

sium, calcium, phosphates, bicarbonates, uric acid and proteins. The impaired transport may lead to aminoaciduria, glycosuria, salt wast, hypercalciuria, hypophosphataemia, proximal tubular acidosis and tubular proteinuria. Inherited disturbance occurs commonly. Similar condition may develop in adults owing to the renal disorders and dysproteinaemia. In Wilsons type amyloidosis and in Love's type a complex of occulo-cerebro-renal alterations occur.

4.15 Urolithiasis

Urolithiasis is a relative common disease occurring in about 1 per cent of inhabitants, mainly in men. In one tenth of cases is the nephrolithiasis diagnosed during their life. In remaining cases is the nephrolithiasis asymptomatic.

The calcium salts are the basis of urinary concrements. 75 to 85 per cent of urinary calculi contain calcium oxalate and calcium phosphate. Apart from the calcium salts the uric acid and cystine contribute to the concrement formation. Uric calculi occur commonly in patients with hyperuricaemia. Magnesium- ammonium- phosphatic concrements appear during urinary tract infections. Urinary calculi are formed when the balance becomes impaired between the water volume and excreted crystal forming material. Under physiological circumstances the calcium salts do not form crystals. If urine is supersaturated with calcium salts the crystallization can start. The supersaturation of urine is considered to be the main mechanism responsible for concrement formation. Another important factor is the pH of urine. Alkaline urine contains more urates and dissociated phosphates. The calcium oxalate solubility however, does not depend on pH of urine. The third important factor is the dehydration, and the fourth is the overloading of organism with some substance which could become the basis for stone forming.

Kidneys release several lyotropic protective substances preventing the stone formation: citric acid, glucuronic acid, magnesium, glycine and other substances. The concentration of all substances impor-

tant for concrement formation in urine depends on water rediffusion in tubuli and on filtered amount of these substances. Thus, increased diuresis reduces the concentration of stone-forming substances in urine. Among further important factors belongs urine stasis, commonly accompanied with infections of urinary outflow tract, enhanced excretion of stone-forming substances and changes of urine colloidal qualities. The stasis of urine and the concomitant infection create favorable conditions for formation of concrements. The calculi commonly originate by a process of crystal nucleation in tubules, or in bends of renal calyces. The crystal nucleation occurs frequently in renal papillae, but they can be formed also in renal pelvis, urinary bladder or elsewhere. The changes in excretion of stone-forming substances appear in metabolic disorders or in primary tubular disturbance in reabsorption of stone-forming substances. Supersaturation of urine with stone-forming substance constitute favorable conditions for crystal nucleation. Further layers of the concrement are formed around it by apposition of stone-forming salts and organic substances.

The hypothesis of colloid protection significance supposes an imbalance between mineral substances and organic substances, termed protective colloids in urine. When the balance has been disturbed by an excess of mineral substances or by deficiency of protective colloids the concrements arise.

The mucoprotein occurring in urine has some qualities identical with the mucoprotein in bone. The origin of urinary mucoproteins is unknown. They can be products of tubular cells or of the substance occurring among the epithelial cells. The mucoprotein, at last could originate in basal osseous mass. Following the parathyroid hormone application depolymerisation of bone mucoproteins occurs. The products of depolymerisation are subsequently filtered in glomeruli and reabsorbed in proximal tubules Calcium binds with mucoprotein forming microlith, which can pass the renal outflow tract. This mechanism may explain the concrement formation during hyperparathyroidism.

The pathogenesis of urolithiasis is not well understood. The mechanism of concrement formation might not be the same for each type of stone. According their composition the concrements are divided into oxalate-, urate-, phosphate- carbonate-, xantine and cystine stones. The succession in the list

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