KIDNEY, LOWER URINARY TRACT AND MALE GENITAL SYSTEM PATHOLOGY

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Morphological changes in kidneys of AIDS patients and their significance in diagnostics

ABSTRACT

Clinical investigations and morphological analyses of the renal tissue in AIDS patients revealed a variety of clinical and pathologic features that could be diagnosed at the time of biopsy.The aim of this study is to check light microscopic, immunomorphological and electron microscopic changes in the kidneys of AIDS patients and to establish their specificity as well as their significance in diagnostics. By light, electron and immunofluorescence microscopy we have observed, the kidney tissue from 9 patients suffering from aids.The results of this study were compared with the results obtained from the morphological investigation of the renal tissue from aids patients performed several years ago in Mount Sinai School of Medicine in New York, USA. While in the previous investigation of 32 kidney biopsies, taken from aids patients performed in the USA the changes were found both on glomeruli and tubules, in our recent study 5 of 9 patients had focal segmental glomerulosclerosis, 2 had minimal change disease and all of them had tubulointerstitial changes. Changes in the kidneys, seen by light microscopy, except cystic dilatation of tubules, are not specific for AIDS; immunomorphological finding is not specific and has no significance in diagnostics; electron microscopic changes are, according to our findings as well as reported by most scientists who investigated renal changes in aids patients, of great importance for diagnostics, especially in early stages when clinical features of the kidney disease are not yet developed.

KEYWORDS: Acquired Immunodeficiency Syndrome; Microscopy, Electron; Kidney + pathology

INTRODUCTION

Different investigators studying morphological changes in the renal tissue of AIDS patients, have shown that in 7-10% of cases, as a result of AIDS, there was a kidney lesion which was described as AIDS-associated nephropathy.Clinically, it is presented as a nephrotic syndrome or proteinuria which progresses into uremia, which appears several weeks after the initial symptoms. In about 10% of the cases, the following manifestation appeared: acute tubular necrosis as a result of hipovolemic shock, sepsis, nephrotoxic drugs or radiocontrast agents; allergic interstitial nephritis as a result of using drugs; immunecomplex glomerulonephritis; focal-segmental glomerulosclerosis.

MATERIALS AND METHODS

By light, electron and immunofluorescence microscopy we have observed the kidney tissue from 9 patients suffering from aids.The results of this study were compared with the results obtained from the morphological investigation of renal tissue from aids patients performed several years ago in Mount Sinai School of Medicine in New York, USA.

RESULTS

In the previous investigation of 32 kidney biopsies taken from aids patients performed in USA, the following changes were found both on glomeruli and tubules:The changes on glomeruli were present in 19 cases: focal-segmental sclerosis in 12 cases; minimal change disease in 6 cases; membranoproliferative glomerulonephritis in 1 case; acute streptococcal glomerulonephritis in 1 case. The changes on the tubules were present in all cases: degenerative changes of microcystic tubules, variable degrees of fibrosis of interstitium and chronic inflammation; In our recent study 5 of 9 patients had focal segmental glomerulosclerosis, 2 had minimal change disease and all of them had tubulointerstitial changes, that were the same as previously described tubular changes in the group of patients investigated in USA. Immunomorphologicaly, neither we in our research, nor anybody else in literature has so far reported the presence of the HIV antigen in the kidneys. P24, P17, GP120 and GP41, used in revealing HIV antigen in other tissues, did not give reliable positive results on our biopsy material, so their diagnostic values are poor and unacceptable. By electron microscopy, we have found tubuloreticular structure inside the endothelial cells and leukocytes and great number of nuclear bodies inside the tubular cells.Many investigated glomeruli were collapsed.

DISCUSSION

In literature, D'Agati and Chander and Treser described to the detail EM changes on the kidneys. According to D'Agati, characteristic changes on the kidneys in cases of AIDS, are: appearance of collapsed glomeruli; increase and swelling of visceral epithelial cells; focal-segmental glomerulosclerosis; tubuloreticular structures in endothelial cells and leukocytes; increased number of nuclear bodies in tubular and interstitial cells; granular transformation of chromatin in tubular and interstitial cells. According to Chander and Treser, characteristic EM findings are: presence of complexes of nuclear bodies III-V type; presence of "membranous profiles", "ring shape" and "test tube" forms in the nucleus and cytoplasm of the renal cells. Our investigation revealed tubuloreticular structure inside endothelial cells and leukocytes and a great number of nuclear bodies inside the tubular cells to be the characteristic change always present in AIDS related nephropathy. Also, very often the glomeruli have a collapsed appearance. Unlike immunomorphological changes that do not give any relevant finding in AIDS nephropathy, electron microscopic investigation of these cases can be useful very early.

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Prostate - specific antigen (PSA) in neoplastic and hyperplastic prostate tissue

ABSTRACT

Prostate - specific antigen (PSA) is a biological marker of prostatic tissue, that is present in the cytoplasm in normal, benign hyperplastic, malignant prostatic tissue and in metastatic prostatic cancer. The aim of this study was to comparate the histopathological features (Gleason grade) of prostate carcinoma with PSA content. This study was performed on 15 neoplastic and 9 hyperplastic and normal prostates (control grup). The PSA expression was evaluated immunohistochemically on formalin fixed and parrafin embedded tumor samples, using ABC method. The immunohistochemical analysis of PSA indicated an immunoreactivity in all prostate adenocarcinoma (except particular fields of Gleason grade 5B). The largest number of analyzed tumor fields had shown trace (+-) or medium (+) reactivity. Content of PSA is reduced with decrease in differentiation of prostatic cancer. Hyperplastic epithelial cells almost always showed intensive PSA staining (++). Statisticaly significant difference in PSA content was observed between neoplastic and hyperplastic tissues. The authors suggest that the content of PSA was reduced with a decrease in differentiation of prostate cancer and that hyperplastic tissue shows statisticaly stronger immunoreactivity than neoplastic tissue.

KEYWORDS: Prostate-Specific Antigen; Prostatic Neoplasm; Prostatic Hyperplasia

INTRODUCTION

Among all the malignant tumors diagnosed in males, approximately 13% are prostate cancer (PCa), while PCa accounts for 8.6% of deaths (5). Prostate - specific antigen (PSA) is a single - chain glicoprotein, normally present in the cytoplasm of prostatic acinar cells and ductal epithelium (6). PSA is present in normal, benign hyperplastic and malignant prostatic tissue, in metastatic prostatic cancer, and also in prostatic fluid and seminal plasma (1,2,4). It appears that this enzyme is detectable in more than 90% to 95% of PCa tissues. Histochemical analysis with PSA has become very useful in determining a prostatic origin in biopsy material showing poorly differentiated cancer of unknown origin (4).

