

Case Report

**A pheochromocytoma in a cadaver kidney donor:
to transplant or not to transplant?**

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Key words: pheochromocytoma; cadaveric kidney; transplantation

Introduction

It is well known that cancer can be transmitted with donor organs. Since the first report by McPhaul and McIntosh [1] in 1965, more than 150 cases of donor-transmitted malignancies have been reported [2]. There have been no reports of documented transmission of malignant pheochromocytoma to organ recipients. Penn has reported a series of six cases with primary renal tumours discovered at the time of donor nephrectomy which were widely excised and the kidneys were successfully transplanted. No tumour recurrences were discovered after an average follow-up of 103.5 months [3]. The question arises whether it is safe to transplant cadaveric kidneys of a donor who is unexpectedly found to have a pheochromocytoma of the adrenal gland. In this context the following observation may be of interest.

Case report

A 49-year-old Saudi woman with end-stage renal disease (ESRD) secondary to chronic glomerulonephritis received a cadaveric renal transplant after 4 years on regular haemodialysis. The kidney donor was a 30-year-old man who was declared brain dead after having sustained spontaneous intracerebral haemorrhage. During donor nephrectomy a 6 × 4 × 2 cm right suprarenal tumour was noticed. It weighed 31 g and most of the external tumour surface was smooth, but in few places slight irregularity and roughening was noticed. The cut surface was of uniform texture, and of light yellow-brown colour. The kidney was dissected and the tumour excised. Further exploration showed

no evidence of spread. Frozen sections yielded the diagnosis of pheochromocytoma. Part of the capsule was present which showed no evidence of tumour invasion. Subcapsular biopsy from the upper pole of the right kidney and biopsies from neighbouring lymph nodes were found to be normal on frozen sections. Subsequently, it was decided to go ahead and transplant the ipsilateral kidney as there was no clear evidence of local or distant spread. The contralateral kidney was sent to another local centre.

Later on, additional blocks were taken for paraffin sections which confirmed the diagnosis of pheochromocytoma but showed evidence of capsular invasion. A thin rim of liver tissue was attached and tumour infiltrates extended through the adrenal capsule into the liver. There were also several areas of vascular invasion in this location. The tumour was highly cellular and was formed of cords and trabecules, cells being separated by fine fibrovascular septae (Fig. 1). The tumour cells were large and polygonal with clearly demarcated cell outlines. Cells had medium sized nuclei, with mild nuclear pleomorphism. The cytoplasm was finely granular and varied from almost colourless to amphophilic. Mitotic figures were infrequent. Some cells contained intracytoplasmic yellow-brown pigment. In view of the capsular invasion and suspicion of malignancy a discussion arose whether to go back and perform nephrectomy or to leave things as they were, but on balance it was felt that there was no solid evidence that the tumour was definitely malignant. The immunosuppression consisted of prednisolone and cyclosporin. The initial post-operative course was complicated by biopsy-confirmed acute vascular rejection for which the patient received three daily pulses of 500 mg methylprednisolone and 14 sessions of plasma exchanges. Azathioprine was added to her initial immunosuppression regime. She was dialysis dependent for about 10 days, but eventually she was discharged home with a creatinine level of 180 mmol/l.

She remained reasonably well and normotensive up till 3 months post-transplant when she developed hypertension for which she was started on Adalat-SR and Labetalol. Catecholamine excretion was normal.

