Results: Immunoreactivity for PTTG was cytoplasmic and nuclear. All small cell carcinomas (3+, 71%; 2+, 29%), the majority of squamous (3+, 26%; 2+, 41%) and almost half of adenocarcinomas (3+, 20%; 2+, 25%) were either 2+ or 3+ immunoreactive. Intensity was moderate to strong in nearly all of squamous (3, 26%, 2, 63%) and small cell carcinomas (3, 29%; 2, 64%) and only one-third of AC (3, 7%; 2, 32%). Fifteen of the patients with AC had survival less than 5 years, with a mean survival of 27 months. Kaplan-Meier curve analysis revealed that AC staining intensity negatively correlated with survival (Chi-square= 13.3114, p = 0.004).
Conclusion: PTTG is differentially expressed in primary lung carcinomas. The level of expression may relate to the malignant potential of these tumors. Intensity of PTTG negatively correlated with survival in patients with AC. Based on our data, PTTG may be a potential prognostic marker in patients with pulmonary AC.

Quality Assurance

810 INTEREST OF EVALUATING KI-67 PROLIFERATION INDEX BY COUNTING 1,000 TUMOR CELLS IN A SERIES OF 42 DIGESTIVE ENDOCRINE TUMORS

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Background: Ki-67 proliferation index (PI) is known to have prognostic impact in digestive endocrine tumors; it is one of the criteria appearing in the current WHO classification of endocrine tumors. However, standard assessment of this index remains to be defined. Our aim was to compare the PI evaluated by counting 1,000 tumor cells (1,000PI) to the initial PI evaluated in our daily practice, in a series of 42 digestive endocrine tumors surgically resected.
Design: Forty-two endocrine tumors (27 of the GI tract, 15 of the pancreas) have been surgically resected in our institution between 1998 and 2005, corresponding to 7 well-differentiated endocrine tumors (WDET) (2 benign, 5 with uncertain behavior), 34 well-differentiated endocrine carcinomas (WDEC), 1 poorly-differentiated endocrine carcinoma (PDEC). Ki-67 (Mib-1) slides have been reviewed by one observer, who evaluated PI by counting 1,000 tumor cell nuclei in the most proliferative area. Kappa coefficients were calculated to measure the agreement between this PI and the initial PI value reported in the pathology report, regarding to the classes appearing in the WHO classification (GI tract: <or = 2 (WDET), 2 < p <or= 15 (WDEC), >15 (PDEC); pancreas: <or = 2 (benign WDET), 2 < p <or = 5 (WDET with uncertain behavior), 5 < p <or = 15 (WDEC), >15 (PDEC)). We also focused on whether 1,000PI was closer to the values suggested in the WHO classification for each diagnosis than the initial PI value.
Results: The kappa value between the two PI values was moderate in GI tract tumors (0.55+-0.17), and in pancreatic tumors (0.43+-0.16). In pancreatic tumors, in case of discordance, 1,000PI was always lower than the initial PI value; 1,000PI was not better correlated to the diagnosis classes of the WHO classification than the initial PI.
Conclusion: Specially in endocrine tumors of the pancreas, there is a need for standard assessment of PI. However, we failed to demonstrate in this study than evaluating PI by counting 1,000 tumor cells improves its correlation with the diagnosis classes of the WHO classification.

Renal (Medical)

