Urinary disorders

1. The most common route of renal interstitial infections is:
    1. Via adjacent tissues
    2. Haematogenic
    3. Lymphogenic
    4. **Ascending (through the urinary tract)**
    5. Descending from the glomeruli
    6. Always a combination of at least two of the above.

2. The chronification and progression of pyelonephritis is associated with:
    1. The impeded activity of macrophages in a hyperosmotic environment
    2. The formation of highly resistant protoplast (L) forms of bacteria
    3. Primary immunodeficiency
    4. **1, 2**
    5. 1, 2, 3.

3. Pyelonephritis is best defined as:
    1. Diffuse renoparenchimal dystrophy
    2. **Non-specific tubulointerstitial bacterial inflammation**
    3. Tubulo-renal virus lesion
    4. Reno-interstitial immune conflict
    5. Specific reno-parenchymal process

4. The urinary tract reflux is a mechanism, associated with the pathogenesis of:
    1. Polycystic kidney disease
    2. Nephrolithiasis
    3. Glomerulonephritis
    4. Wilms‘ Tumor
    5. **Pyelonephritis**

5. Gram-negative bacteria are the most common etiology of pyelonephritis:
    1. They predominate in the urinary tract
    2. They bind easier to the epithelium of the urinary tract
    3. They easily reproduce in the primary urine
    4. **1,2**
    5. 1, 2, 3.

6.The main pathogenetic unit for the development of diffuse glomerulonephritis is:

    1. Acute intoxication
    2. Idiopathic sclerosis

3. **Immune inflammation**
4. Glomerular capillary thrombosis
5. Virus inflammation

7.Which of the following bacterial antigens are markedly nephritogenic:

1. Haemophilus influenzae
2. **Beta-haemolytic streptococcus group A**
3. Pneumococcus
4. Escherichia coli
5. Proteus

8.The immune lesion in immune complex glomerulonephritis is a result of:

1. Degranulation of mast cells and basophiles
2. Isolated T-killer cells activity
3. Single NK-cell stimulation
4. **Antibody-dependent cell mediated and/or complement dependent cell mediated cytotoxicity**
5. Thrombocyte adhesion

9. In glomerulonephritis the immunologic conflict is a result of:

1. In-situ formed immune complexes in the glomeruli
2. Deposition of soluble circulating immune complexes in the glomerulus
3. Renal type of Arthus phenomenon
4. 1,3
5. **1,2**

10. Point out the representation of renal tubular dysfunction:

1. Disturbed mechanisms of urinary concentration and dilution
2. Hyperazotemia due to retention
3. Renal hypertension and/or anemia
4. Renal polyuria and tubular acidosis
5. **1, 4**
6. 1, 2, 3

11. The pathogenesis of nephritic edemas in the acute phase is:

1. Hypoproteinemia
2. Increased permeability (membranogenic)
3. Lymphostasis and/or primary hyperaldosteronism
4. Primary (glomerular) hypervolemia
5. **2, 4**
6. 1, 2, 3

12. The main pathogenetic unit of nephrotic edema is:

1. Increased capillary permeability

2. **Decreased plasma oncotic pressure**
3. A block in the lymph drainage
4. Increased capillary hydrostatic pressure
5. Primary NaCl retention

13. Point out the specific pathophysiological representation of the nephrotic syndrome:

1. **Massive proteinuria, hyperlipidemia, hypoproteinemia and edema**
2. Arterial hypertension, hematuria, oligo- and anuria
3. Hyperlipidemia, hypoproteinemia, hematuria
4. Microhematuria, pyuria, cristaluria
5. Pollakiuria, hyperproteinuria, arterial hypotension

14. The supression in erythropoesis in renal failure is mainly due to:

1. **Decreased secretion of renal erythropoetic factor**
2. Microangiopathic hemolysis
3. Decreased activity of erythropoetin in the bone marrow
4. Relative deficit and decreased utilization of iron
5. Accumulation of erythropoesis inhibitors

15. Which changes in the urine are indicative for chronic renal failure:

1. Disuria and pollakiuria
2. Oliguria and hypersthenuria
3. Hematuria
4. Varying massive proteinuria
5. **Polyuria and isosthenuria**

16. Uremic intoxication leads to:

1. Increased permeability of barriers
2. Cellular membrane functional lesions with ion asymetry
3. Sepsis
4. **1, 2**
5. 1, 2, 3