MATERIALS AND METHODS

This study was performed on 15 neoplastic prostates, obtained by radical prostatectomy and TUR resection, Gleason grade and PSA content were studied in them. Hyperplastic and normal prostate tissue has been used as control group (9 prostates). The PSA expression was evaluated immunohistochemically on formalin fixed and parrafin embedded tumor samples, using ABC method.

RESULTS

The immunohistochemical analysis of PSA indicated an immunoreactivity in all prostate adenocarcinoma (except particular fields of Gleason grade 5B). We have shown characteristic staining of cancer cell cytoplasms. The largest number of analyzed tumor fields have been shown trace (+-) or medium (+) reactivity. With a decrease in differentiation of the prostate cancer, the content of PSA was reduced. Well-differentiated carcinomas Gleason grade 1 and 2 have shown medium (+) and intensive immunoreactivity (++) in 66.66%. All moderate differentiated tumors (Gleason grade 3) showed trace (46.67%) and medium reactivity (53.33%). Poorly differentiated tumors (Gleason grade 4 and 5) have shown reaction in trace. Prostate adenocarcinoma Gleason grade 5 have not shown PSA content in 40% of the analyzed fields. Hyperplastic epithelial cells showed intensive staining (++) to PSA troughout the entire cytoplasm. The intensity was uniform between cells and indistinguishable between ducts and acini. A statisticaly significant difference in PSA content was observed between neoplastic and hyperplastic tissue (x^2 =29,05 $p<0,001$).

DISCUSSION

Prostate-specific antigen has been shown to be the most effective immunohistological marker for prostate cancer, as well as the most useful serologic test in staging and monitoring prostate cancer and in early detection of the recurrent disease (1,2,4). The results of the current study are similar to other reports in the literature. Staining of normal and hyperplastic prostate epithelium for PSA was uniform and strong. Staining of carcinomas was less than that seen in benign prostatic tissue. In contrast to the benign tissue, prostatic carcinoma demonstrated heterogeneity in field-to-field staining for PSA. In our studies, an apparent correlation between staining variability and increasing tumor grades was noted. Variations of PSA expression seen with immunohistochemical staining could be a result of aberrant gene transcription or from decreased translation of the PSA mRNA. Either of these mechanisms would result in a decreased PSA expression (3). Immunohistochemical staining for PSA has been particularly useful in the identification of metastatic prostatic cancer and of morphologic variants of prostate cancer in differentiating prostatic carcinoma which secondarily involves the bladder from primary urothelial carcinoma and vice versa (1). Clinical studies have shown that PSA is a very good serological marker for monitoring the clinical course of PCa. Some studies have indicated that as many as 20-25% of all prostatic tumors may not express or have very low levels of PSA (5). Others, have found PSA in 30-40% of breast tumors and at a lower percentage in other tumors, including lung, colon, ovarii, liver, kidney, adrenal and parotid tumors. Some authors have found PSA in skin and salivary gland tumors and in normal endometrium. The physiological role of PSA in these tissues and tumors is currently unknown (3).

CONCLUSION

The authors suggest that the content of PSA was reduced with a decrease in differentiation of prostate cancer and that hyperplastic tissue shows statisticaly stronger immunoreactivity than neoplastic tissue.

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Disseminated prostatic carcinoma with normal serum level of prostate specific antigen

ABSTRACT

High sensitivity and specificity, short serum halftime and standardized diagnostic, monitoring and therapeutic levels establish PSA as an ideal tumor marker. When used alone, the diagnostic value of the prostate specific antigen (PSA) used to predict tumor extent is limited for the individual patients with prostatic carcinoma (PC). The study aim was to establish the correlation of the serum PSA level in addition to grade, histology type, and clinical stage of PC in 37 patients with low preoperative PSA serum level. In 37 untreated PC patients with preoperative serum PSA level ranged 0,1 to 9,6 ng/ml, paraffin-embedded tissue and serum samples were studied by immunohistology and immunoassey for PSA. Most representative were 27 (73%) cases of poorly differentiated PC with GGS>7. In 9 (24.3%) PC cases the C stage was obtained, and in 28 (75.7%) PC cases the D stage. In serum samples from PC patients 27 (73.7%) had normal ((4,0 ng/ml), and 10 (27.3%) intermediate (4,1-10 ng/ml) PSA levels. Immunohistochemistry (IMM), in 35 PC (94.5%) had demonstrated expression of PSA. Our study results show that serum PSA levels in PC patients with D stage are not representative marker for progression in some poorly differentiated PC.

KEYWORDS: Prostatic Neoplasms; Prostate-Specific Antigen

INTRODUCTION

More than 70% of new diagnosed PC are staged as local progressive or disseminated form. The serum levels of PSA represent a significant diagnostic and monitormg parameter of PC progression. However, the elementary experiences based on immunoasseys and IMM analysis showed a low serum level of PSA in advanced stage of some poorly differentiated PC (1,2).

MATERIALS AND METODS

The study utilized the biopsy material of 37 PC cases obtained by transurethral resection and radical prostatectomy. The serum PSA level was performed by Hybritech method of monoclonal immunoassey. PC grading

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follows the parallel standards of WHO and Gleason grade score(GGS). Tumor staging was classified by Whitmore-Jewet system. The level of PC exocrine differentiation by monoclonal antibodes against PSA was detected through IMM- PAP analysis in two representative tissue sections (containing over 50% of the tumor tissue). Tissue sections containing over 50% of the reactive tumor cells exhibited significant IMM level. Statistic data processing was performed by Student T test.

RESULTS

We analyzed 37 cases of advanced stage PC with preoperative serum PSA level rangig 0,1 to 9,6 ng/ml. Most representative were 27(73%) cases of poorly differentiated PC with GGS~7. In 9 (24.3%) PC cases the C stage was obtained, and in 28(75.7%) PC cases the D stage. The histology type of the analyzed study cases includes acinar carcinoma, two ductal PC(DPC), one Signet Ring, one urothelial PC(UPC), and two carcinosarcomas (CPC). Normal serum PSA levels (`4ng/ml) were found in 27(73%) PC, included UPC, two CPC and both DPC, and intermediate PSA levels (4-lOng/ml) in 10 (27%) PC. The average (X±D)serum levels of PSA in advanced stage PC are presented in Table 1.