811 CHRONIC ALLOGRAFT NEPHROPATHY IN RENAL TRANSPLANTED PATIENTS EXPERIENCING ACUTE REJECTION EPISODES

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Background: Chronic Allograft Nephropathy (CAN) is the most frequent cause of late graft failure in transplanted patients. The occurrence of acute rejections is a risk factor for development of CAN. The severity of histological lesions in acute rejection biopsies has been proposed to play a role in CAN development. Aim of this study was to evaluate clinical and histological parameters that may be relevant for the development of CAN in patients with biopsy proven episodes of acute rejection.
Design: We examined 216 renal transplanted patients, time interval 1988-2000, mean follow up: 203.5 +/- 42.5 months. Immunosuppression was based on cyclosporine in all cases. Of these 216 patients 137 did not show evidence of acute rejection (group 1) whereas 79 experienced one or more biopsy proven acute rejection episodes, classified for type and grade according to the Banff ` 97 classification (group 2). We compared in the two groups creatinine levels at different time intervals and the occurrence of CAN, evaluated according to the Banff ` 97 classification. We then analysed within group 2 clinical and histological parameters that could significantly correlate with CAN.
Results: 216 renal transplanted patients were examined, 43 developed CAN and their graft survival (108.9 months, 95% C.I 87.5-130.3) was significantly lower than that of patients without CAN (154.2 months, 95% C.I. 142.1-166.4) (p<0.003). In group 1 mean serum creatinine levels evaluated six months, 2 and 5 years after transplantation were lower than in group 2 (p< 0.001). In group 2 the number of patients that developed CAN (27/79 = 34.2%) was higher than in group 1 (16/137 =11.7%) (p< 0.0001) and graft loss due to CAN was also higher (9/137 = 6,6% in group 1 vs 13/79 = 16.5% in group 2, p< 0.02), while no significant difference was observed in graft loss due to death among the two groups (24/137 = 17% in group 1 vs 9/79 = 11.4% in group 2). Interestingly CAN occurred later in group 1 (168.6 months, 95%C.I. 156.6-180.4) than in group 2 (116.7 months, 95% C.I. 101.6-131.9) (p< 0.0002). In group 2, considering the histologically more severe acute rejection episode per patient, 18 patients had a borderline rejection (1-4 mononuclear cells/tubular cross section), 30 had type IA (5-10 mononuclear cells/tubular cross section), and 29 had type IB rejections (> 10 mononuclear cells/tubular cross section). Only 2 had type II rejection and were not considered. The severity of rejection correlated with development of CAN (p< 0.02). Interestingly, it did not correlate with the mean % increase of serum creatinine at the time of the biopsy. Among patients of group 2, HLA A, B, and DR matching was not significantly different. A late timing of rejection was significantly associated with CAN (p< 0.01), while age, male/female ratio, living/cadaveric donor ratio, cold ischemia time, occurrence of post-transplant tubular necrosis and mean % increase of serum creatinine at the time of the biopsy were not.
Conclusion: In patients with acute rejections CAN develops earlier and the degree of tubulitis correlate with CAN development.

812 HIV RENAL DISEASE AT TYGERBERG HOSPITAL, WESTERN CAPE, SOUTH AFRICA

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Background: Despite the high incidence of HIV/AIDS in sub-Saharan Africa, there are as yet few well documented reports of HIV-related renal disease from Africa. HIV associated nephropathy (HIVAN) is the most common chronic kidney disease found in these patients in other parts of the world.
Design: From 1992-2006 (February), 86 HIV positive patients underwent 89 biopsies. Demographic, clinical and pathological data was collected and correlated.
Results: The patients were divided into 2 groups based on biopsy morphology. Forty seven (55%) showed HIVAN. This group comprised 24 females and 23 males with 33 black (African) and 14 coloured (mixed race). Mean age on presentation was 33 years and mean serum creatinine 708 ìmol/L. The biopsies showed segmental sclerosis/collapse of glomeruli as well as tubular necrosis, dilated tubules and prominent interstitial inflammation. Immunofluorescent positivity was frequent for IgM 30/37(81%) and C3 22/37 (59%) as were tubuloreticular bodies, seen in 31/47 (66%). The 2nd group of 39 non-HIVAN biopsies showed a spectrum of pathology including mesangial proliferative glomerulonephritis (GN) (10), postinfectious GN (7) (one also crescentic on rebiopsy), mesangiocapillary GN (6), membranous GN (4), diabetic nephropathy (2), IgA nephropathy (1), crescentic (1), myeloma cast nephropathy (1), amyloid (1), tubulointerstitial (2), tubular necrosis (2) and end stage changes (2). A few unusual patients deserve a special mention: Two of the cohort showed lymphoproliferative disease. A 41 year old female who presented with a creatinine of 53 ìmol/L and a 24 hour protein of 40 grams. She was found on biopsy to have myeloma cast nephropathy as a result of kappa light chain myeloma. She also showed AL amyloidosis in glomeruli and vessels. A 48 year old patient with atypical Burkitt`s lymphoma showed mesangial proliferative GN. A 31 year old male with HIVAN was rebiopsied after 6 weeks on anti-retroviral (ARVs) and was found to have developed early membranous deposits on ultrastructure. The possible connection between the two remains uncertain. A 32 year old female with HIVAN also showed granulomatous inflammation and acid fast bacilli with Ziehl Neelsen stain in the renal biopsy. The only white patient in this HIV positive cohort, showed an IgA nephropathy. A small number of our most recent patients have been treated on ARVs but the effects are not yet clear.
Conclusion: Renal disease in HIV positive patients is being increasingly documented in our centre. HIVAN is the most frequent form and has similar features as described elsewhere. Though frequent in our black (African) population, both male and female, HIVAN has now also been documented in coloured (mixed race) patients in South Africa. This small cohort almost certainly represents just a fraction of a huge pool of South African HIV positive patients with renal disease.

