A REVIEW OF POST-TRANSPLANT RENAL ARTERY STENOSIS IN SINGAPORE

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SYNOPSIS

Renal Artery Senosis occured in 44% of 34 consecutive renal transplants performed at the Singapore General Hospital over a 3 year period. In 79% of patients, renal angiography was performed routinely. Clinical and laboratory indices which suggested the diagnosis were examined. The occurrence of hypertension did not invariably reflect the presence of renal artery stenosis. The commonest type of stenosis in this series was suture line stenosis. All cases were treated conservatively. In two patients, spontaneous reversal of the stenosis occurred. The high incidence of stenosis in this series is a reflection of the policy of routine arteriography even in the absence of clinical indications.

INTRODUCTION

Renal artery stenosis (RAS) is a complication of renal transplantation which may produce hypertension, declining renal function and graft loss. A review of this complication is undertaken in a series of 34 consecutive living and cadaveric transplants performed in the Department of Renal Medicine, Singapore General Hospital, between 1978 to 1980. All patients had transplant arteriograms. The main purpose of this study is to determine the incidence of transplant renal artery stenosis in a transplant population where all patients had arteriographic studies as well as to examine the predictive value of hypertension in the diagnosis of this complication.

MATERIALS AND METHODS

Post transplant renal arteriograms were performed in 34 patients, mostly as a routine (27/34 or 79%). The immuno-suppression regime consisted of azathioprine (1.5mg/kg per day) and prednisolone (100mg initially, gradually reducing to 10mg per day six months after transplantation). Where the serum creatinine rose by more than 0.3mg%, patients were treated provisionally as for acute rejection with a 3 day course of 1 gm per day of methylprednisolone. Renal arteriograms were sometimes performed after there was no response to the steroid pulse. Hypertension (diastolic more than 100mmHg) either of sudden onset or refractory to treatment was another indiciation for arteriography.

There were 20 males and 14 females. Their ages ranged from 17 to 41 years at time of transplant. The median age was 25 years. Seven were cadaver allograft recipients while the rest were living donor related transplants.

All the arterial anastomoses were end-end between the donor renal artery and the recipient internal iliac artery.

Renal arteriograms were performed by introducing the catheter via the femoral artery.

RESULTS

Of the 34 patients studied, 15 (44%) were found to have transplant renal artery stenosis. 10 were males and 5 females. Of the 15 patients with R.A.S., 6 of them had renal arteriograms because of hypertension; in 4 patients the indication was deteriorating renal function which had not responded to a course of methylprednisolone, one because of both declining renal function and hypertension, and the remaining 4 had arteriography performed as a routine investigation.

In the group of patients without renal artery stenosis there were 9 females and 10 males. Apart from 1 patient who had hypertension, renal arteriograms in the other 18 were done as a routine.

The incidence of hypertension in these two groups of patients is summarised in Table I.

TABLE I
INCIDENCE OF PRE AND POST TRANSPLANT HYPERTENSION

	Patients with RAS	Patients with no RAS	Total
Pre-transplant Hypertension	10/15 (67%)	14/19 (74%)	24/34 (71%)
Post-transplant Hypertension	8/15 (53%)	13/19 (69%)	21/34 (62%)

The difference between pre-transplant and post transplant hypertension was not significant.

Patients were further classified as having normal BP, stable hypertension (i.e. hypertension easily controlled on the same dose of antihypertensives) or worsening hypertension which required increasing does of medication for adequate control in the post transplant period. The proportion of patients with R.A.S. in each of these categories of hypertension is illustrated in Fig. 1. Patients with R.A.S. were distributed among the three groups in similar proportions to patients without R.A.S.

Two patients who had severe hypertension from renal artery stenosis were found on repeat arteriogram 1 year later to have a marked reduction in the degree of renal artery stenosis. In one of them there was appreciable improvement on repeat arteriogram while in the other the arteriogram became normal. One patient's requirement for hypotensive drugs progressively decreased while the other did not require any at all.

Renal artery stenosis in this study was classified according to location into preanastomotic stenosis (donor artery stenosis), anastomotic or suture line stenosis (Fig. 2, 3), postanastomotic (recipient artery stenosis) and intrarenal stenosis (Fig. 4, 5) which included branch artery stenosis. Eight (53%) patients had suture line stenosis, one (7%) had preanastomotic stenosis, three (20%) postanastomotic stenosis and the remaining three intrarenal branch artery stenosis (20%). Five of the patients also have changes suggestive of vascular rejection evidenced by branch artery stenosis associated with widespread peripheral vascular pruning and focal areas of renal infarction.

