Letters

Increased platelet thromboxane release in focal segmental glomerulosclerosis

Sir.

It has remained an unanswered question as to whether minimal change nephrotic syndrome (MCNS) and focal segmental glomerulosclerosis (FSGS) are different entities or merely different manifestations of one disease. There are significant dissimilarities in the course of the disease and in the effectiveness of the treatment of MCNS and FSGS.

We recently reported our findings on the platelet function in children with different forms of nephrotic syndrome [1]. There were increases in platelet aggregation, thromboxane B₂ (TxB₂) release and ATP release and a decrease in the cAMP concentration in the platelets. We concluded that this increased platelet activation may play a role in the pathogenesis of nephrotic syndrome. Oral prednisolone and plasma lipids did not change the platelet function significantly. We found a moderately higher in vitro platelet aggregation in FSGS than in MCNS, but statistically it was not significant. Nevertheless, we did not compare these disease groups as concerns the platelet TxB₂ release and cAMP concentration, mainly because of the small number of FSGS patients. We have now repeated such examinations, focusing on a comparison of the platelet TxB₂ release and cAMP concentration in FSGS and MCNS with nephrotic syndrome and with early remission.

Sixteen patients (pts) with FSGS (eight adult and eight paediatric pts) and 27 children with MCNS (12 in relapse and 15 in remission) were investigated. At the time of the study, patients with clinical symptoms were within 3 months after kidney biopsy. Of the controls, 12 were adults (aged 31 ± 5.0 years) (Control adult) and 15 were children from the orthopaedic clinic (Control paed). None of the patients had renal or extrarenal thrombosis. The patients with MCNS in relapse or in FSGS had a urinary protein excretion level of >3 g/24 h and a plasma albumin concentration of 20 ± 3 g/l (x ± SD). All patients with MCNS in relapse received 60 mg/m² prednisolone, and those with FSGS received 2 mg/kg/day cyclophosphamide with 35 mg/m² prednisolone on alternate days. The patients with MCNS in remission had no proteinuria (<1 month), their plasma albumin concentration was 28 ± 2.5 g/l and they were on a tapering dose of prednisolone. None of the patients were given non-steroidal antiinflammatory drugs, albumin, or blood transfusion within the 2 weeks prior to the study. Neither the patients nor the controls received any medication known to affect the platelet function. The study was approved by the University Research Ethical Committee.

Platelet aggregation was studied by laser aggregometry, platelet thromboxane B₂ (TxB₂) levels by radioimmunoassay, and cyclic AMP (cAMP) levels by binding assay [1].

The platelet aggregation was significantly higher in all patient groups compared with the age-matched controls (P<0.01 and P<0.05). The platelet TxB₂ release was also significantly higher in all patients compared with the controls (FSGS: P<0.01, MCNS rel. and MCNS rem.: P<0.05). The correlation coefficient for the platelet TxB₂ release and platelet aggregation was 0.63 (P<0.05).

Nevertheless, the TxB₂ release was significantly higher in the FSGS than in the MCNS rem. or MCNS rel. pts (P<0.01). Platelet cAMP concentrations were significantly lower in all patient groups than in the controls (P<0.01). A significant negative correlation was observed between the platelet cAMP concentration and the platelet aggregation (r = −0.62, P<0.05) and between the platelet cAMP and the TxB₂ release (r = −0.60, P<0.05).

The increased platelet aggregability and TxB₂ release suggest that these abnormalities may be involved in the pathogenesis of both FSGS and MCNS. Platelet TxB₂ is significantly higher in FSGS than in MCNS, which may have an impact in the progression of glomerular damage by an increased vasoconstriction and intraglomerular pressure. Intraplatelet cAMP is a regulator of platelet activity by inhibiting Ca release from the cytosol [2]. An increased production of cAMP results in a diminished adhesion and aggregation ability in the platelets. In our FSGS and MCNS pts, the inhibitory effect of cAMP on platelet activation is decreased by a reduction of its platelet concentration.

Interestingly, the pts with MCNS in remission still had platelet disorders. Koyama et al. [3] reported a vascular permeability factor, a postulated lymphokine, which induces nephrotic syndrome by impairing the GBM anionic charges. This factor impairs not only GBM permeability, but also other cell membranes, as presumed by Levin et al. [4] and Boulton-Jones et al. [5]. If this factor impairs the platelet function and still remains functional shortly after the proteinuria remission obtained with corticosteroids, this could explain our findings.