813 TUBULAR DYSPLASIA IN CONTRALATERAL KIDNEY IN PAPILLARY RENAL CELL CARCINOMA

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Background: The premalignant lesions of renal cell carcinoma (RCC) have not been fully described. The findings of intratubular epithelial dysplasia (IED) in areas around the tumor, free of neoplasia, suggests that this could be a biological precursor of some RCC. We describe for the first time the identification of IED in contralateral kidney (left) of a patient with papillary renal cell carcinoma in right kidney.
Design: A 35 y.o. white male while investigation of nephrotic syndrome, had the identification by TC of a renal mass in right kidney. After partial right nephrectomy a diagnosis of papillary renal cell carcinoma was made in december 2005. As he continued the investigation of nephrotic syndrome a left kidney biopsy was performed in january 2006.
Results: The kidney biopsy showed Focal Segmental Glomerulosclerosis and intratubular epithelium dysplasia in the absence of renal carcinoma.
Conclusion: Dysplasia of tubular epithelium is probably a biological precursor of some RCC. The findings of dysplasia in a contralateral kideny free of neoplasia warrants further study as an important finding that will provide new insights into the pathogenesis, biological behavior, and natural history of RCC.

814 RENAL DAMAGE CAUSED BY ESSENTIAL HYPERTENSION: STUDY BY DETERMINATION OF RENAL INTERSTITIAL FIBROSIS

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Background: The main histological lesions caused by Systemic Arterial Hypertension (SAH) occur in the target organs, the kidney being one of them. Renal hypertensive nephrosclerosis is the 2nd main cause of the end stage kidney disease (ESRD). Malignant nephrosclerosis (MN) is a known cause of ESRD. However, diagnosis of benign

nephrosclerosis (BN), caused by Essential SAH, as an isolated etiology of ESRD is still controversial. This is curious seeing that there is a predominance of benign nephrosclerosis associated with ESRD whereas the cases of malignant nephrosclerosis are more and more rare. Benign nephrosclerosis is caused not only by the ischemic lesion, but also by the glomerular sclerosis, synthesis of inflammatory factors as TGF-ß and hyperactivation of the systemic renin-angiotensin system, and has long been recognized as a poor prognostic feature in human renal disease. Our aim is to quantify the interstitial renal fibrosis from hypertensive autopsied patients and correlate this with the severity of clinical hypertension.

Design: From the autopsy records of the General Pathology Division (between 1985 and 2004) were selected patients with characteristic autopsies findings of SAH, as hypertensive cardiopathy, and gross features of BN or MN. Excluded were those patients with underlying renal parenchymal disease due to other diseases. Light microscopic images from the medium pole of the right kidney of Picrossirius-red stained sections were taken by a color video camera, connected to a light microscope at an original magnification of 40x. Images were processed with the KS-300 (Carls Zeiss) program. Five fields of each slide were analyzed under polarized light microscopy. The amount of fibrosis was expressed in percentage/field.