Data processing resulted in significantly higher PSA levels within moderatly differentiated PC than in poorly differentiated PC (p`0,05). Significantly higher PSA levels were found in patients with C stage related to D stage (p`0,01). Significant level of IMM reactivity was found m 36 PC, while in UPC

Table 1. The average (X±D) serum levels of PSA in advanced stage PC

PSA ng/ml	G1	G2	G3	GGS<7	GGS>7	C	D
No	1	6	27	7	27	9	28
X	5	6.80	3.86	4.98	4.04	6.73	2.96
SD	0	2.23	2.16	2.61	2.51	2.37	1.46

immunohistochemical PSA reaction wasnot detected.

DISSCUSION

High serum levels of PSA follow over 90% of advanced PC(1,2). The tumor marker specificity and sensitivity may be very variable in diagnostic and monitoring of advanced PC with low serum PSA level (2,3). In rare variant of disseminated PC, PSA as tumor cell secretion product may be not/or minimally detected (4). That was the case of one UPC and two CSP presented in our study series. Our analysis results presented the difference in low serum and significant tissue PSA detection. Low serum PSA level identified in advanced PC, may be a consequence of genetic copy. In 1996 Baffa et al. showed that malignant and normal prostatic tissue express same PSA genetic copy. PSA molecule changes in PC patients represent certain post-translation event (5). Unrecognized PSA antigenicity by low serum titer in disseminated PC, may be consequence of binding this molecule to serum antiprotease. Serum antiprotease activity is irrelevant for PSA tissue expression. However, PSA molecule with masked epitope was detected in cytoplasmatic protein complexes. The loss of some PSA epitope m PC may cause persistent low serum PSA levels during diagnosis and monitoring of PC progression (6). The serum PSA level in our study group ranged 0,1 to 9,6 ng/ml, while in 27 patients the normal PSA level was detected. In our study group the average serum PSA level for D stage ranged 2,96±1,46 ng/ml, and for C stage ranged 6,73±2,37 rig/ml. This paradoxical PSA activity according to C stage was approved by statistically significant high PSA levels (p<0,01) detected in lower stage of PC. Accessible literature contains no similar results for undetectable serum PSA level in advanced PC. Our study results differ from numerous clinical studies conclusions that established a correlation in PSA serum level and PC grade and stage (2,3). In most cases (75.7%) we found disseminated PC. Our study results show that semm PSA levels in D stage are not a representative marker for progression in some poorlly differentiated PC.

CONCLUSION

Our study results show that serum PSA levels in PC patients with D stage are not representative marker for progression in some poorly differentiated PC.

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Extended abstract
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Classification of drugs‘ risk degree for spermatogenesis

ABSTRACT

After the thalidomide disaster in 1960/1961 and its awfull consequences on the offspring of the exposed women in the countries of Western Europe and Australia, many countries made different classifications of teratogen risk. None of these classifications include estimattions of the risk degree of drugsí use for spermatogenesis and male reproductive potential. In recent 10-20 years many data (experimental and clinical) about the influence of drugs on male reproductive system have accumulated, giving an opportunity to make such a classification. The airn of this study was to propose numerical classification of drugsí risk degree for spermatogenesis. Comparision of different classifications of teratogen risk and evaluation of the data from 6561 references were source for classification. This work resulted in the first world‘s classification of drugs‘ risk degree for spermatogenesis. This classification has four different numerical levels of risk. This classification of drugs‘ risk degree for spermatogenesis is clear and simple for use.

KEYWORDS: Spermatogenesis; Pharmaceutical Preparations + adverse effects + classification; Teratogens

INTRODUCTION

Much evidence has accumulated in the recent years to suggest that there has been a gradual increase in male reproductive pathology over the past 3-4 decades, as evidenced by an increased rate of testicular cancer and declining semen quality. The most popular hypotheses for pathogeneses of these disorders is one that this phenomenon is casually related to the ability of male germ cells to generate reactive oxygen metabolites. It is thought that low levels of such metabolites enhance sperm function by stimulating DNA compaction and promoting a redox- regulated cAMP-mediated pathway that is crucial for the induction of sperm capacitation. But, when the same metabolites are produced in excessive amounts, they stimulate DNA fragmentation and a loss of sperm function associated with peroxidative damage of the sperm plasma membrane. In light of these considerations free-radical induced mutations in the male germ line may also be involved in the aetiology of childhood cancer and increases in the incidence of seminoma. Different studies confirm that male sperm counts are declining, and environmental factors, such as pesticides, food additives, heavy metals and different drugs (exogenous estrogens, cytotoxic drugs, sulphasalazine, etc.) may negatively impact spermatogenesis. Spermatogenesis is the process of cellular differentiation by which immature male germ cells pass through a complex series of events involving mitosis, meiosis and become mature spermatosoa capable to fertilize an ovum. This process involves the developmental progression of male germ cells through a number of spermatogenic cell types, each of which is characterized by unique features of morphology, cellular association and specialized functions. Worldwide a health administrations of different countries has made a more rigorous regulations concerning the requirements for registration of the drugs after thalidomide disaster in 1960/1 961 and its awfull consequences on the offspring of the exposed women of Western Europe and Australia. The first established classification of teratogen risk hierarchy system in the world appeared in 1978. But later, many countries made their own classification systems: Denmark, Netherlands, USA, Switzerland and so on. Altough there exist many different classifications of teratogen risk, nobody has been concerned with the effect of the drugs on spermatogenesis, but only on the course and outcome of pregnancy. The aim of this study was to propose numerical classification of drugsí degree risk for spermatogenesis.

MATERIALS AND METHODS

The comparision of different classifications of teratogen risk and the evaluation of the data from 6561 references concerning drugsí effects on spermatogenesis and the course and outcome of pregnancy were the source of new classification (1).

RESULTS

Classification of drugs‘ risk degree for spermatogenesis (legend for examples: S - spermatogenesis): 0∞/3∞ - no known risk; 1∞/3∞ - clear risk, animal studies show harmfull effects; 2∞/3∞ - clear risk, but harmfull effects are transient; 3∞/3∞ - clear risk, but harmfull effects are permanent or harmfull effects are possibly related to mechanism of drug action.

Examples: Paracetamol - S: 0∞/3∞; Diazepam - S: 1∞/3∞; Sulfasalazin - S: 2∞/3∞; X rays - S: 3∞/3∞.

