RUPTURE OF TRANSPLANTED KIDNEY

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According to the available literature rupture of transplanted kidney (RTK) is 3—8% of all cases with renal transplantation. This severe complication leads to a loss of the graft and possible death of the patients in 75% of them. The etiology and pathogenesis until now are not enough studied.

Out of all 666 operations for transplantation of cadaver kidneys from September 1974 till August 1981 we diagnosed 69 RTK in 65 patients (10.3%). This complication was most of all established in the first 2 weeks, between the 4-th and 9-th day after operation. RTK in 5 patients was established 20, 21, 22, 28 and 34 day after transplantation and it was connected with rejection crisis.

The frequency of RTK through the studied period was the following: 1974 — 6 RTK (15.8%), 1975 — 8 (9.5%), 1976 — 13 (9.3%), 1977 — 19 (22.1%), 1978 — 4 (3.6%), 1979 — 8 (8.7%), 1980 — 6 (7.0%), 1981 — 5 (15.6%). In the period between 1974 and 1977 was established bigger number of RTK — 46 (67%).

2 patients died in the first hour after rupture due to a heavy haemorrhage (3%). From 67 kidneys with ruptures, revised in the course of operation, 25 (38%) were operatively corrected and the rest 42 (62%) — taken away. The most important reason for the latter was the combination between acute canal necrosis in heavy form and graft rejection. Out of the left 25 kidneys later 13 were also taken away due to various reasons: non-functionating graft — 7, acute rejection — 3, secondary rupture at the peak of rejection crisis — 3. At the end of our investigation 12 operatively corrected kidneys (19%) functionated normally.

According to the character of the renal disorders the RTK were distributed as follows: superficial — 48%, profound — 33%, massive — 19%; according to the location RTK were: apex — 38%, renal edge — 36%, side surfaces — 19%, total kidney — 7%. The profound and massive ruptures were, as a rule, accompanied by a significant haemorrhage and haemorrhagic shock. According to the character of the haemorrhage the blood-loss was: up to 0.5 l — 47% of the patients, from 0.5 to 1.5 l — 42% and more than 1.5 l — 11%.

Early ruptures — 6—8 days after operation, were most of all as a result of an acute canal necrosis; late ruptures — of a rejection crisis. More profound and massive ruptures were the early ones, whereas the late ruptures were more superficial and dispersed.

According to our data the reasons for RTK are complex (polyetiological) but certain of them are leading: 1) Destruction and oedema of the kidneys due to their ischemia in the period before operation; 2) Mechanical factors due to unproper blood current in the organ and lymphostasis in the course of operation and the early periods after it; 3) Early rejection crisis. We include the following factors which can provoke RTK: 1) Heparinization of the patients

at the moment of setting to haemodyalisis or treating the rejection crisis (73% of all RTK); 2) Extrarenal mechanical factors, such as increased intravoluminal tension and increased hypertension in V. cava inferior (defecation, caughing, sudden stand up of the patient, etc. — 43% of the patients).

The clinic of ruptures coincides with the signs of internal peritoneal haemorrhage with various intensity. Haemorrhagic shock was established with 8 of the patients. The following symptoms have importance in the diagnosis: acute pain under ribs in the operative side (100%), swelling in the same region due to haemotoma (92%), tension of the abdominal wall and muscles in the illiac area (83%), palpatory pain of the rib edge (78%), lower blood haemoglobin (75%), lowered blood haematocrite (73%), tachycardia (72%), decreased arterial blood tension (67%), psycho-motor irritation (58%), lower diuresis (47%), intestinal paresis (32%), haematuria (18%) etc.

Analysing the question of RTK-mechanism, first of all we have to point out the factors of ischemia, being the leading pathogenetical moment. Three main reasons, separately and in combination between each other, can cause a serious renal oedema (stasis and inlammation oedema combined with lymphostasis). The damaged microcirculation in the kidney due to provoking factors, such as increased blood tension in V. cava inferior and the condition "block-oedema" in the kidney, make possible the considerable rise of the intrarenal tension in both, the blood vessel system and the system of intrarenal urinal tract. The present renal destructions due to ischemic damages causes certain disorders of the elasticity and stability of renal parenchyma to stretching. As a result of the serious renal oedema a rupture of the kidney is performed in the place where tissue structures are most of all affected. Most often RTK is located laterally and apically, thus indicating the most weak and unstable places of the organ. Another explanation is that by placing the kidney in the operative region under the ribs the aforementioned zones of the organ are less covered by the peritoneal membrane. Therefore, the intraperitoneal tension can not balance the intrarenal one, thus creating suitable conditions for a possible rupture in the weakest places of the transplanted graft. The same mechanism can be discussed with the so called intraoperative ruptures of kidneys in the course of revision of the grafts due to various reasons. When the kidney is liberated from its location in the abdominal cavity, the small "subcapsular" rupture in front of the eyes of the operator becomes a large and massive one.

РАЗРЫВ ТРАНСПЛАНТИРОВАННОЙ ПОЧКИ

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РЕЗЮМЕ

На опыте 666 операций трансплантации трупной почки с сентября 1974 по август 1981 г. авторы наблюдали 69 разрывов почек у 65 больных (10,3%). Из 67 почек, имевших разрывы и подвергшихся ревизии во время операции, 25 ушиты (38%) и 42 удалены (62%). Причиной удаления чаще всего было сочетание острого канальцевого некроза тяжелой формы с отторжением почки. Анализированы причины разрыва почки.