Results: From the 41 cases that were selected, the average age was 59 years, with mle predominance. All of the patients had hypertensive cardiopathy and BN. No cases of MN were identified. Eleven patients presented with severe hypertensive disease (SHD), with severe left ventricle hypertrophy and parenchymal cerebral hemorrhage. In 17 cases (41,4%) the average interstitial fibrosis was over 20%, with renal lesions characterized as moderated to intense. The percentage of interstitial fibrosis had a negative correlation with the amount of renal parenchyma. The fibrosis was significantly higher in the patients who died due to cardiovascular causes. In those with SHD the average interstitial fibrosis was 21,2% compared to 16,95% from those patients without SHD ($p < 0,001$).

Conclusions: Interstitial fibrosis is an important renal parenchymal lesions in benign nephrosclerosis. The percentage of fibrosis showed a significant correlation with a decrease of the renal parenchyma, the gravity of the SAH and could be used as a predictor of major cardiovascular disease. As such, it appears to be an excellent marker of the impact of the hypertension disease on the patient`s cardiovascular and renal system. The "benign" term for essential hypertension should be considered a misnomer as we have showed that the chronic renal lesion is a sign of severity and poor prognosis.

815 TWO CASES OF DIFFUSE MESANGIAL SCLEROSIS WITH DIFFERENT CLINICAL PRESENTATION

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Background: Diffuse Mesangial Sclerosis (DMS) is one of the main congenital causes of infantile nephrotic syndrome. The DMS present as a isolated syndrome or as a feature of Denys Drash Syndrome (DDS), the triad of congenital nephrotic syndrome leading to end-stage renal failure (ESRF), XY pseudohermaphroditism and Wilms' tumor caused by mutations in the chromosome 11p13, WT1gene.

Design: Case reports of diffuse mesangial sclerosis (DMS) with early and late onset end-stage renal failure.

Case Report 1: A 13 year old male was referred to a Pediatric Hospital for evaluation of nocturnal enuresis, bilateral cryptorchidism and a history of nephrectomy for Wilms' tumor at age 2. Laboratory findings showed total proteinuria 4.5g/dL and serum creatinine, 1.5 mg/dL. During hospitalization total serum protein was always elevated (between 4.5 and 5.4 g/dL/24h) and renal function exams were borderline. The laboratory and clinical exams worsened with a serum creatinine, 2.1 mg/dL and blood pressure 150x110 mmHg. Renal biopsy (light, electronic and immunofluorecence microscopies) showed diffuse mesangial sclerosis. The hypertension was managed by nifedipine and captopril. Three months later the patient's renal function worsened and the patient it was initiated on peritoneal dialysis. After initial recuperation he was transferred to his hometown, for continuing program of dialysis.

Case report 2: A 5-month-old girl was referred for evaluation of diarrhea with oligúria, anasarca and increased blood pressure with partial response to furosemide and nifedipine. She was born at 38 weeks' gestational age with a birth without intercurrences. She had progressive loss of the kidney function (serum creatinine= 1.2 mg/dL and urea = 145md/dL) and a renal biopsy was performed. Light microscopy showed diffuse mesangial sclerosis. Her external genitalia were normal female and no other malformations were identified.

Conclusion: These two patients showed isolated diffuse mesangial sclerosis without typical genital abnormalities. The first patient in question presented alterations of Denys Drash with commemorative of WAGR (Wilms' Tumor, Congenital cataract and bilateral cryptorchidism). The most important fact was the late evolution of the DMS in this patient (13 years). This differs of the literature in that cases of end-stage renal disease usually occur around 3 years of age. The second patient had only DMS, but was very young at diagnosis. Mutations in the gene WT1 caused severe and frequent malformations resulting in the WAGR Syndrome (Wilms' Tumor, Aniridia, Genitals Abnormalities and Mental Retardation). The account of these two cases suggests that diffuse mesangial sclerosis could be a common manifestation in the broad spectrum of alterations diagnostic of the WARG syndrome. The diagnosis of isolated DMS alerts the pathologist and the clinician to possible associations with the manifestations of the WARG syndromes.