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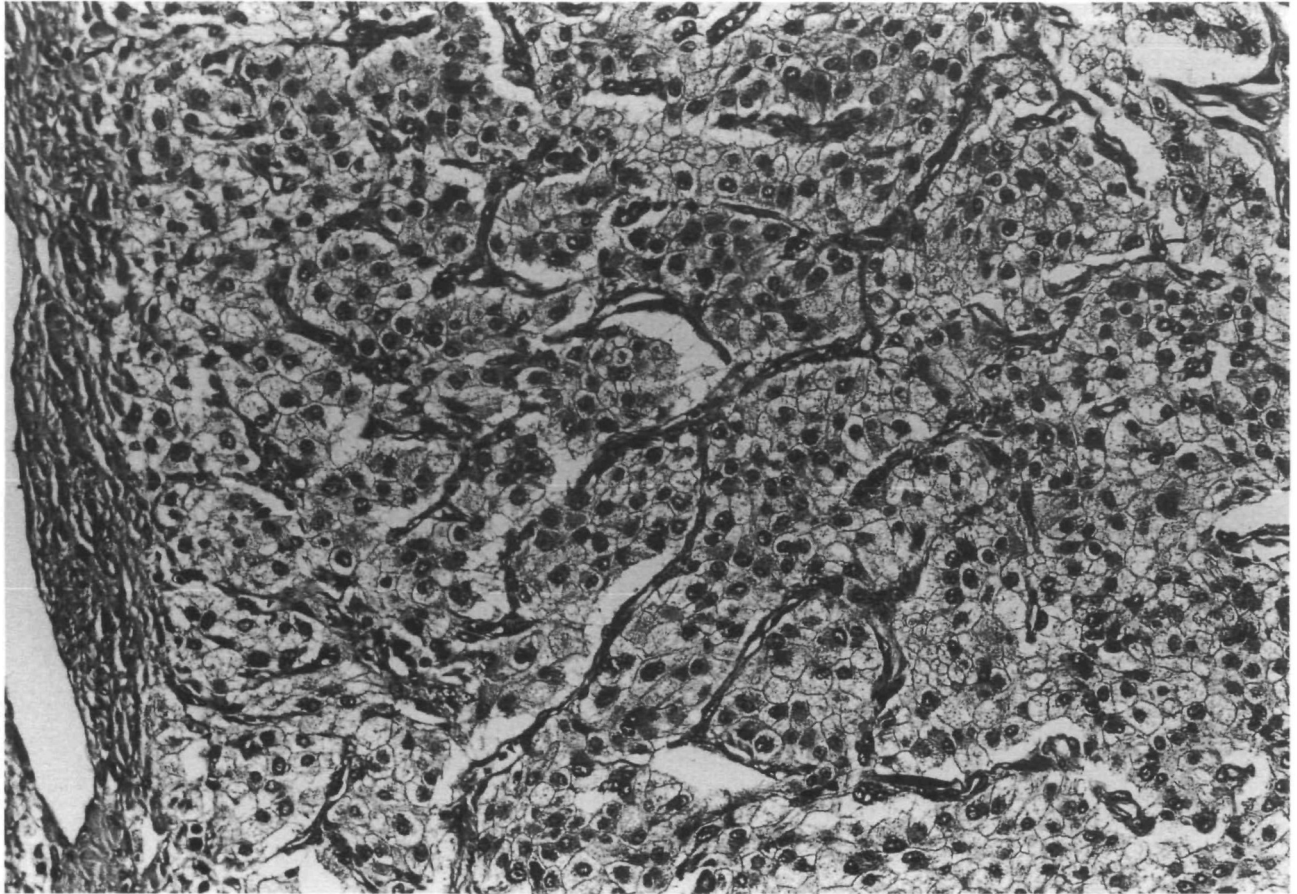


Fig. 1. Numerous clusters of cells separated by thin fibrous septae.

¹³¹I-metaiodobenzylguanidine (MIBG) radionuclide scan showed normal tracer distribution and no evidence of recurrence. Transplant renal artery stenosis was confirmed by angiography and subsequently she had a successful percutaneous angioplasty. Her blood pressure settled and the antihypertensive drugs were eventually discontinued. A repeat MIBG scan done 1 year later was found to be normal. She is now 2 years post-transplant, remains normotensive, and her graft function is fairly stable with serum creatinine around 170 mmol/l. A repeated 24-hour urinary catecholamines at 6-month intervals has remained normal. Unfortunately, the recipient of the left kidney at another centre died a few days post-operatively from fulminant septicaemia.

Discussion

In this report we describe the successful transplantation of a kidney of a donor who was unexpectedly found to have pheochromocytoma of the adrenal gland. The recipient showed no evidence of recurrence during 2 years of follow-up. Pheochromocytoma is a rare tumour, which occurs in less than 0.1% of the hypertensive population. Less than 10% of the tumours are malignant [4]. Although a number of gross and micro-

scopic features have been suggested as criteria for differentiating between benign and malignant pheochromocytoma, most authors agree that the presence of secondary tumour deposits in sites where chromaffin tissue is not normally present is the only absolute criterion of malignancy [5]. Vascular and capsular invasion, criteria for malignancy in many other organ systems, occur in both benign and malignant pheochromocytoma and cannot therefore be used to assess malignant potential [5].

Cerebral haemorrhage is a common cause of death in pheochromocytoma and therefore one could anticipate that pheochromocytomas would occasionally be seen in cadaveric kidney donors. We are not aware, however, of any previously reported case of a pheochromocytoma in a donor or transmission of malignant pheochromocytoma to recipients of cadaveric kidneys. Based on our experience we believe that it is probably safe to transplant kidneys of donors who are incidentally found to have pheochromocytoma, provided that the tumour is totally resected and there is no evidence of local or distant spread. We acknowledge, however, that a definite risk will always remain.

Most pheochromocytomas recur within 5–6 years following surgery, but much longer intervals have also been reported [5]. Our patient was followed up by 6 monthly 24-hour urine catecholamines and yearly

MIBG radionuclide scanning. The recurrence rate in malignant pheochromocytoma is less than 10% [4].

Acknowledgements. We are very grateful to Mrs Jazeela Farouk for typing the manuscript and Ms Mary Crickard for her generous contribution of the literature search.

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Received for publication: 12.4.96

Accepted in revised form: 29.5.96