DISCUSSION

In recent 10-20 years many data (experimental and clinical) about the influence of drugs on male reproductive system have accumulated, giving us an opportunity to evaluate the risk degree. For that reason it is necessary to make a classification system of drugsí risk degree for spermatogenesis, what would be of practical importance and which use would prevent many dilemmas and wrong decissions. Itís well documented that some drugs or groups of drugs have adverse effects on spermatogenesis only in animal studies. It is established that benzodiazepine ñ diazepam application in mice (2) and nitrazepam in mice (2) and rats (3) induce adverse effects on spermatogenesis, but until now such effects have not been reported in men. Other drugs or groups of drugs have been found to have transient adverse effects in men. In that case it is important to advise the patient who used such drugs to avoid the possibility of fertilization over a certain period. For example, men exposed to 131I during the diagnostic procedure could plan to make offspring after at least 120 days (4). There is a similar situation with sulfasalazine. Men who take sulfasalazine should avoid to make offspring during six months after its discontinuation or if its use is necessary sulfasalazine should be replaced by mesalazine. Also itís well known that some drugs (example: some antineoplastics with alkylating effects) and X-radiation may induce irreversible damage of male germinative epithelium and lead to permanent sterility. Whenever possible a patient should be informed about such effects of drugs. It should be suggested to the patient to store sperm and to give him a chance to have offspring later, when he gets cured (1). Also, we should not neglect the psychological effects of this procedure. It gives a hope to patient that he will survive and lead a normal life.

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CONCLUSION

The benefit of proposed classification of drugs' degree risk for spermatogenesis is that it gives simple and fast assesment of the risk degree and precise evaluation of risk degrees compared to drugs with similar effects. In information for drug use should not be only this classification of drugs' degree of risk for spermatogenesis, but also short text with explanation of its adverse effects.

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CMV infection in renal transplant recipients

ABSTRACT

The aim of this retrospective study was to evaluate effect of CMV infection on allograft dysfunction, graft or patient survival after transplantation. The study includes 46 patients undergoing isolated kidney transplantation during the period from 1996 to the end of 1999. In the last studies CMV is found associated with increased risk of bacterial and opportunistic infections, acute and chronic rejection, allograft loss, and death in kidney transplant recipients. It was found that CMV infection is added to the posttransplant risks in most patients. CMV infection was diagnosed in 16 of 46 (34.78%) patients with a corresponding serological proof. Based on literature data and our study results we conclude that conducted CMV monitoring is necessary for adequate therapy approach providing consequtive better allograft function.

KEYWORDS: Cytomegalovirus Infections; Transplantation, Homologous, Kidney Transplantation; Graft Rejection

INTRODUCTION

In the last decade of XX century Cytomegalovirus remains a significant source of morbidity and mortality in transplanted immunocompromised hosts. CMV infection was found as common, serious complication of transplantation (1), that may trigger the organ dysfunction, or lead to severe pulmonary or other diseases, which untreated might have negative curse of disease. In the last studies CMV is found associated with increased risk of bacterial and opportunistic infections, acute and chronic rejection, allograft loss, and death in kidney transplant recipients.(2) It was found that CMV infection is added to the posttransplant risks in most patients. The risk factors of CMV infection in allograft recipients are summed up as follows: 1) transplantation of CMV seropositive donor to seronegative recipient; 2) increased antithymocyte drug levels; 3) increased number of steroid boluses in treatment of rejection; 4) second transplantation; 5) young age of allograft donor (3); and 6) blood transfusions before as after the transplantation. Pathohistology verification of CMV infection in allograft tissue is possible only in individual rare cases. Major histomorphology elements of CMV infection are foamy cells with bloated cytoplasm in tubular epithelium or interstitial located, and occasionally inclusion bodies "owl's eye cell". These morphology changes more often precede cell death, lytic necrosis. The aim of this retrospective study was to evaluate effect of CMV infection on allograft dysfunction, graft or

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patient survival after transplantation.

MATERIALS AND METHODS

The study includes 53 patients undergoing isolated kidney transplantation during the period from 1996 till end 1999. All relevant clinical and laboratory data were analyzed with accent to 26 patient with CMV infection. The study abstracted information concerning: relevant diagnosis, age of transplantation, date of allograft failure, and the date of the retransplantation, date of death, onset of rejection episodes with attached pathology findings, recipients and donor serological status, medication protocols for: antiviral therapy, rejection therapy and intensity of immunosuppression and study outcomes. The CMV infection detection and monitoring were carried out by standard virological assays of CMV, including shell viral assay, standard fibroblast culture, and serology, as well as white blood cell count. Measuring the antibody level was provided by Microparticle-enzyme immunoassay (MEIA) technique for the IgM and IgG titer, complement fixation reaction (CFR) for the virus antigen detection, immunofluorescence test (IFT) for PP 65, and isolation test (ISOL) for CMV virus culture isolation. Four indexes were used to determine the presence of CMV infection: fourfold rise in antibody titer; conversion to an antibody titer >1:8; positive antigenemia; and direct isolation of the virus from urine. Renal allograft biopsies were obtained in 16 patients in which 7 patients with active CMV infection. The bioptic material underwent standard methods of tissue processing for EM.

RESULTS

During the follow-up period 46 patients underwent isolated kidney transplantation and 2 patients were retransplanted. 4 (%) kidney grafts within study group failed as follows: 1 caused by renal thrombosis, 2 due to chronic rejection and 1 with recovered primary renal disease. In the study population was varified 42 (72,41%) rejection episodes. There was no death during the follow-up period. Demographic and immunologic characteristics of the recipients: the study subjects were 16 of 46 (34.78%) patients identified as CMV infected patients with a corresponding serological proof; the average age of the recipient was 34,42 years (range, 15 to 53 years); the ratio of males to females was 3,5:1. In course of the pretransplantat diagnosis of chronic renal failure there were verified 26 patients with chronic glomerulonephritis unknown origin; in 7 patients mesangial nephropathy; in 3 patients Alport sy; endemic nephropathy, nephrolithiasis, Goodpasteur sy and Wegenerís granulomathosis in 2 patients each, and hypertensive nephrosclerosis, focal glomerulosclerosis and focal/segmental glomerulonephritis of infective endocarditis in 1 patient each. CMV IgG-status constellation in the study group preoperative was 19 (41,30%) seropositive and 27 (58.69%) seronegative patients.