Table 1. Platelet function in nephrotic syndrome

<table>
<thead>
<tr>
<th></th>
<th>FSGS (A + P)</th>
<th>MCNS Rel.</th>
<th>MCNS Rem.</th>
<th>Cont. A</th>
<th>Cont P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of pts</td>
<td>16</td>
<td>12</td>
<td>15</td>
<td>12</td>
<td>15</td>
</tr>
<tr>
<td>Platelet aggr.</td>
<td>28.7 ± 3810**</td>
<td>26.95 ± 3016**</td>
<td>25.53 ± 3165*</td>
<td>19.898 ± 3206</td>
<td>18.648 ± 3165</td>
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<tr>
<td>(± SD) AU</td>
<td></td>
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<tr>
<td>Platelet TxB₂</td>
<td>17.1 ± 2.2**</td>
<td>12.1 ± 3.4*</td>
<td>11.9 ± 2.9*</td>
<td>7.1 ± 1.6</td>
<td>6.9 ± 1.8</td>
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<td>(± SD) ng/ml</td>
<td></td>
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<tr>
<td>Platelet cAMP</td>
<td>14.1 ± 2.9**</td>
<td>12.1 ± 3.4**</td>
<td>11.7 ± 2.7**</td>
<td>26.8 ± 4.8</td>
<td>25.5 ± 2.1</td>
</tr>
<tr>
<td>(± SD) pmol/10⁶ plat.</td>
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</table>

A: adult, P: paediatric, FSGS: focal segmental glomerular sclerosis, MCNS: minimal change nephrotic syndrome, AU: Aggregation Units, TxB₂: thromboxane B₂, CAMP: cyclic AMP, *: P<0.05, **: P<0.01.
Failure of cyclosporine A in controlling Schoenlein-Henoch purpura

Sir,

The treatment of Schoenlein-Henoch purpura (SHP) is still under debate. It has been suggested [1,2] that pulse doses of i.v. methylprednisolone followed by oral steroids may be beneficial. Schmaldienst et al. [3] recently suggested that treatment with cyclosporine A (CyA) permitted steroid withdrawal and remission of severe nephrotic syndrome in a patient with SHP. We report on a patient with SHP who was treated with prednisone and CyA, but never became steroid-independent and eventually died due to massive reoccurrence of the acute vasculitic syndrome.

A 64-year-old man (90 kg, height 176 cm) was admitted due to acute renal failure and a purpuric rash. He was an ex-smoker and hypertensive. In 1993, he suffered a myocardial infarction and non-insulin dependent diabetes mellitus was diagnosed. After two more years, he had anaphylactic shock with macrohaematuria and purpura in the lower limbs secondary to celifraxia. A skin biopsy was suggestive of leucocytoclastic purpura. Two years later, he presented with right lower lobe pneumonia, which was successfully treated with i.v. ceftazidime. However, he developed a purpuric rash involving the lower extremities and within 2 weeks, serum creatinine increased from 1.5 to 8.6 mg/dl (132–757 μmol/l). He became oliguric and was then transferred to the renal unit on 22 April 1997. The erythrocyte sedimentation rate (ESR) was 121, C reactive protein (CRP) 56 mg/l (normal reference values <5 mg/l), serum IgA 850 mg/dl (normal reference values <300). ANCA were negative. Urinalysis showed >100 rbc/ml and proteinuria 100 mg/dl. Renal histopathology was suggestive of SHP with mesangial proliferation and IgA deposits in the mesangium, but without crescent formation. The patient was treated with three boluses of 1 g methylprednisolone (MP) daily for 3 days followed by prednisone 100 mg daily. The CyA dose was reduced to 75 mg b.d. in order to maintain a trough CyA level between 100 and 150 μg/l. He was afebrile and no macrohaematuria was present. However, control of the diabetes was difficult and the patient complained of marked astenia. Again we reduced the prednisone dose to 90 and then 80 mg daily. On 17 July 1997, he was re-admitted with oliguria and a new purpuric rash on the lower extremities.