816 RENAL BIOPSY IN DIABETIC PATIENTS: LOCAL EXPERIENCE AND REVIEW OF THE LITERATURE

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Background: The increased incidence of diabetes mellitus (DM) has been described as a `pandemic`: an estimated 3-5% of the US population is currently estimated to have DM and it has been estimated that, world-wide, over 300 million persons will be affected with DM by 2010. Renal biopsy (RB) is usually indicated for patients with DM only when atypical clinical or laboratory features are present (such as rapid onset of severe proteinuria, acute renal failure, or hematuria). Recent atypical findings in some RB from patients with DM prompted a review of our recent experience and the relevant literature.

Design: Native RB performed in patients with DM at our institution between 1999-2005 were identified in the Anatomic Pathology LIS and these results were reviewed. A literature search (PubMed) was performed to identify publications describing results of RB in patients with DM and these reports were reviewed.

Results: Local Experience: Forty-seven (47) RB in patients with DM were identified. Twenty-three (23/47, 49%) showed only the usual features of diabetic nephropathy (DN). Seventeen (17/47,36%) showed a wide-range of diseases other than DM with membranous glomerulopathy being the most common. The remaining diagnoses were diverse and included acute interstitial nephritis, acute tubular necrosis, amyloid, lupus nephritis, and myeloma. Seven (7/47, 15%) contained DN with unusual features (such as crescent formation) or co-existence of DN and another condition (including IgA nephropathy and acute interstitial nephritis). Literature Review: Results from the many studies of RB findings in patients with DM demonstrate a wide range in the incidence of non-diabetic renal disease either alone or in combination with DN. Overall, DN alone is present in 50-85% of biopsies. Findings other than DN or DN in combination with other diseases are reported in from 25-66% of patients with most series reporting approximately 33% of RB to contain findings other than DN.

Conclusion: The number of renal biopsies in patients with DM is likely to increase due to the increased prevalence of DM. Our recent experience and review of the literature suggests that findings such as DN with unusual features, DN co-existent with other conditions, and conditions not directly related to DN are common and may therefore represent an increasing proportion of a RB practice.

817 EVALUATION OF CIRCULATING ENDOTHELIAL CELLS (CEC) IS A SUITABLE TOOL TO MONITOR ACUTE REJECTION IN RENAL TRANSPLANT RECIPIENTS

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Background: Endothelial damage is a major problem in transplant recipients. Recent data suggest that endothelial cell apoptosis precedes epithelial damage in the gastrointestinal tract in mice. However, little experimental data exists concerning early endothelial damage in human transplant recipients. Here we tested whether the evaluation of circulating endothelial cells is a suitable tool to monitor transplant associated endothelial damage.

Design: 19 renal transplant recipients were included in this study. Blood samples were collected prior to transplantation, 2 weeks, 3 and 6 months after transplantation. In addition, blood samples were analyzed when symptoms of acute rejection occurred. Endothelial cells were isolated by magnetic bead separation with an anti-CD 146 antibody counterstained with UEA-1 and counted. Routine biopsy specimens were evaluated for rejection and analyzed by a double labeling technique to monitor endothelial cell apoptosis and proliferation with either cleaved Caspase-3 (Cas 3) or Ki67 together with CD31.

Results: 4 out of 6 renal transplant patients with clinical and histopathological evidence of interstitial and vascular rejection revealed a significant increase of CEC. The number of CEC significantly dropped in these cases after treatment. The remaining two patients had only a slight increase in CEC, which stayed at the same level during the observation period. 7 patients without a rejection had a significantly lower number of CEC prior to transplantation, with a moderate increase at day 14 and a drop down to baseline levels after three months. Double labeling experiments only occasionally showed proliferating Ki67 positive cells and none of the cases showed apoptosis in endothelial cells with Cas 3. The remaining 6 patients had other histopathological signs such as acute renal failure.

Conclusion: Our results show that the number of CEC reflects episodes of interstitial/vascular rejections in renal transplant recipients. Thus, this method may be suitable to monitor ongoing acute rejection or may even predict vascular complications in transplantation. Interestingly, baseline levels from patients without rejection were lower than in patients with this complication, suggesting that patients with high CEC levels are more susceptible to develop vascular complications after transplantation. Surprisingly, endothelial cell apoptosis and proliferation was only occasionally detected. Thus, further evaluation of endothelial apoptosis is needed to define endothelial damage in transplant patients.