17. The most important pathophysiological representation of uremic intoxication is:

1. **Encephalopathy**
2. Hemorrhagic diathesis
3. Normocytic anemia
4. Peripheral neuropathy
5. The disappearance of polyuria

18. Uremic encephalopathy is associated with:

1. Increased permeability of the hematoencephalic barrier
2. Continuous activation of the reticular formation
3. Deficit of neurotransmitters

4. Neuronal bioelectric destabilization
5. 1, 2, 3
6. **1, 3, 4**

19. In chronic renal faiure the remaining glomeruli compensate decreased glomerular filtration by:

1. **Increased glomerular filtrartion per single functional glomerulus**
2. Generation of new glomeruli
3. Supression of periglomerular lymph drainage
4. Severe increase of glomerular membrane permeability
5. Phenomenon of podocyte injury

20. In chronic kidney failure the tubules of the intact nephrons compensate the nephrone deficit by:

1. Increased number of cells in the tubule
2. A connection of several tubules with one glomerulus
3. Increase of the reabsorption and secretion capabilities of the single tubule cell
4. 2, 3
5. **1, 3**

21. The main pathophysiologic presentation of oligoanuric stage in acute renal failure is:

1. Progressive azotemia, hyperhydration
2. Movement of intracellular ions into the circulation and of extracellular into the cells
3. Blockage of the albumin synthesis in the liver
4. Fatigue of the sympathetic-adrenal system
5. **1, 2**
6. 2, 3, 4

22. Which is the most characteristic (pathognomonic) stage of acute renal failure:

1. Shock, septic, toxic
2. **Oliguanuric**
3. Polyuric
4. Isostenuria
5. Dehydratation

23. Which of the following has a leading role for the development of renal failure:

1. Overall disturbance of vital functions
2. **Severe deficit of nephrons**
3. The urinary syndrome
4. Nitrogen retention
5. 1,4

24. Acute renal failure is a presentation of:

1. Destroyed nephrons

2. Ineffective nephrons
3. **Functionally switched-off nephrons**
4. Genetically insufficient nephrones
5. Chronic hypoperfusion of nephrones

25. Which are the pre-renal causes for the development of acute renal failure:

1. **Shock, hemolysis, dehydration**
2. Intoxications with heavy metal salts
3. Urinary tract obstruction
4. Ureteral stricture
5. Acute pyelonephritis

26. Which are the renal causes, leading to acute renal failure:

1. Ileus, acute pancreatitis, peritonitis
2. **Acute glomerulonephritis and pyelonrphritis**
3. Acute abnormalities in the acid-base balance
4. Prostate hypertrophy
5. Burns

27. Glomerular mechanisms of acute renal failure are:

1. Renal interstitial edema
2. Afferent arteriolar spasm
3. Efferent arteriolar dilation
4. Decreased permeability of glomerular basal membrane
5. 1, 2, 3
6. **2, 3, 4**

28. Osmotically dependent renal polyuria develops in cases of:

1. Supressed reabsorption of Na^+/Cl^-
2. Decreased levels and/ or activity of ADH
3. Genetic deficit of aquaphores
4. Increased excretion of glucose, urea, etc.
5. **1,4**
6. 1, 2, 3, 4

29. The main mechanism, disturbing urinary concentration and dilution in the course of acute renal failure is:

1. Inability to establish a corticomedullary osmotic gradient
2. Inability to utilize the gradient
3. Augmented „wash-out“ of the gradient
4. Decreased secretion of ADH
5. **1, 2, 3**

6. 1, 2, 3, 4

30. During the polyuric stage of acute renal failure there is a risk for:

1. Disturbed metabolism, due to the fast nitrogen clearance of the organism
2. **Hypokalemia, hyponatremia and dehydration**
3. Hypervolemia, heart failure
4. Tubular necrosis and rrhexis
5. Nonselective proteinuria, hypoproteinemia

31. The uremic stage of the chronic renal failure develops when :

1. **90% of nephrones are not functioning**
2. 80% of nephrones are not functioning
3. 75% of nephrones are not functioning
4. 70% of nephrones are not functioning
5. 100% of nephrones are not functioning

32. The main pathogenetic unit for the development of proteinuria is:

1. A primary supression of proximal reabsorption of proteins
2. Increased secretion of proteins in the tubules
3. Decreased excretion of proteins with lymphatic drainage
4. **Increased permeability of glomeruli for proteins**
5. Decreased mesangial phagocyte activity