4. Levin M, Walters MDS, Smith C, Gascoine P, Barratt TM. Oliguria and a new purpuric rash appeared to be associated with reacutization of SHP and the treatment with CyA, each reduction of the prednisone dose associated with a decrease in proteinuria. In spite of chronic shock with macrohaematuria and purpura in the lower limbs, the CyA permitted steroid withdrawal and remission of severe nephrotic syndrome in a patient with SHP. This patient had two relapses while on CyA treatment when the prednisone dose was reduced to ~75 mg/day and, therefore, the CyA did not have a steroid sparing effect. Based on the experience of our case, the favourable development reported by Schmaldienst et al. [3], may have been a spontaneous remission rather than secondary to CyA therapy. However, the CyA treatment may have had a positive effect on the proteinuria. In any case, the role of CyA treatment in vasculitides is still under debate [4,5] and a controlled trial is necessary to determine whether CyA is beneficial in SHP.
Percutaneous drainage by multiple and bilateral puncture of infected renal cysts in autosomal dominant polycystic kidney disease

Sir.
ADPKD is a condition which culminates in chronic renal failure. The cystic infection is relatively frequent and represents a difficult diagnostic and therapeutic problem [1]. In some instances, when there is no response to antibiotic treatment, an aggressive therapy such as a nephrectomy may be mandatory. This last option shortens the period without dialysis. A few cases of percutaneous drainage of solitary infected cysts have been published [2,3] and so far none concerning multiple and bilateral puncture.

We report here the case of a 54-year-old woman with an ADPKD and a history of chronic sinusitis. After the menopause, aged 53 years, she developed three lower urinary tract infections that were treated with antibiotics, without complications.

She developed lower urinary tract symptoms and fever. She was treated with cotrimoxazole by her general practitioner. Four days later, she was admitted to our hospital because of persistence of fever and bilateral lumbar pain. The urinalysis showed Gram negative bacilli and the urine culture yielded a multisensitive E. coli, blood urea nitrogen 45.7 mg/dl and creatinine 3.6 mg/dl. Oral ciprofloxacin was started. An echography showed complicated cysts in both kidneys (Figures 1 and 2). While there was no clinical improvement in spite of the antibiotic treatment, bilateral

Fig. 1. Echography (A) and drawing (B) of the right kidney: large cyst with echogenic contents.

Fig. 2. Echography (A) and drawing (B) of the left kidney: two cysts (A and B) with hyperechoic and heterogeneous contents.
and multiple renal cyst percutaneous drainage was undertaken: two 9 Fr. catheters in the left and one in the right kidney (Figures 3 and 4). The culture of purulent material from the cysts was positive to *E. coli*. Afterwards we began treatment with i.v. cefotaxime and aztreonam. The patient improved and the fever disappeared so that the catheters, with negative culture, were withdrawn after 10 days. But on the subsequent day, the fever reappeared and 4 days later we placed a bilateral 9 Fr. percutaneous drainage again and the antibiotics meropenem and vancomycin were i.v. administered, a few days later we added chloramphenicol. No irrigation of the cysts was undertaken. Forty-eight hours later the patient became afebrile and the echography showed evident shrinkage of the cysts. There was an improvement of the creatinine to 2.8 mg/dl and blood urea nitrogen to 36.1 mg/dl. The catheters were withdrawn once the patient became afebrile and the drainage sterile, and the antibiotic treatment was maintained for 1 month with cefixime p.o. The urine culture was negative. The patient is at present treated with intravaginal cream of 0.5 mg estriol and has been free of infection for 9 months.

The prevalence of infection in ADPKD is abnormally high [1], and more common in women [1,3]. The hormonal changes induced in the vaginal flora have an important role in the development of infections of the urinary tract. In premenopausal women, the estrogens encourage colonization of the vagina by Lactobacilli which maintain a low vaginal pH that inhibits the growth of uropathogens. However, with menopause the vaginal pH increases, the Lactobacilli disappear from the vaginal flora, and the introitus and the vagina are colonized by Enterobacteriaceae, especially *E. coli* [4]. The main mechanism of infection is retrograde migration, and if there are structurally distorted kidneys, both conditions predispose to cyst infection in these women.

The first problem in the management of these patients is to differentiate between parenchymal and cyst infection. Echography and CT are not always diagnostic. But in some cases, such as the one described above, the presence of debris and echogenic material give valuable pointers to the diagnosis. According to the reports on Gallium-67 scanning this technique confirms the diagnosis of renal infection in 50% of patients [1,5].

The response to antibiotic therapy in the infected renal cyst is inversely related to its size and to the penetration of the antibiotic into the cyst [3]. Bennett *et al.* [6] studied the penetration of antibiotics in the cysts and observed that ampicillin and cotrimoxazole had the best profiles.