818 EXPERIENCE OF RENAL INVOLVEMENT IN MULTIPLE MYELOMA FROM A TERTIARY CARE CENTRE IN INDIA

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Background: Renal involvement is a major complication in multiple myeloma (MM) and approximately 20% patients develop progressive renal failure. Renal pathology in myeloma includes cast nephropathy (CN) and paraprotein deposition (AL- amyloidosis and monoclonal immunoglobulin deposition disease{MIDD}). Kidney biopsy often provides the initial diagnosis of myeloma in elderly patients presenting with renal failure. Prognosis of MM with renal involvement remains poor.

Design: Forty three renal biopsies from 400 diagnosed cases of MM with Durie & Salmon's criteria over last 15 years (1990 to 2005) from a tertiary care centre in India were retrospectively analyzed. Clinical details, laboratory parameter, radiological investigations, initial diagnosis, and follow up were retrieved from files. Renal biopsies were reviewed and correlated with clinical details and laboratory parameters.

Results: Four hundred cases with MM were admitted over a 15-year period to this tertiary care center in India. Renal involvement occurred in 43 cases (10.8%) with mean age of 56.1 years (age range: 37-76 years) and male: female ratio of 3:1. On clinical presentation renal failure and nephrotic syndrome was in 87% and 13% cases respectively. Mean S. creatinine was 8.4 mg% and 70% cases had M band in serum electrophoresis. On histology

37 cases had cast nephropathy and 6 cases had parproteininemia (AL- Amyloidosis). In 25 cases (58%) cases renal biopsy provided the first clue to the diagnosis of myeloma. Majority of cases presented in Stage III B (40/43 cases). On mean follow-up of 4.6 months (range: 0.3 - 30 months); 61% cases had persistent renal dysfunction, 14% had reversal of renal dysfunction and 25% cases died of disease.
Conclusion: Renal biopsy is helpful in differentiating patients with cast nephropathy from those with widespread amyloid deposits or with MIDD. In elderly persons with unexplained renal dysfunction, renal biopsy is indicated to rule out or confirm myeloma.

819 IMMUNOHISTOCHEMICAL STUDY OF MULTILOCULAR CYSTIC RENAL CELL CARCINOMA: HISTOPATHOGENETIC AND CLINICAL SIGNIFICANCE
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Background: Multilocular cystic renal cell carcinoma (MCRCC) is characterized by characteristic imaging and gross appearances, low grade clear cell renal cell carcinoma, and innocuous clinical outcome. The histogenesis of MCRCC is not well elucidated, and the relationship with renal multilocular cysts and particularly with mixed epithelial and stromal tumor (MEST) has not been studied.
Design: Six MCRCCs and eleven MESTs were retrieved from the Ottawa Hospital files. All cases were submitted for immunohistochemical studies.
Results: MCRCC and MEST had a Female:Male ratio of 1:2 and 9:2 respectively. There was no statistically significant difference in patient age or tumor size. Two MCRCCs consisted of solid foci of clear cell renal cell carcinoma and sclerotic, paucicellular intercystic septae and capsule. The remaining four MCRCCs (F:M ratio of 2:2) had areas with cellular septae and capsule similar to those of MEST. In addition, four MESTs displayed focal areas of clear cell change (CCC) of the epithelial lining. Immunostaining the cellular stroma and capsules of the latter four cases of MCRCC showed immunohistochemical patterns similar to MEST (positive immunoreactivity for ER, PR, and CD10). The epithelium of the latter four MCRCCs and the epithelium with CCC of the four MESTs also showed similar immunohistochemical patterns (positive immunoreactivity for CK7 and negative immunoreactivity for CD10).
Conclusion: There is a subset of MCRCC that develops from MEST as evidenced by the common immunohistochemical properties of the stroma of some MCRCC and MEST as well as CCC in some MEST.