Mean interval from transplantation to CMV infection onset was 157,93 ± 251,6 days (median 26,5 days). From the epidemiologic point of view, CMV infection was featured as a primary infection at 6 (37.5%) recipients; reactivation took place in 10 (62.5%) recipients. In the transplantation groups there have been found 16 patients who were treated for CMV infection, 9 (56.25%) patients with mild infection and 7 (43.75%) asymptomatic infection. The blood count disorders for the duration of CMV infection were found as follows: of the 16 CMV infected recipients thrombocytopenia developed in 10 recipients, lymphocytosis in 15; leucopenia developed in 10 recipients, but neutropenia and thrombocytopenia in 9 recipient each. CMV excretion from urine was observed in 7 (43.75%) infected recipients.

In case of a CMV infection, gancyclovir was administered in 26 (56,52%) patients and in 16 CMV infected patients for therapy purpose intravenously until resolution of the clinical symptoms or viremia and afterwards continuously by oral application. In 4 patients during anti-rejection treatment and in 14 patients during the antilymphocyte induction reliable CMV infection prophylaxis by gancyclovir was simultaneously applied as a short course (14 days). In the CMV group of 26 patients undergoing ganciclovir administration, the average graft survival was 1,4 years. In 23 study cases, immunosuppression therapy began peri-operative as a Quadruple therapy (Cyclosporine, Azathioprine, Steroids, ATG). No significant influence was noticed between ATG administration and infection outbreake. Rejection therapy was administered in 36 rejection episodes as steroid bolus dosed 25o-5oo mg over 3 consecutive days. 3 (6.5%) of the recipients showed a steroid resistant rejection, so that initial polyclonal antibody application was recommended. Observed rejection episodes were without effect to the onset of CMV infection. 5 patients with CMV infection didnot undergo rejection episodes, and 11 patients displayed irrelevant period between CMV infection outbreak and rejection.

From 16 biopsied patients 7 (43.75%) had CMV infection, and only in 2 (12.5%) of these patients CMV infection was subsequently distinguished in revision-analyze of pathohistology findings. The characteristic elements of CMV infection in biopsy findings were present as foamy cells with bloated cytoplasm in tubular epithelium or interstitialy located, and inclusion bodies „owl's eye cell".

DISCUSSION

Haberal found CMV infection as abnormally more frequent in allograft recipients and that immunosuppressive drugs play an important role in this increased prevalence (6). However, the transplant itself represents a major source of viral infestation at a time of maximal immunology risk in recipients lacking specific preexisting defense. In addition, it is generally known that immunologic stimulation or immunosuppressive drugs may reactivate viral production from genome provirus. In the most cases pathohistology findings confirm the CMV infection but not disover. Only in rare cases of chronic rejecton morphologcal elements of CMV infection may be first varified (recognized) in allograft tissue, and later confirmed with sensitive serology technique. In these cases electron microscopy (EM) represent usefull method of cellular CMV evidence. This analysis also allows estimation of CMV infection activity (i.e. CMV replication). This method of patients monitoring enable correct therapy approach and prevent poor infection course. Renal biopsy findings benefit the diagnostic of CMV infection and viral activity in allograft tissue itself (i.e. CMV replication causing degenerative changes and lytic necroses of infected cells). The principal value of biopsy findings is to estimate the rejection episode and steroid toxicity or to diagnose recurent and de novo glomerulonephritis, glomerulopathy, arteficial surgical complications etc. (4,5). In all diagnosed cases the CMV infection was consider as numerous infection with no life-thratening course. Positive effects of administered antiviral prophylaxis and therapy stand out in fast resolution of clinical signs and serology findings. Based on literature data (7) and our study results we conclude that conducted CMV monitoring (particcularly in serology findings) is necessary for adequate therapy approach providing consequtive better allograft function. According to our experience and other study data best effects in controling CMV infection were achieved with short course gancyclovir prophylaxis during ALA application (8,9). Our point of view is that this way of CMV infection monitoring proceeds the multitude, reliable important results that can be applied for further CMV infection investigations.

CONCLUSION

This retrospective study was designed with the following relations between CMV infection itself (as typ, outbreak, duration, infection, monitoring etc), CMV infection under transplantation conditions (as transplantation typ with attaching events as rejection, immunosuppression influence, ALA therapy) and medicament treatment of CMV infection. The study aim was conducted, in attempt to indicate appropriate relation mechanisms.

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Histological, histochemical and immunohistochemical changes of benign prostatic hyperplasia (BPH) after the Chernobyl accident in Ukraine

ABSTRACT

After the Chernobyl accident during the last years the incidence of prostate carcinoma in Ukraine increased from 5.1 to 18.3 per 100 000 of the male population. It is known that a cancer risk at high doses of ionizing radiation implies a cancer risk at low doses. Morpholigical, histochemical and immunohistochemical changes in prostatic tissues from patients with benign prostatic hyperplasia who underwent surgery before and after the Chernobyl accident were studied. BPH samples were obtained by adenomectomy from 45 patients operated before the accident (group I), and 47 patients from Kiev-City (group II), and 76 patients from radiocontaminated areas of Ukraine (group III). Their BPH samples were examined histologically, histochemically (apoptosis and Ag-NORs proteins) and immunohistochemically (p53, Ki-67 and PCNA). The levels of the Cs137 in the urine of 51 patients with BPH (20 patients from group II and 31 patients from group III) have been measured. The present study shows the increase of incidence of the prostatic intraepithelial neoplasia (PIN) in BPH, proliferative activity and apoptosis in BPH as well as in PIN from patients who live in the radiocontaminated areas of Ukraine and in Kiev-City. The radionuclide Cs137 has been excreted via the urine of these patients to compare with the pre-Chernobyl group. Those changes could be a result of influence of the long-term low dose ionizing radiation.

KEYWORDS: Prostatic Neoplasms; Immunohistochemistry; Apoptosis; Accidents, Radiation; Neoplasms, Radiation Induce

INTRODUCTION

Fourteen years passed after the accident at the Chernobyl power plant in Ukraine. Chernobyl accident - is not only the terrible technical disaster that took place in 1986, it is a reality in which Ukrainian people live now and will live in the future. After the Chernobyl accident about 10 million people were exposed to low doses of the ionizing radiation during 14 years already. It is

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known that a cancer risk at high doses of ionizing radiation implies a cancer risk at low doses (1). Last years the incidence of the prostate carcinoma increased significantly from 5.1 in 1986 to 18.3 in 1998 per 100000 of the male population in Ukraine. It is very important to understand the causes of this increasing and the association with the long-term influence to low dose ionizing radiation. The present study shows the correlation between the morphological changes, proliferative activity of the prostate epithelium and apoptosis that could be associated with radiation exposure. People who exposed the influence of the low doses of the ionizing radiation contain the whole bodies radionuclide Cs137 and excrete it via urine (3). We have measured the levels of the Cs137 in the urine of these patients.