According to Wetzel *et al.* [2] antibiotics penetrate with difficulty in the cysts. The best levels in the cyst fluid are obtained with cotrimoxazole, cloramphenicol, ciprofloxacin, and norfloxacin. The authors propose cefotaxime in renal failure. Schwab *et al.* [1] recommend to begin the treatment with ampicillin and aminoglycosides in all APDK patients with kidney infection. If they don’t respond after 5 days, the authors propose to use a lipo-soluble drug, e.g. chloramphenicol or trimethoprim-sulfamethoxazole.

We think that the antibiotic treatment should begin with cotrimoxazole or quinolones, i.e. drugs with low toxicity and
5.


6. Bennet WM, Elzinga L, Pulliam JP et al. Cyst Fluid antibiotic Ninety-three patients received RRT in the 7 years between January 1, 1992 and December 31, 1993. Data were extracted for all 101 patients (M: F ratio 45:36) who had a serum creatinine \( \geq 300 \, \text{mol/l} \) between January 1, 1992 and December 31, 1993.

**Chronic renal failure and end-stage renal disease in St Petersburg, Russia**

Sir,

The Russian health care system, once viewed as a model of universal access to health care, is currently facing the same considerable problems as many other Russian institutions. Mortality rates for the Commonwealth of Independent States (CIS) have increased (from 739 to 1216 per 100,000 between 1960 and 1992) and infant mortality has remained static since 1970 whereas it has continued to fall in Western countries [1]. There is relatively little data available about health care in general in the former Soviet Union, and even less concerning renal failure [2].

The Commission of the European Community initiated the PECO-1993 scheme to support studies which promoted research collaboration between EC countries and the ex-communist states. One such project, supported by the BIOMED-1 programme, was entitled ‘End stage renal disease: approaching a consensus’ and involved five EC countries (France, Germany, Holland, Greece, and Scotland) and Albania. PECO-94 extends this international collaboration to St Petersburg (Russia).

We report the incidence and prevalence of chronic renal failure and survival on RRT in a single centre in St Petersburg which serves a population of 1 million. The Pavlov Institute is one of five nephrology centres serving the five million people of St Petersburg. The eight nephrologists who work in the clinic also practice general medicine whereas the 13 dialysis unit nephrologists perform only dialysis. Each year 10 nephrologists from all over Russia receive training at the clinic. The four other hospitals in St Petersburg employ a total of 22 nephrologists to provide dialysis to 186 patients. In addition there are eight nephrologists specifically for general nephrology and the transplant unit is staffed by six surgeons and four nephrologists. In total in St Petersburg there are currently \( \sim 350 \) patients on haemodialysis and 30 patients on CAPD and on average 50 patients receive renal transplant each year. Records of patients attending the nephrology clinic were studied to identify those with a serum creatinine \( \geq 300 \, \text{mol/l} \), first recorded between January 1, 1992 and December 31, 1993. Demographic details (age, sex, date of birth) were noted as were the renal diagnosis, co-morbidity, and where appropriate, cause and date of death. In addition data were recorded for all patients receiving RRT at the Pavlov Institute between January 1, 1985 and December 31, 1991. In both cases the data were recorded on questionnaires completed by nephrologists in St Petersburg. The questionnaires were then sent to Aberdeen and analysed using the SPSS statistical package. Using previously described methods patients were stratified into low, medium, and high risk groups [3] and survival was estimated using the Kaplan Meier method.

Data were extracted for all 101 patients (M: F ratio 45:36) who had a serum creatinine \( \geq 300 \, \text{mol/l} \) during a 2-year period. Seventy-nine had their first serum creatinine \( \geq 300 \, \text{mol/l} \) between January 1, 1992 and December 31, 1993. Of these 79 patients, 31 (39.2%) were in the low risk group, 41 (51.9%) in the medium, and seven (8.9%) in the high risk group. The incidence of chronic renal failure was therefore found to be 39.5 per million population per year. Ninety-three patients received RRT in the 7 years between 1985 and 1991 (M: F ratio 58:34). Primary renal diagnoses and co-morbidity were determined as for the CRF patients. Sixty-six (71.0%) of patients were in the low risk group, 22 (23.7%) in the medium risk group, and five (5.3%) in the high risk group. Two-year survival was 87.8% (91.9% in the low risk group, 85.2% in the medium and 60.0% in the high risk group). Very few patients (only five out of 90) belonged to the high risk group and the majority (71%) were in the low risk group.

The study showed that referral rates for chronic renal failure in St Petersburg are low compared with those in Western centres. Mostly low risk group patients were offered dialysis in St Petersburg, and had survival rates comparable with those seen in similar patients in Western European centres.

