



Vascular Surgery and Vascular Complications in Renal Transplantation: The Case of Renal Thrombosis

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Editorial

Technical perfection of vascular surgery anastomosis of the renal vein and artery to the elected vessels of the recipient during renal grafting is mandatory. Most common vascular complications cited by Renal Transplant (RT) experts and programs, independently that the renal transplant is performed open, laparoscopic or robot assisted, are: haemorrhages, arterial or vein thrombosis, renal thrombosis and others. A recent publication on the European experience on robot assisted RT, 120 renal grafting from living donor performed in 8 different hospitals [1], listed as vascular complications: 4 cases of haemorrhages, 5 cases of intraperitoneal haematomas that motivated re-intervention, 3 cases of massive renal thrombosis with loss of the kidney and 5 cases of delayed renal function. Obviously, haemorrhages and haematomas are due to technical difficulties and suturing imperfections. However, massive renal thrombosis and delayed renal function of kidneys from living donors with short ischemia time and excellent washing perfusion need a more in deep interpretation. The following comments about the singularities of renal flow and renal flow autoregulation may facilitate the understanding of those complications, mainly when the surgeon is fully satisfied with the technical perfection of the anastomosis.

Vascular Surgery and Vascular Flow

Renal artery flow is very high and auto-regulated by the resistances localised in the intrarenal microcirculation. The flow of the iliac artery in the dog is of 40 ml/min and increases to 150 ml/min when the iliac artery is converted in renal artery by grafting a kidney: 3 times more flow with the same calibre!!! [2,3]. High flow through vascular anastomosis prevents focal thrombosis: in fact, a way of reducing the risk of immediate or delayed thrombosis in complex vascular surgery is by increasing flow, creating an arteriovenous fistula [4]. In conclusion, when vascular thrombus is detected during renal grafting, even in the worst vascular scenario, toxic factors reducing renal flow should be suspected and identified.

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The Case of Massive Renal Thrombosis

The most common causes of reduced renal flow in renal transplantation, both during surgery and in the immediate postoperative period, are endothelial lesions that increase resistance in the renal microcirculation territory. There are a number of causes of difficult detection and variable intensity, but mainly related with immunological factors and drug toxicity. In our long experience with living donors RT program [5] we have been able to document 3 cases of renal thrombosis motivated by angiotoxicity of immunotherapeutic agents incorrectly used. There was one case of massive renal thrombosis, visible in the operating room (black kidney), with a kidney from cadaver due to ABO incompatibility.

Delayed Function in the Living Donor Renal Transplantation

Diuresis should start immediate after a living donor renal grafting. If it does not occur in a period of 6 hours to 12 hours, once confirmed the ureteral permeability, the suspicions of a vascular problem is explored by eco-Doppler evaluation which may show good pulsatile activity of the renal artery, reduced arterial flow and increased resistance to flow. If the anuria persists longer, other exams may be proposed such as arteriography or even surgical re-intervention suspecting renal artery or vein stenosis. To better understand this clinical setting we propose to consider those old experiences demonstrating the reduced value of vascular constriction and stenosis versus minor lesions of the renal endothelial microcirculatory territory.

- To reduce, simultaneously, the renal flow and the filtration arterial pressure promoting anuria, renal arterial stenosis should be over 50% of its original calibre [6].

- Increase pressure in the renal vein by anastomosing the renal vein to the portal system does not modify or impair renal flow or renal function neither at short or long term [7].

- Extreme reduction of renal artery flow and pressure, below filtration pressure motivating anuria, surprisingly permits oxygenation of the kidney in the warm ischemia setting during more than 24 hours without noticeable renal function lesions, both in the experimental animals [8] and in the human clinics [9].

In conclusion, minor or severe endothelial lesions of the micro vascular territory due to immunologic factors, angiotoxic drug effects or other undetected toxic causes should be explored before vascular surgery technical problems or deficiencies are considered as the primary etiologic factor motivating these different clinical settings: vascular thrombosis, massive renal thrombosis and delayed renal function in the living donor.

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