PATIENTS, MATERIALS AND METHODS

We have analyzed morphological, histochemical and immunohistochemical changes in prostatic tissues from 168 patients with BPH who underwent open adenomectomy in the Institute of Urology and Nephrology in Kiev, Ukraine. Group I, the control group - 45 cases from patients with BPH who underwent surgery before the Chernobyl accident in 1984. Group II - 47 cases from patients with BPH who are living in the city of Kiev after the Chernobyl accident (contamination level at inhabiting area4 - 0.5 - 5 Ci/km2). Group III - 76 cases from patients with BPH who are living in the radiocontaminated areas of Ukraine (contamination level at inhabiting area - 5 - 30 Ci/km2). The surgical specimens were fixed in 10 % buffered formalin and processed by standard technique to paraffin wax. Sections measuring 4 mm in the thickness each were cut and stained with hematoxylin and eosin for routine histologic examination and were used for histochemical and immunohistochemical studies.

The mouse monoclonal antibodies PCNA #DAKO# (Glostrup), mouse monoclonal antibodies Ki-67 clone MIB-1 #Immunotech# (Westbook, ME) and Vectastain Elite ABC Kit (Vector Laboratories) were used to identify PCNA and Ki-67 expression. The mouse monoclonal antibodies p53 #DAKO# (Glostrup) and Vectastain Elite ABC Kit (Vector Laboratories) were used to identify p53 accumulation. The ApopDETEK Cell Death Assay System (Enzo Diagnostic) and Simply Sensitive Horseradish Peroxidase-DAB in situ Detection System (Enzo Diagnostic) were used to identify apoptosis. We have assessed PCNA, Ki-67 and p53 immunostaining and apoptosis using an Olympus light microscope in areas of PIN as well as in BPH. The index of immunoreactivity defined as the percentage of positively stained cells was determined after evaluating 2 000 prostatic epithelial cells. To detect nuclear organizers (Ag-NORs - proteins) were stained in the mixture of 5 % gelatin, 50 % AgNO3 and 1 % HCOOH in the dark. Then sections were incubated with 5 % Na2S2O3 for 5 minutes. We have assessed Ag-NORs - proteins using an Olympus light microscope in areas of PIN and in BPH. The average contents of Ag-NORs ñ proteins were calculated in 2 000 prostatic cells. Standard (-radiometer ìRUB ñ 01î was used for measurement of Cs137 in the urine of 51 patients with BPH (20 patients from group II and 31 patients from group III).

RESULTS

Excretion of Cs137. The radionuclide Cs137 was detected in the urine in all cases of groups II and III. The difference between the levels of Cs137 in those groups is not significant (1.14 (0.43 and 3.22 (1.90 correspondingly; p(0.05).

Microscopically, multiple foci of prostatic intraepithelial neoplasia (PIN) were observed in 15.55 %, 29.79 % and 35.53 % of groups I, II and III respectively. Only the differences between the incidences of PIN in groups I and III were significant (p< 0.02).

Histochemistry: the apoptotic indices were greater in PIN than in BPH. The apoptotic indices in BPH and PIN of groups II and III (Table 1) were significantly greater than of group I (p(0.001). The average contents of Ag-NORs ñ proteins were greater in PIN than in BPH in all groups. The differences between the average contents of Ag-NORs ñ proteins in BPH and in areas of PIN of groups I, II and III (Table 1) were not significant (p (0.05).

Immunohistochemistry: the indices of PCNA immunoreactivity were greater in PIN than in BPH in all groups. The indices of PCNA immunoreactivity in BPH and PIN of groups II and III (Table 1) were significantly greater than of group I (p(0.001). The indices of Ki-67 immunoreactivity in BPH of groups II and III (Table 1) were significantly greater than of group I (p(0.001). The differences between the indices of Ki-67 immunoreactivity in PIN of groups I and II were not significant (p (0.05); index of Ki-67 immunoreactivity in group III was significantly greater than in group I (p(0.001). p53 protein expression was detected in nuclei in all cases of groups I, II and III in areas of PIN. The majority of p53-positive cells were occurred in basal layer cells. The indices of p53 immunoreactivity in groups II and III (Table 1) were significantly greater than in group I.

DISCUSSION

The present study has demonstrated a two-fold increase in incidence of PIN in BPH patients who are living in the radiocontaminated areas of Ukraine and in Kiev-City after the Chernobyl accident. We have suggested that such increasing of the proliferation of the prostatic epithelium could be a result of the long-term radiation exposure (7). The observed increase of apoptotic index in BPH and PIN from patients living in the less contaminated (city Kiev) and high contaminated areas of Ukraine could be a result of direct influence of the chronic long-term to low doses ionizing radiation on the prostatic epithelium. Stable p53 protein expression was detected in majority of basal cells in PIN. It has been reported that basal cell represents stem cells of the prostatic epithelium (2). We have detected the great increase of indices of p53 immunoreactivity in PIN patients exposed by low doses ionizing radiation (groups II and III) as compared with the control group I. It is known that high doses of the ionizing radiation induct rude damages in the cell genome, such as gene deletions and rearrangements, gross chromosomal deletions and rearrangements and point mutations. On the contrary, low dose radiation exposure leads to the epigenetic changes of proteins (8). We suppose that p53 stable protein expression, which mimicking the mutant p53, is a result of such epigenetic changes arising by influence of low dose radiation. The significant increase of basal stem cells which expressed the epigenetically changed stable p53 protein in areas of PIN in patients that were exposed to low doses of the ionizing radiation indicates the early steps of prostate carcinogenesis pathway. Influence of low dose ionizing radiation on the p53-positive cells might remove initiated cells PIN via promotion phase of the carcinogenesis that leads to the proliferation. As it is known that carcinogenesis consists of three phases: initiation, promotion and progression the low doses of the ionizing radiation could be a potential progressor of carcinogenesis in previously initiated and promoted cells (6). But only the regular and sustained influence of low-dose radiation exposure could act as a mitogen or tumor promoter (5).