Our study is limited by the fact that it is difficult to define the catchment area served by the Pavlov Institute with certainty as the city of St Petersburg is served by other renal units. We therefore plan to undertake a study of the prevalence of renal disease in general and chronic renal failure in particular in North-West Russia (population 20 million).

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Recurrent Kaposi’s sarcoma in a renal transplant recipient maintained on minimum doses of immunosuppression

Sir,
Kaposi’s sarcoma (KS) is generally a rare tumour and in its classic form affects elderly men from Eastern Europe and the Mediterranean region [1]. Following renal transplantation the frequency of KS is 200–400 fold greater than in the general population [2,3]. On cyclosporine therapy KS is more frequent than azathioprine and prednisone [4]. Reduction or withdrawal of immunosuppression results often in complete regression of KS [5,6]. Local radiotherapy after reduction of immunosuppressive drugs is also effective [7,8]. After re-introduction of cyclosporine or high dose corticosteroids there is a high risk of recurrence in patients with a history of KS [9]. We observed a renal transplant recipient who had three episodes of recurrent KS over 12 years in spite of minimum doses of azathioprine and prednisone.

A 55-year-old man had a living related kidney transplant in December 1984 at the age of 41 years. The transplantation course was smooth and he was maintained on cyclosporine and prednisone. Three months after transplantation he noticed a skin nodule on the left forearm which was followed by similar lesions on the legs and the tip of the nose. Biopsy of the left forearm nodule was diagnostic of KS. Cyclosporine was withdrawn, azathioprine started at the dose of 100 mg/day and prednisone was reduced from 30 to 20 mg/day. The KS lesions on the nose were irradiated and the other skin lesions excised. Evaluation for visceral involvement of KS including endoscopy and CT of thorax and abdomen were negative. Five years after transplant he developed nodular skin lesions on the distal part of the right leg and biopsy of the lesions confirmed KS. Azathioprine was reduced from 100 mg to 50 mg/day and the lesions were irradiated. There was no evidence of KS involving other sites or internal organs and the HIV test was negative. The lesions disappeared within 3 months but the graft function deteriorated significantly (Figure 1).

After > 10 years from the first KS and ~6 years after the second, nodular KS lesions appeared on the distal part of his left leg. HIV serology was negative and KS was not detected in internal organs. Azathioprine was reduced from 50 mg to 25 mg/day and the lesions were treated by irradiation.

Twelve years after transplant he developed nodular KS lesions on the distal medial side of the right leg (the same site of previously healed KS). He underwent bronchoscopy and endoscopy which did not show KS. HIV test was negative. The KS lesions disappeared after irradiation.

At present he is under follow-up for more than 9 months since the last recurrence of KS, on 25 mg of azathioprine and 10 mg of prednisone/day, without symptoms or signs of KS and stable graft function with a serum creatinine of ~300 μmol/l. KSHV antibodies were positive in the serum of the patient. A tissue sample from the KS lesions demonstrated the presence of KSHV DNA in the lesion [10].

Post-renal transplant KS usually manifests as lesions on the skin and oropharynx but mucovisceral involvement is also common. Following treatment, cutaneous lesions usually undergo complete remission [5,6]. Our patient was on cyclosporine, had KS in the fourth month after transplantation, and went into complete remission after withdrawal of cyclosporine. After 4 years of remission, KS skin lesions recurred.

Fig. 1. Time course of recurrent KS in a renal transplant recipient. CSA, cyclosporine; KS, Kaposi’s sarcoma; ---, azathioprine; --, prednisone.
Sterile leukocyturia as a manifestation of urinary tuberculosis in renal transplant patients

Tuberculosis is a relatively infrequent disease in renal transplant patients [1]. Very few cases of urinary tuberculosis have been described. The infection may produce granulomatous interstitial nephritis in the graft whose diagnosis generally requires the performance of a graft biopsy [2-5]. Others cases have been published in which the urinary manifestations and diagnostic methods are poorly described. In most of them, however, the diagnostic suspicion has not been aroused by the clinical features that characterizes non-immunosuppressed patients, and consequently the final diagnosis has not been made by the usual bacteriological methods.

We describe three renal transplant patients with urinary tuberculosis, in which the disease was suspected by the appearance of sterile leukocyturia and whose diagnosis was made by the detection of acid-fast bacilli in urine samples.

