

indicates the incidence of individual concrements.

Oxalate concrements contain predominantly calcium oxalate. In the proper form they occur rarely. Commonly they contain also the salts of uric acid – the urates. They are hard and of whitish colour and of mulberry form. Oxalates are poorly soluble and can arise in acidic as well as in alkalic urine. Oxalate stones are formed in concentrated urine, during insufficient water supply, in urine stasis, and during dehydration. They arise only sporadically owing to the enhanced oxalate excretion in oxaluria – a seldom metabolic disturbance with deposits of oxalates in various tissues of other organs.

Urate concrements arise in supersaturation of urine by urates. The salts of uric acid are poorly soluble in urine with low values of pH. Their precipitation occurs therefore in acidic urine. Oxalate concrements may occur in hyperuricaemic syndrome and after consumption of food with high purine content. The idiopathic urate concrements arise however mostly either without observed increase of plasmatic uric acid concentration, or its enhanced urinary excretion. Urate lithiasis is encountered also in conditions with increased uric acid excretion due to tumorous diseases and cytostatic treatment polycythaemia.

Phosphate concrements contain magnesium ammonium phosphate. They occur during infections caused by urease producing microbes. This enzyme cleaving urea into CO₂ and NH₃ elevates the urine pH and enables the phosphate precipitation. Phosphate concrements are found also in occurrence of foreign matter (*corpus alienum*) in renal outflow tract.

Carbonate as well as oxalate stones occurrence appears in relation with calcium metabolism disorders. Elevated glomerular filtration of calcium due to hypercalcaemia enables the concrement formation.

Nephrocalcinosis may develop owing to the hypercalcaemia. Nephrocalcinosis is characterised by calcium salts deposition into the renal parenchyma, mainly in the papillary region. Hypercalcaemia with the consequent stone formation may appear also in increased calcium resorption from the gut and in enhanced calcium release from bones. According to the concrement location nephro- uretero- cysto- urethro- and prostatico-lithiasis may be distinguished.

4.16 Urinary outflow tract disturbances

After its excretion in the renal papillae is the urine transported by peristaltic contractions of renal calyces smooth muscles towards the urinary bladder. The lumen of renal pelvis and ureters is adjusted to some degree to the volume of urine. Single peristaltic waves with frequency of 2 to 5 per minute increase the pressure in ureters towards the urinary bladder. The peristaltic activity can be seen on roentgenograms as fusiform local dilations of ureters following contrast medium application. In children they can imitate even a pathologic dilation of ureters.

In acute inflammations of renal outflow tract without urine discharge disturbances, spastic constrictions of urinary "ways" with enhanced peristalsis can be observed. **In chronic inflammatory processes of renal outflow tract** are the urinary "ways" atonic. The smooth muscle action of renal pelvis, ureters and of urinary bladder is coordinated. Following transversal ureter cross-section a transient disorder of urinary outflow tract peristalsis usually occurs.

In complete acute obstruction of ureters, e.g. by ligation, the peristaltic movements disappear without dilation and pain and the urine production stops. The partial obstruction of ureters e.g. by a concrement causes, at the beginning, an elevation of the basal pressure in ureters and increase in peristaltic frequency with concomitant lowering of peristaltic amplitudes; subsequently the activity of ureters disappears. Colics occurring during this condition are not due to hyperperistalsis, but to the rise in ureter and renal pelvis tension. The dilatation of upper parts of urinary outflow tract occurs in longstanding incomplete obstruction.

Complete or partial obstruction of renal outflow tract leads to morphologic and functional alterations in kidneys - termed - obstructive nephropathy. The pressure in the renal outflow tract raises above the site of obstruction and is transferred to the tubules and glomeruli. The glomerular filtration rate falls and so does the renal blood flow. In obstructive nephropathy functions of distal tubules, the concentrating ability, the acidifying tubular activity and the

renal reactivity to antidiuretic hormone are mainly affected.

Azotaemia develops only in bilateral, more severe obstruction. Unilateral obstruction with intact contralateral kidney does not cause the retention of nitrogenous substances because of compensatory hypertrophy. **After the urinary outflow tract obstruction removal** a transient polyuria with subsequent dehydration and salts loss usually occurs. Polyuria reaches as much as 15 l daily and salt loss of 5 g daily. The underlying cause are: the endogenous osmotic diuresis, tubular irreactivity to antidiuretic hormone effects and specific disturbances of tubular sodium reabsorption.

When the renal outflow obstacle is early removed a complete recovery of renal functions can be expected in 10 to 20 days. Longer persisting obstructions result in irreversible lesions of renal parenchyma. The stasis of urine in dilated urinary collecting system enables arise of infections. Chronic bacterial infection usually results in interstitial nephritis.

Apart from the mechanic obstructions, **inherited dynamic disorders of miction** (urine discharge) occur, being due to a disproportion between the force of urinary collecting system smooth muscle contraction and the tonus of sphincters. Dynamic urine outflow disturbances are encountered e.g. in congenital nephroses with spastic constriction of ureteral upper orifice and subsequent pelvis dilatation. Another example is the so-called megaureter caused by spastic constriction of some part of the ureter. Inherited neuromuscular dysplasia is considered to be the cause of these dynamic disorders of urine discharge.

The dilation of upper parts of urinary collecting system affects about one third of pregnant women. Functional alterations of renal outflow system during pregnancy are due to hormonal effects, to the ureter compression by enlarged uterus, or to neuromuscular disturbances of outflow ducts during infections.