DISCUSSION

In this series of 34 patients, 15 (44%) were found to have post transplant renal artery stenosis. The incidence observed in this study is way above the range of 5.1% to 31% reported in other series (1-5). This wide variation in incidence probably reflects several variables such as surgical technique and most importantly, the indications for arteriography. Lacombe (3) performed arteriography as a routine investigation in all transplant recipients and reported an incidence of 23%. The incidence of 44% in this series is therefore not suprising as 79% of patients had arteriography as a routine investigation.

It was found that the occurrence of hypertension did not invariably reflect the presence of renal artery stenosis. However its presence was helpful in suggesting the diagnosis. It would have been useful to have done transplant biopsies to exclude transplant rejection as a cause of hypertension and/or declining renal function (6).

The management of all patients was conservative. Seven patients required an increase in antihypertensive therapy for control of hypertension initially. Subsequently all had improvement of their hypertension which was reflected by the gradual reduction in the drug dosages required.

The underlying etiology of the different types of stenosis has been discussed by various authors. (7,8). The commonest type of stenosis in this series is suture line stenosis (anastomotic). Stenosis of the artery of the transplant kidney was divided into two categories by Lacombe (3): localised and diffuse. He

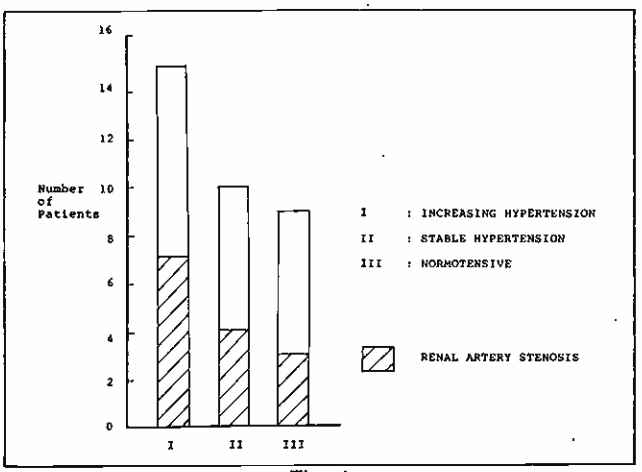

Fig. 1
Relationship of Hypertension to Transplant Renal Artery Stenosis.

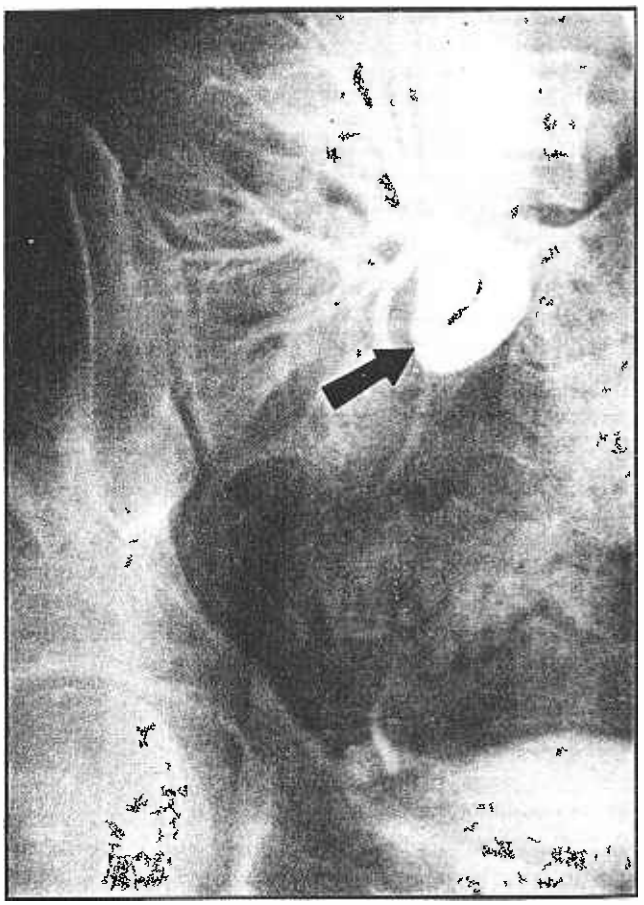

Fig. 2
Shows transplant renal artery stenosis at the Anastomotic Site. (Suture Line Stenosis)