820 GALLOWAY - MOWAT SYNDROME: A PODOCYTE DISORDER INCLUDING COLLAPSING GLOMERULOPATHY ASSOCIATED WITH A TRANSLOCATION T (3;16)
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Background: In 1968, Galloway and Mowat described a clinical triad in two sibs: an early nephrotic syndrome, a congenital microcephaly and a hiatus hernia. The Galloway Mowat syndrome is described as an autosomal recessive disorder of which genetic anomaly had not yet been identified.
Design: We report the case of a boy presented an early nephrotic syndrome, microcephaly, seizures, psychomotor retardation and cerebellar atrophy who died at 3 years and 11 months in a context of end stage renal function. He was the second child of a non-consanguineous marriage. There was no family history of nephrotic syndrome or end stage renal failure but his mother had moderate mental retardation. He presented dysmorphologic features including round face, full cheeks, narrow forehead, downslanting palpebral fissures, large mouth with thin lips, pointed large ears, rare and thick hair, and short second phalange of the second and third toe.
Results: Renal biopsy showed some focal segmental glomerulosclerosis associated with collapsing glomerulopathy with abundant visceral epithelial cells proliferation. But the majority of the glomeruli were sclerotic. For the first time, in Galloway-Mowat syndrome, the constitutional karyotype revealed a translocation t (3;16)(p14,p13.3) found in the patient and in his mother. She did not present renal desease but she could have a different breakpoint than her son or the child could have a second mutation in the other allele of his chromosome 3 or 16. Finally, we can not exclude that the gene responsible of the disease has an incomplete expressivity or penetrance.
Conclusion: We report the first case of Galloway-Mowat syndrome associated with a specific translocation and a collapsing glomerulopathy.

821 ADENOVIRUS INFECTION IN RENAL ALLOGRAFTS; AN ULTRASTRUCTURAL STUDY
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Background: Immunosuppressed patients are more susceptible to adenoviral infection and suffer a significantly higher mortality rate than immunocompetent patients. Renal transplant patients with adenoviral infection often present with infection of the kidney and urinary tract within weeks to months of transplant surgery, suggesting reactivation of a latent adenovirus in the immunosuppressed host as the source of infection.
Design: We describe five cases of adenovirus infection: four cases in renal transplant patients and one case associated with renal amyloidosis.
Results: Four renal transplant patients suffered graft dysfunction from two months to 3 years after transplantation. Immunosuppression was reduced and modified, which was followed by improvement in graft function. However, the renal dysfunction of one patient was aggravated and a subsequent biopsy showed C4d-positive acute humoral rejection associated with adenovirus infection. The renal amyloidosis patient, a 79-year-old female, exhibited proteinuria (3.1mg/day) and hematuria (many/HPF), but her serum creatinine level was within normal limits. On renal biopsy, the five cases exhibited common findings, including clear or smudged degenerated nuclei in tubular cells. Ultrastructurally, tubular cells showed perinuclear virus-like particles measuring 77nm to 97nm in diameter that were considered to be adenovirus.
Conclusion: Adenoviral nephritis should be considered in the differential diagnosis of allograft renal dysfunction.

