrax does not appear to contribute appreciably to its toxicity[3] and

the neuropathy was probably due to methaqualone. Methaqualone ingestion has been followed by transient paraesthesiae preceding sleep[4]; however, no persistent neuropathies have been reported. He took roughly 30 tablets—7·5 g of methaqualone—a severe overdose, since the minimal fatal dose of methaqualone may be under 8 g.[5]

Hence peripheral neuropathy should be considered as a complication—though a rare one—of Mandrax intoxication.

I am grateful to Dr. G. Danta, consultant neurologist, for his expert advice and the electromyographic studies of the patient.

[1] Lawson, A. A. H., and Brown, S. S., *British Medical Journal*, 1966, 2, 1455.
[2] Matthew, H., *et al.*, *British Medical Journal*, 1968, 2, 101.
[3] Matthew, H., and Lawson, A. A. H., *Treatment of Common Acute Poisoning*, p. 81. Edinburgh and London, Livingstone, 1970.
[4] Goodman, L. S., and Gilman, A., *Pharmacological Basis of Therapeutics*, p. 131. 4th edn. Baltimore, Williams and Wilkins, 1970.
[5] Sanderson, T. H., Cowdell, R. H., and Higging, G., *Lancet*, 1966, 2, 803.

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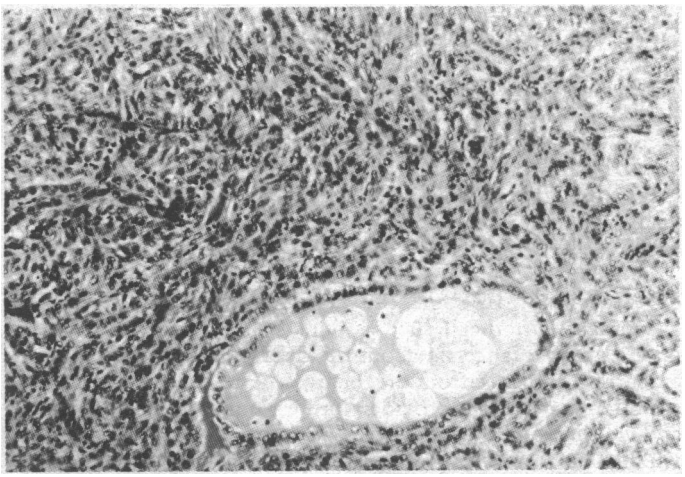
Renal Carcinoma in a Cadaver Kidney Graft Donor

There have been two reports on the unexpected finding of a small nodule on the surface of a kidney being removed from a living donor before transplantation. In Cerelli's case reported by Penn,[1] after biopsy of the nodule, the kidney was transplanted but removed 48 hours later because of the suspicion of malignancy. In Fox's case[2] histological examination of the nodule showed a renal adenocarcinoma; the kidney was removed for therapeutic reasons and not used in the recipient. Transplantation from donors with primary malignant disease outside the central nervous system has been abandoned[1 3] and apparently normal kidneys containing hidden secondary deposits have caused the recipient's death from transplanted tumour.[4] This report describes the accidental transplantation of a kidney containing a primary renal carcinoma from a cadaver donor.

Case History

The donor was a 61-year-old man who had had a fatal head injury after a road accident. Both kidneys were removed after cardiac arrest and the left kidney was used at the donor centre. The right kidney was taken by the organ matching service to Southmead Hospital, Bristol, for transplantation into a 30-year-old recipient. When the kidney was revascularized a small yellowish nodule, 1 cm in diameter, appeared at the upper pole. The nodule was biopsied and the operation completed. The transplant functioned immediately, with a creatinine clearance of 38 ml/min by the fifth day. Microscopy of routine paraffin sections of the nodule, however, showed an incompletely removed papillary renal cortical adenoma with cellular areas suspicious of malignant change. After normal excretion urography and a normal renal arteriogram, the area of the previous biopsy was widely excised.



Photomicrograph from the renal cortical tumour showing a well-differentiated tubular pattern with a solid area showing loss of acinar structure and pleomorphism very suggestive of malignant change (H. & E. × 139).

On microscopy the appearances were essentially similar to the previous biopsy but there was also an area of probable early invasion (see fig.). The final pathological diagnosis was a well-differentiated primary renal cell adenocarcinoma. One month after transplantation the kidney underwent irreversible rejection and was removed on the 44th day. No residual tumour was found on careful histological examination of the allograft. Meanwhile, necropsy had failed to show any evidence of carcinoma in the donor. The patient returned to regular haemodialysis and 18 months later, remains well, despite having had a second transplant removed because of rejection.

Comment

However carefully donors are selected, there is always the risk of transplanting a kidney containing a small malignant tumour. This case, and the recently reported similar cases,[1 2] underline the importance of viewing any nodule on a donor kidney with the gravest suspicion. The histological differentiation between adenoma and renal cell adenocarcinoma is often difficult and proved carcinoma, have been found as small as 0·5 cm diameter.[5] In the present case we thought that the tumour had been adequately excised, and no residual tumour was found in the rejected kidney. During cadaver nephrectomy any nodule, however apparently insignificant, should be removed at the time and subjected to immediate frozen section examination.

[1] Penn, I., *Transplantation*, 1973, 16, 674.
[2] Fox, M., *Transplantation*, 1973, 15, 523.
[3] Penn, I., *Malignant Tumours in Organ Transplant Recipients*, p. 7. New York, Springer-Verlag, 1970.
[4] Jeremy, D., *et al.*, *Transplantation*, 1972, 13, 619.
[5] Crocker, D. W., in *Pathology Annual*, ed. S. C. Sommers, p. 252. London, Butterworths, 1967.

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Recurrent Iritis

Catterall and Perkins[1 2] have described cases of uveitis associated with chronic prostatitis, and bacteriological examination of the prostatic fluid showed no particular organism to be predominant. Streptococci, staphylococci, and diphtheroids were grown in most cases and *Escherichia coli* in a few, but, as they stated, these organisms are usually regarded as contaminants of the male urogenital tract. They found pleuropneumonia-like organisms after careful search for them in the prostatic fluid and centrifugal deposit from these in about 10% of cases with uveitis. They emphasized that acute anterior uveitis is not necessarily caused by chronic prostatitis but is strongly associated with it. Dark and Morton[3] have discussed the association of acute anterior uveitis with chronic prostatitis. This is a case of iritis associated with trichomonal prostatitis.

Case History

An unmarried white man, aged 29, was referred to the special clinic in Manchester Eye Hospital from the uveitis clinic with a history of recurrent attacks of iritis for six months which had failed to respond to various topical treatments. There was no history or clinical evidence of syphilis or gonorrhoea and no history of a urethral discharge. Blood Wassermann reaction and gonococcal fixation test were both negative. The patient had an acute iritis; the external genitalia were normal; no urethral discharge was detected, and his urine was normal. Microscopic examination of an unstained specimen of prostatic fluid obtained by prostatic massage showed clumps of pus cells and numerous trichomonads. A presumptive diagnosis of trichomonal prostatitis was made and a course of metronidazole (200 mg, thrice daily for seven days) was prescribed. The iritis rapidly cleared up and over a course of several months there was no recurrence.

The prompt response to metronidazole suggests that there was an association between the iritis and the trichomonal prostatitis.

[1] Catterall, R. D., and Perkins, E. S., *British Journal of Ophthalmology*, 1961, 45, 109.
[2] Catterall, R. D., *Lancet*, 1961, 2, 739.
[3] Dark, A. J., and Morton, R. S., *British Journal of Ophthalmology*, 1968, 52, 907.

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