Table 1. Histochemical and immunohistochemical study of the prostatc epithelium in BPH and PIN

	Group I		Group II		Group III	
	BPH	PIN	BPH	PIN	BPH	PIN
Index of p53 immunoreactivity, %	0	12.40±0.64 p<0.001	0	14.40±0.20 p<0.001	0	23.76±1.00 p<0.001
Index of Ki-67 immunoreactivity, %	0.02±0.005 p<0.001	0.1±0.008 p>0.05	0.20±0.08 p<0.001	0.83±0.24 p<0.001	0.43±0.10 p<0.001	1.92±0.43 p<0.001
Index of PCNA immunoreactivity, %	2.65±0.41 p<0.001	7.07±0.52 p<0.001	3.51±0.36 p>0.05	9.22±0.77 p<0.001	3.79±0.29 p<0.001	12.02±1.62 p<0.001
Apoptotic index, %	0.05±0.03 p<0.001	0.44±0.38 p<0.001	2.64±0.05 p<0.001	3.15±0.09 p<0.001	3.96±1.32 p<0.001	10.21±4.44 p<0.001
Ag-NORs, %	1.48±0.21 p>0.05	4.41±1.18 p>0.05	1.58±0.32 p>0.05	6.53±0.95 p>0.05	1.67±0.18 p>0.05	6.92±1.43 p>0.05

CONCLUSION

Prostatic stem cells of patients with BPH and PIN who are living in the radiocontaminated areas of Ukraine and in Kiev city all have been exposed

such regular and sustained influence to low doses of the ionizing radiation that could be one of the causes of the prostatic carcinoma.

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Case report
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A case report of a variant of Ivemark's syndrome

ABSTRACT

A newborn male with respiratory distress due to bilateral pneumothorax died at 6 hours of age. Autopsy revealed multiple pancreatic cysts, liver cysts with mild fibrosis, bilateral cystic and dysplastic kidneys, bullous change with hypoplastic lungs, and polysplenia. These findings could constitute a variant of Ivemark's syndrome: dysplasia of the pancreas, liver and kidneys.

KEYWORDS: Abnormalities, Multiple; Kidney, Polycystic; Pneumothorax; Pancreatic Cysts; Spleen + abnormalities

INTRODUCTION

Polycystic renal disease is real curiosity and exists frequently in association with malformation in various other organ systems - liver, pancreas, brain and heart. Our aim, in this work, is to describe and compare our case of Ivemark's syndrome, to other reported cases and to find peculiar differences between them.

CASE REPORT

Clinical history. A male child was born at term after a normal pregnancy and labor. The birth weight was 2650 g with crown-heel length (CR) 47 cm. The child was transferred from Gynecologic Clinic to Children Surgical Clinic because of severe respiratory distress due to bilateral pneumothorax.

Autopsy findings. Corps of mail child weighted about 2650 g; CR was 47 cm, biparietal diameter (BPD) 8 cm and foot length (FL) 7,8 cm. The skin of head and arms was livid, with Potter face. Thorax was cylindrical, and abdomen was in the same level as thorax.

Head. Fontanels were rhomboid and larger than usual. Parietal bones were connected by wide fibrous tissue of sagittal suture, and on the frontal bone existed frontal (metopic) suture. Leptomeninx was severe congested. Brain and spinal cord were grossly and microscopically normal.

Lungs. Visceral pleura showed few bullous changes of which some were intact and measure in diameter from 1 to 4 mm, but some other ruptured. Parenchyma of lungs was atelectatic, and mediastinum showed emphysema.

Liver was slightly larger but on gross examination showed usual appearance. In some parts, microscopically liver tissue showed proliferated and

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dilated bile ducts, with slight fibrosis of portal tracts.

Pancreas was firm, twice larger than normal, and showed small cysts about 1 - 2 mm in diameter. On microscopic examination it revealed cysticaly dilated ducts with parenchymal atrophy.

Kidneys. Kidneys were larger than normal and with deformed shape. Left kidney was twice larger than the right. The structure of kidneys was almost destroyed by the presence of multitude of cysts filled by serous yellowish fluid. Cysts were with smooth walls and measuring from 1 mm to 7 mm. Lower urinary tract was normal. Microscopically, cysts were lined by cuboidal epithelium, and surrounded by immature mesenchyme.

Miscellaneous findings. In oral cavity and lumen of stomach was present small amount of hemolysed blood. Spleen was larger than normal, and in its proximity were found two accessory spleens of which one was 1 mm and another was about 4 mm in diameter. Thymus was larger than normal but was microscopically normal.

DISCUSSION

The incidence of Ivemark's syndrome is very low. More than 200 cases where reported. Roth et al. (1993) report incidence in their department of 1 case of Ivemark's syndrome on 6000 deliveries. The etiology of this syndrome is not yet clearly defined. Some authors consider teratogenic effect, caused by exogen factors, which could occur between the 30th and 40th days (7,1) of intrauterine life, but precise etiology is unknown. Genetic cause has not been defined yet (1,6), but some authors have been proposed an autosomal recessive pattern of inheritance for renal-hepatic-pancreatic dysplasia (9). In our case, the parents are close siblings (fifth generation).

Malformations of the liver, pancreas, and other organs may variously accompany the different kinds of polycystic renal disease (3,4,6). To our knowledge, there is no identical case to originally Ivemark's. Some reported cases are similar, but differ in one or more aspects. In 1959 Ivemark, Oldfelt, and Zetterstrom described two siblings who died with renal failure several weeks after birth. These children had bilateral dysplastic kidneys, congenital hepatic fibrosis, and dysplasia of the pancreas without grossly visible cysts. Strayer and Kissane (1979) described one case of female newborn with liver containing large cysts, and fibrosis; kidneys with innumerable tiny cysts throughout; and finally a large solitary cyst of pancreas. At their opinion this case showed a closeness to Ivemark's syndrome. Our findings are closer to these in report of Ivemark et al. (1959). Osthanondh and Potter (1964) gave four types of polycystic kidneys. Type I, infantile polycystic kidney contains ectatic collecting tubules and is bilateral; the disease is inherited as autosomal recessive, and kidneys are usually several hundred grams in weight. Type II, dysplastic kidney, is characterized by collars of immature fibrous tissue, occasionally containing cartilage. This type may be unilateral or as well as bilateral, there is familial grouping of cases, and dysplasia may be associated with trisomias and other conditions. Type III is more familiar adult type of polycystic kidney, and the disease is inherited as an autosomal dominant. Type IV is associated with obstruction in the lower urinary tract, but is otherwise indistinguishable from dysplastic kidneys (type II). Changes found in Ivemark's syndrome could be consider as type II of polycystic kidneys, where are present dysplastic changes in kidneys, but genetically etiology is not precisely defined. Beside usual renal-hepatic-pancreatic dysplasia seen in Ivemarkís syndrome we find polysplenia, as is described in report of Torra et al (1996) and Bernard et al (1965), and additionally, tardive development of cranium (present frontal suture). Many polymalformation syndromes include cystic affectation of liver, pancreas, and kidneys, so this syndrome could be an isolated entity or a final common pathway of response of these organs to a variety of developmental disturbances, which could also include splenic abnormalities.