In the pathogenesis of pyelonephritis, above all, has the **reflux of urine** a great importance. Every retrograde urine flow should be considered to be a pathological phenomenon. The so-called primary reflux of urine is thought to be an inborn valve mechanism disorder in the region of ureter orifices. **The secondary reflux** occurs commonly in congenital disturbances of urine discharge. It can be due to the valve presence in ureters, to fibroelastose of the urinary bladder orifice, or eventually to the spinal cord

lesions. Reflux of urine occurs also in central nervous system lesions. Severe inflammatory of the urinary collecting system alterations with subsequent fibrosis, e.g. in tuberculosis of collecting system result in the loss of their motoric function. The urinary "ways" are changed into rigid tubes without any peristaltic movement and antireflux function. In children the vesicoureteral reflux occurs commonly in renal outflow tract infections, where an organic cause always should be considered. Each reflux retards or even abolishes the peristaltic movements of urinary "ways" and the retrograde overpressure with severe consequences in renal parenchyma.

A special group of urinary tract disturbances comprises **disorders of urinary bladder discharge**, the congenital disorders of miction, above all. The underlying cause is the functional insufficiency of urinary bladder smooth muscles. This group includes the so-called myogenic atony, or the achalasia of sphincter, a condition with increased tonus of sphincter and failure to relax. Owing to the hypotony and decreased contractility of urinary bladder muscles the bilateral vesicoureteral reflux arises: the syndrome of megaureter megacystis occurring in central nervous system lesions. If the urinary bladder sphincter is hypertonic the bladder muscles become hypertrophic. In the urinary bladder failing to discharge completely residual urine remains.

The underlying causes of acquired miction disorders are commonly the hypertrophy of prostata, carcinoma prostatae, strictures in urinary collecting system or acquired hypertony of urinary bladder sphincter. In these cases the vesicoureteral reflux is not observed. The basal tonus of ureters is usually normal, the ureter peristalsis however, is reduced.

Disorders of miction are frequently due to various central nervous system diseases. In this group are included: urine incontinence, urine retention (inability to discharge the urinary bladder in response to an appropriate, normal stimulus). In these neurogenic disorders of miction is the balance between the sphincter tonus and urinary bladder muscle contractility impaired.

Transverse lesion of spinal cord induces complete urinary bladder atony with inability to discharge. The so-called upper lesion (above the sacral medulla) leads to urinary bladder automatism. The urinary bladder filling elicits stimulus spreading towards the medullary centre. About 100 ml of residual urine

remain in bladder. In so-called lower lesion (below the 11th thoracic vertebra) are the nervous pathways in sacral medulla eliminated and so are the afferent pathways leading from urinary bladder, and the medullar centre with efferent pathways.

In the state of automatism is the urinary bladder atonic. Irregular contractions of m. detrusor single fibres appear, induced by stimuli arising from intramural ganglia. The urinary bladder can be evacuated only by use of abdominal press and the volume of residual urine exceeds 300 ml.

4.17 Tumors of urinary tract

Benign renal tumors originate in parenchyma and are usually small. The adenomas reach 5 to 10 mm in diameter. They can cause recurrent haematuria without pains. Malignant transformation of adenomas is exceptional. The hamartomas can be uni- or bilateral. They contain vessels, adipose tissue, smooth muscle. Benign tumors may arise also from capillaries adjoining the juxta-glomerular apparatus. They are producing renin.

Malignant renal tumor (adenocarcinoma, hypernephroma) becomes clinically manifest by haematuria, lumbar pains and palpable resistance. The most important symptom is the haematuria. Tumor penetrates the renal capsule and renal veins, therefore haematuria can be very dramatical. It may spread into the peritoneal lymphatic tissue and liver. Metastases are found mainly in lungs, brain and bones.

Intermittent fever may occur without overt infection. Anaemia is present in about 50 per cent of patients. Occurrence of polyglobulia is rather exceptional. It is induced by erythropoietin produced in tumor cells. It is very important to realise that the renal carcinomas can produce many various hormones or hormone-like substances. It is frequently the parathyroidal hormone, prolactin, prostaglandins, gonadotropins and glucocorticoids. Thus, it is not surprising, when a condition develops with dominant Cushing's syndrome.

In kidney arterio-venous fistulae may arise, which can create conditions for heart failure with increased minute cardiac volume. Tumor may occlude v. cava; induce severe liver deterioration and changes in blood flow through the splanchnic region.

In children occurs malignant nephroblastoma (Wilms' tumor). It is composed of various types of cells: epithelial, muscle-, cartilage-, bone cells. It grows to large dimensions and occurs bilaterally. The most frequent symptoms are haematuria, pains, fever and hypertension.

Except for kidneys, tumors of urinary tract can arise from renal pelves, ureters and urinary bladder. Tumors of the urinary bladder prevail. The tumors of renal pelvis and ureters use to be accompanied with renal colics. The carcinomas of urinary bladder are manifested by painless haematuria, nycturia and sacral pains.

Carcinoma of prostata can stay long time asymptomatic. Its incidence raises. The prognosis is favorable if the extent of the tumor is small. This type of tumor penetrates later the lymphatic nodes, bones and lungs.

Benign hypertrophy of prostata can lead to alteration due to partial obstruction of urinary collecting system.