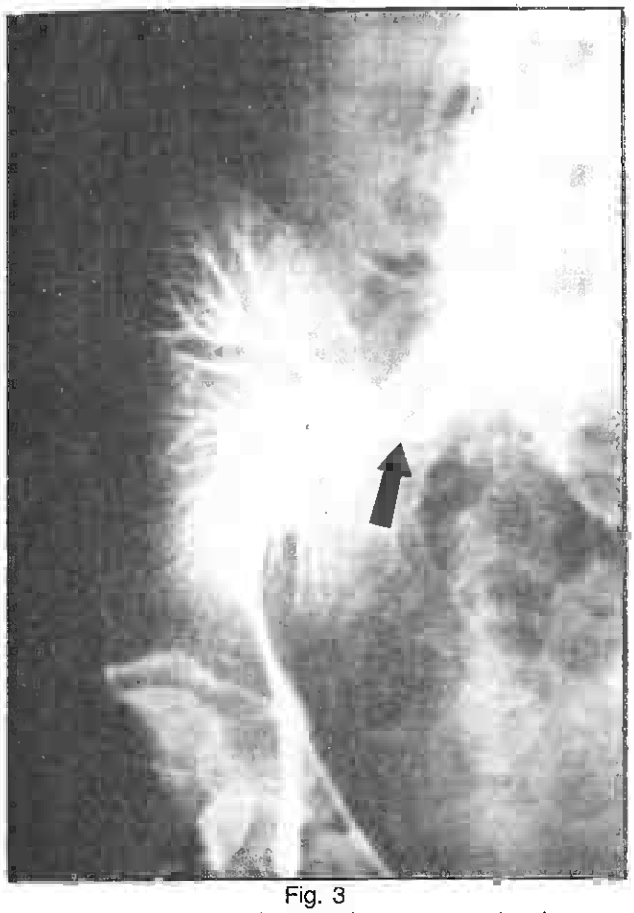

Fig. 3
Shows another transplant renal artery stenosis, also at Anastomotic Site. (Suture Line Stenosis)

Fig. 4
Shows a long intra-renal tapering stenosis (2cm long), distal to Anastomotic Site. (Post-Anastomotic Stenosis)

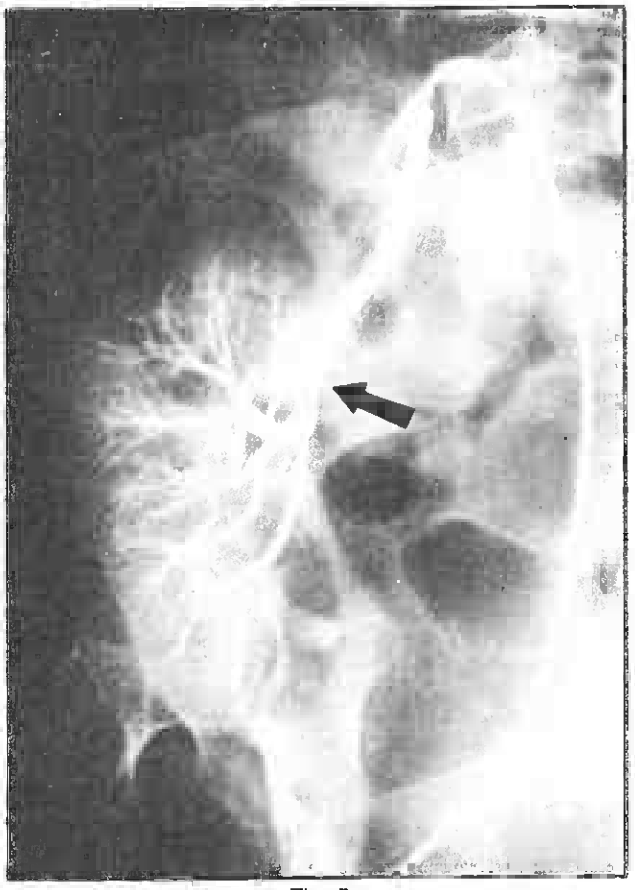

Fig. 5
Shows a slight circumferential stenosis (1.5cm long), distal to Anastomotic Site. (Post-Anastomotic Stenosis)

found underlying fibrous endarteritis with severe intimal thickening in resected segements of these arteries. This type of renal artery stenosis has been attributed to an immunologically mediated cause associated with vascular rejection (9). Four of our patients had chronic rejection and one had acute rejection.

Of the fifteen patients with renal artery stenosis, 3 have since died. Of the remaining 12, one has mild renal impairment (creatinine 2.0mg%) and another 3 have gradually deteriorating renal function. The remaining 8 patients have normal renal function.

In this review of 34 patients who underwent post transplant arteriography, hypertension was useful in suggesting the presence of renal artery stenosis but its absence did not exclude it. All patients in this series were treated consevatively and there was no necessity for surgical intervention. In fact, in 2 of our patients, spontaneous reversal of the stenosis even occured. Vegter (10) also reported reversibility of transplant renal artery stenosis (TRAS) in three patients. This reversal was associated with an improvement in hypertension as well as renal function. We believe that in both of our cases the cause of the stenosis was due to an acute vascular rejection episode. Unfortunately there was no renal biopsy documentation of rejection in these cases.

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