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Correlation of clinical and morphological parameters in patients with prostatic carcinoma

KEYWORDS: Prostatic Neoplasms; Prostate-Specific Antigen; Prostatic Diseases; Classification

The correlation between results of histopathological examination in patients with prostatic carcinoma (PC) and clinical parameters. 51 biopsy samples were analyzed. All samples were classified according to standard pathohistological classification (well, moderately and poorly differentiated PC) and three groups were formed. In each group Gleasonís grade and score, and presence of both prostatic intraepithelial neoplasia (PIN) and chronic prostatitis were evaluated. Clinical parameters (prostate specific antigen (PSA) serum level, complete urinary obstruction) were estimated in all groups and matched by age and established pathohistological degree. Well-differentiated PC was detected in 21/51 cases. It is equivalent to 5/21 cases in Gleason's grade 1, 13/21 in grade 2, and 3/21 in grade 3. Average PSA level is 12.54 ng/ml. PIN was established in 12/21 (57.14%) and chronic prostatitis in 12/21 (57.14%). The difference between clinical and pathohistological diagnosis was found in 9.52%. Moderately-differentiated PC was detected in 12/51 cases. It is equivalent to 4/12 cases in Gleason's grade 3, 8/12 in grade 4. Average PSA level is 98 ng/ml. PIN was established in 3/12 (25%) and chronic prostatitis in 7/12 (58.3%). The difference between clinical and pathohistological diagnosis was found in 8.34%. Poorly-differentiated PC was detected in 17/51 cases. It is equivalent to 4/17 cases in Gleason's grade 4 and 8/17 in grade 5. Average PSA level is 115,19 ng/ml. PIN was established in 1/17 (5.88%) and chronic prostatitis in 7/17 (41.17%). No difference between clinical and pathohistological diagnosis was found. The higher degree of correlation between pH and clinical parameters is found in moderately and poorly differentiated PC.

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p53 in benign prostatic hyperplasia and prostatic carcinoma

KEYWORDS: Prostate Neoplasms; Carcinoma; Prostatic Hyperplasia; Protein p53

The dramatic increase in the diagnosis of prostatic carcinoma in the past decade, has coaused a rise in the number of the investigations, on carcinogenesis and individual approach to the patients with prostatic carcinoma. This study was designed to evaluate the potential mutation in the P53 molecule. 50 cases including 25 PCa (Gleason sum 2-10), 16 BPH, 4 BCH and 5 AAH were immunostained using DAKO D0-7 primary antibody against the mutant form of 53. 8 cases of PCa with various Gleason sum were positive, including one case with positive limph node metastasis. 4 BCH samples were negative, and 16 BPH as well. One case out of 5 AAH was positive suggesting that AAH is one step in carcinogenesis. The results show that P53 expression is a potentiially useful prognostic factor and additional criterion in defining correct prognostic category in this molignancy. More resarch is needed to asses new biomarkers and, most importantly, to standardize the methodology for sampling and assaying biomarkers in heterogenous prostatic cancer.

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Treatment of end-stage kidney disease by kidney transplantation

KEYWORDS: Kidney Transplantation; Kidney Diseases; Kidney Failure, Chronic; Graft Rejection

Kidney transplantation is preferred mode of renal replacement therapy for virtually all patients with end-stage renal disease. During past decade, new discoveries and better understanding of transplant immunobiology have led to an improvement in patient and graft survival. The aim of our study was to present our four-year experience in kidney transplantation. A total of 56 patients (13 female and 43 male) were enrolled in this study. There were 46 living (45 living-related and 1 living-unrelated) and 10 cadaveric kidney transplantation. In two patients kidney transplantation has been performed before starting dialysis. In all patients the standard triple immunosuppressive therapy including steroids, cyclosporine and azathioprin or mycophenolate mofetil was used. In patients with high immunological risk and delayed graft function antithymocite globulin in the course of 7- 14 days was administered for rejection prophylaxis. In 3 (6.97%) patients graft loss was caused by vascular complications and in 1 (2.32%) by infective complication. During first post-transplant year acute rejection episode was noticed in 12 (23%) patients and in 4 (33.3%) it was steroid resistant. In none patients acute rejection was a cause of graft loss. In two patients graft loss was caused by recurrent glomerulonephritis and in one patients by chronic allograft nephropathy. One-year graft survival was 92,9% and patient suvival was 100%. Short term results in kidney transplantation are excellent and nowdays, due to improvement in immunosuppressive therapy, the success in kidney transplantation is mainly limited by surgical and infective complications.

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Incidental parasite finding in urinary bladder carcinoma

KEYWORDS: Bladder Neoplasms; Carcinoma, Transitional Cell; Bladder Fistula; Ascaris; Parasitic Diseases

The presence of parasites in some tumorous and nontumorous processes is not quite uncommon. With certain tumor types and localizations, a concomitant presence of a parasite is much more often registered, e.g. the concomitance of Schistosoma haematobium and the urinary bladder cancer, reported by Dimmette and coll. in even 55% of patients. The patient JM, born in 1948, with the symptoms of haematuria, was submitted to cystoscopy this year in January. The diagnosis of a transitional cell papillary invasive cancer of the urinary bladder, stage II was established. Due to the volume, localization and spread of the tumor, a total cystectomy was performed. Besides a moderately differentiated invasive transitional cell carcinoma of the urinary bladder confirmed in the samples sent for a histologic analysis, a necrotic cross-section of an intestinal worm -Ascaris was also identified in the tumor involved portion of the bladder wall, at the orifice to a fistulous fissure. An intestinal worm - Ascaris in the tumor involved wall of the bladder is considered an uncommon incidental finding. Unlike a well-established etiologic correlation of the tumor development in Schistosoma of the urinary bladder, the finding of Ascaris in our patient is probably incidental, due to Ascaris' migration from the intestinal tract to the lumen of the urinary bladder through a carcinomatous rectovesical fistula.