822 A COMPARISON OF METHODS OF BK VIRUS DETECTION IN RENAL BIOPSIES
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Background: BK-virus (BKV) seropositivity can be found in 80% of the general population, resulting from an asymptomatic infection that often remains latent. In kidney transplant patients, BKV reactivation causes a tubulointerstitial nephropathy. This will manifest itself as progressive graft dysfunction, a condition with a wide differential diagnosis including graft rejection. Allograft biopsy is currently the preferred method to definitively diagnose BKV nephropathy. The aim of this study was to compare the agreement between three commonly used methods of BKV detection in paraffin-embedded renal biopsies: immunoperoxidase staining for the related SV40 polyoma virus (IPX), in-situ hybridization for BKV DNA (ISH), and BKV qualitative real time PCR.
Design: Thirty-one consecutive renal biopsies from February 2003 to May 2005 that were submitted for BKV detection were selected from the files at our institution, irrespective of the findings on H&E stained sections. Twenty-eight cases were evaluated by at least two of the methods. A central reviewer examined the H&E-, ISH-, and/or IPX-stained slides from each case. When performed, the real time PCR curves were also reviewed. A comparison of each method was performed via a 2 x 2 analysis utilizing Fischer's exact test.
Results: Nineteen cases were identified in which IPX and ISH were performed, with agreement in 18 cases (94.7%, p=0.0002). The discrepant case was positive by IPX but negative by ISH. ISH and PCR results agreed in 9 of 10 cases (90%, p=0.667), the discrepant case being positive by PCR and negative by ISH. IPX and PCR were in concordance in 9 of 12 cases (75%, p=0.11). One case was positive by IPX but negative by PCR with 2 cases being positive by PCR but IPX negative. In each instance, the cases that were positive by PCR and negative by the alternate method did not show typical findings of BKV nephropathy on corresponding H&E-stained sections.
Conclusion: There was at least 90% concordance between ISH results and those of both IPX and PCR with a lower agreement between IPX and PCR (75%). There was no statistically significant difference between BKV detection by IPX and ISH. Although it appears that IPX and ISH are less sensitive than PCR, discrepant PCR-positive cases may represent latent BKV infections and not BKV-induced nephropathy.

823 CHARACTERIZATION OF INFARCTS IN TRANSPLANT NEPHRECTOMIES
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Background: No clear guidelines are available for histologic examination of the transplant nephrectomy. Because multifocal thrombi are present in large and small renal arteries, extensive infarcts are a frequent finding. When the infarct is extensive, a common practice is to evaluate only a Hematoxylin and Eosin (H and E) stained section because tissue necrosis appears to obliterate any underlying histologic features. The renal parenchyma is uniformly pink. Architectural, nuclear and cellular detail are decreased or absent. However, the transplant nephrectomy is a dynamic organ in which persistent active cellular rejection is superimposed on chronic rejection. Because basement membrane persists in areas of renal infarct, the Periodic Acid Schiff (PAS) stain is a candidate stain that can be used to highlight tissue architecture within infarcted transplant nephrectomies and therefore allow histologic examination for the morphologic features of active and/or chronic rejection.
Design: Three micron, paraffin embedded sections from eleven transplant nephrectomies, with more than 50% infarction, were evaluated with H and E as well as PAS stains. The presence or absence of glomerulosclerosis, glomerulitis, tubular atrophy, tubulitis, interstitial fibrosis, endothelialitis and chronic allograft vasculopathy was recorded. If viable kidney was identified, a similar list of histologic features was recorded for each stain. Review of clinical and laboratory data yielded information regarding the presence of anti-HLA antibody in the recipient's sera, type of allograft kidney and the length of graft survival.
Results: Six transplant nephrectomies were from deceased donors and four were from living related donors. The type of donor of one transplant nephrectomy was not known. The graft survival ranged from 5 days to 165 months. Two transplant nephrectomies (Group A) were completely infarcted. Glomerulosclerosis, tubular atrophy, interstitial fibrosis, thrombi and chronic allograft vasculopathy were identified only in the PAS stained sections of the Group A, transplant nephrectomies. The status of anti-HLA antibody in the corresponding recipient`s sera was unknown. Nine transplant nephrectomies (Group B) had partial (subtotal) infarcts. Glomerulosclerosis (6/9), tubular atrophy (6/9), interstitial fibrosis (6/9), thrombi (9/9) and chronic allograft vasculopathy (8/9) were present only in the PAS stained sections of infarct from the Group B, transplant nephrectomies. Similar histologic findings were also noted in the H and E as well as PAS stained sections of the viable kidney. Anti-HLA antibody was present in the serum of five recipients before the corresponding Group B transplant nephrectomy. However, the presence or absence of antibodies to HLA antigens was not determined in four recipients.
Conclusions: The PAS stain can identify features of chronic damage in infarcts within a transplant nephrectomy specimen. Tubulitis, a feature of active cellular rejection and cellular infiltrates cannot be detected in the infarct because of the absence of nuclear detail. In spite of this limitation, the PAS stain can be used as an additional stain to evaluate