Patient 1. A 42-year-old male with membranoproliferative glomerulonephritis received a renal transplant from a 24-year-old donor in September 1988. In March 1990 the patient complained of dysuria. Urinalysis revealed innumerable leukocytes per high power field. Urine culture for usual bacterial pathogens was negative. A Zielh–Neelsen stain was made and abundant acid-fast bacilli were observed on five consecutive days. Bacilli grew in the five samples cultured in Lowenstein medium. Treatment with isoniazid, ethambutol, and rifampicin was begun. Dysuria and leukocyturia rapidly disappeared, and acid-fast bacilli turned negative. Ethambutol was suspended after 2 months, rifampicin after 9 months, and isoniazid after 24 months. The patient is now completely asymptomatic, urinary sediment is normal and acid-fast bacilli examinations are repeatedly negative.

Patient 2. A 42-year-old female with lupus nephritis received a renal transplant from a 47-year-old male donor. From January 1991, she presented asymptomatic sterile leukocyturia. In July 1991, the urinalysis with Zielh–Neelsen staining in three samples on three consecutive days showed numerous acid-fast bacilli. The patient was treated with ethambutol, isoniazid, and rifampicin, the leucocyturia disappeared and acid-fast bacilli were negative. Ethambutol was suspended after 4 months, rifampicin after 9 months, and isoniazid after 20 months. The patient is currently asymptomatic with normal urine sediment and negative acid-fast bacilli.

Patient 3. A 70-year-old female with renal failure of unknown origin received a renal transplant from a 61-year-old male in December 1993. In December 1996, she was admitted with pyrexia and deterioration of renal function. Mycobacterium tuberculosis grew in urine culture. A mictorial cystography showed reflux of the graft and marked changes in the calyces (Figure 1). The patient was treated with isoniazid, ethambutol, and rifampicin. In spite of the anti-biogram showing sensitivity of Mycobacterium tuberculosis to drugs used, pyrexia and pyuria continued and renal function worsened until haemodialysis became necessary. Treatment was changed to pyrazinamide, isoniazid, and streptomycin, but urinalysis continued to show acid-fast bacilli. The patient refused removal of the graft and died in April 1997.

Isolation of Mycobacterium tuberculosis in urine may be due to an infection localized in the graft and/or in the original native kidneys. Very few cases of infected grafts have been documented. In 1982, Walker et al. [2] described a patient with pulmonary and graft involvement demonstrated by bacteriological and radiological methods. Later,
renal dysfunction and cystography changes compatible with those described by Walker et al. The normal treatment involves the use of three drugs, maintaining two of them (rifampicin and isoniazid) during 9 months. This regime proved useful in patients 1 and 2, but patient 3 kept shedding bacilli in urine during 4 months in spite of the fact that the germ demonstrated in vitro sensitivity to the drugs administered. Her death may well be due to the continued persistence of graft pathogens, as has been previously suggested in others works [6]. Transplantectomy should probably be indicated in these cases unresponsive to medical therapy in order to avoid the loss of lives.

The diagnosis was achieved in our patients by bacteriological methods, in contrast to that described by other authors which in \(\sim 60\%\) of the cases needed aggressive diagnostic manoeuvre [7]. Alterations of the urinary sediment in those specimens whose concomitant conventional culture is negative should immediate search of acid-fast bacilli in urine, both by staining an culture methods. Implementation of early therapy is the only way of avoiding the risk of graft loss.

Al-Suliman et al. [3] described four cases of interstitial nephritis of the graft whose diagnosis was made by biopsy, but not bacteriologically confirmed. Gonçalves et al. [4] also described three cases of interstitial nephritis diagnosed by graft biopsy and in one case acid-fast bacilli were shown in urine. More recently, Adeva-Andany et al. [5] have described a similar case with bacteriological confirmation. In seven of nine cases the clinical course was progressive, finally producing the lost of the graft. Only one of our three patients had renal dysfunction and cystography changes compatible with those described by Walker et al. The normal treatment involves the use of three drugs, maintaining two of them (rifampicin and isoniazid) during 9 months. This regime proved useful in patients 1 and 2, but patient 3 kept shedding bacilli in urine during 4 months in spite of the fact that the germ demonstrated in vitro sensitivity to the drugs administered. Her death may well be due to the continued persistence of graft pathogens, as has been previously suggested in others works [6]. Transplantectomy should probably be indicated in these cases unresponsive to medical therapy in order to avoid the loss of lives.

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Fig. 1. Micturating cystogram of patient 3 demonstrating reflux to the transplant and multiple calyceal alterations.

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