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Question

Discuss the diseases of the renal system

Renal system disease, any of the diseases or disorders that affect the human urinary system. They include benign and malignant tumours, infections and inflammations, and obstruction by calculi.

Diseases can have an impact on the elimination of wastes and on the conservation of an appropriate amount and quality of body fluid. Many of the manifestations of renal disease can be accounted for in terms of disturbance of these two functions, and the alleviation of symptoms in those renal diseases that cannot be cured depends on knowledge of how these two functions are affected.

The eliminatory process does not, end with the formation of urine; the urine has to pass down the ureters to the bladder, be stored there, and voided, usually under voluntary control. The whole mechanism can be deranged by structural changes in the lower urinary tract, by infection, or by neurological disorders that lead to abnormal emptying of the bladder. Disturbance of the lower urinary tract is an important cause of pain and distress, notably during pregnancy and in the elderly; and it can lead to serious and progressive damage to the kidneys, either by interfering with the drainage of urine or by allowing bacterial infection to have access to the kidney.

Bacteria such as Pseudomonas aeruginosa can cause infections of the urethra and bladder. These infections occur more often in women than in men and are typically associated with significant pain and distress.

Functional Aspects

Effects of abnormal renal function on body fluid

Renal disease in its diverse forms can lead to bodily deficits or excesses of water, sodium, potassium, and magnesium, and also to protein deficits occasioned by great losses of protein in the urine. Inability of the kidney to function normally may lead to retention in the blood of the waste products of protein metabolism, such as urea and uric acid, and of other nitrogenous compounds such as creatinine. There may be abnormally high levels of phosphates in the blood, which in turn can lead (for reasons about which there is still some disagreement) to low blood levels of calcium. The calcium deficiency can cause tetany, a condition marked by muscular spasms and pain, and calcium may be lost from the bones in the process of restoring normal calcium levels in the blood and tissue fluid. For descriptive purposes, changes in volume, changes in composition, and protein depletion of renal origin will be discussed separately, but these disturbances can and often do coexist.

Though body fluid is most readily apparent in the bloodstream, it is present, and in larger amounts, in the tissues, both between the cells (interstitial fluid) and within them (intracellular fluid). Extracellular fluids, which include interstitial fluid and blood plasma, amount to 25 percent of body weight and contain sodium as their predominant cation (positive ion; metals and hydrogen in solution are cations). Intracellular fluids, amounting to 33 percent of body weight, have potassium as their predominant cation. These various “compartments” of body fluid are in osmotic equilibrium, so that if solute (e.g., sodium chloride) is added to the extracellular compartment so as to increase the concentration of the extracellular solution, water will join it to reduce the concentration, and that compartment will increase. An increase in extracellular fluid, if it is considerable, may be clinically apparent as edema, a swelling of the tissues by fluid, which can usually be displaced by firm pressure. Edema is present in acute inflammation of the kidney (nephritis), in protein deficiency of renal origin, and in chronic nephritis complicated by heart failure associated with abnormally high blood pressure; a factor common to all these states is failure of the kidneys to excrete sodium and water in adequate amounts.

The kidneys in such edematous states need not themselves be diseased; for example, normal kidneys, in a patient with heart failure, may retain sodium when handicapped in their function by poor circulation and by abnormal amounts of sodium-retaining hormones, such as aldosterone. Increase in extracellular fluids is the only volume change that is both common and easily discernible in renal disease, but the opposite condition, sodium depletion or clinical dehydration, is more commonly the result of vomiting and diarrhea when they are complications of terminal renal disease. Sodium and water depletion can be recognized by a lack of elasticity in the superficial tissues and by poor filling of the blood vessels, as well as by signs of impaired circulation, including a fall in blood pressure and an increase in pulse rate. Though changes in intracellular fluid volume occur in some diseases, especially when the potassium content of the body is affected, there is no easy way of detecting them.

Apart from these general changes in body fluid, the pattern of individual constituents can be distorted in renal disease. For many substances, the problem is one of failure of excretion, with consequent increased concentration in body fluids. Insofar as excretion is achieved by filtration, the rise in concentration may assist excretion, permitting prolonged states of balance, at the cost of increased, but often tolerable, levels of concentration. For example, an individual in renal failure must put out as much urea as a healthy individual taking the same diet; but that person can only do so at a blood-urea concentration of 100 milligrams per 100 millilitres, instead of a normal blood-urea of 25 milligrams per 100 millilitres. Substances whose concentration increases in this way include urea, creatinine, uric acid, phosphate, sulfate, urochrome, and indeed all the usual constituents of urine apart from those that are "regulated" rather than simply "excreted." Potassium should be mentioned because of the special danger associated with its retention, which can lead to fatal irregularity of cardiac action. This is a recognized danger of acute renal failure, now commonly prevented by use of the artificial kidney and its semipermeable membranes, and sometimes by the use of resins that will take up potassium in the alimentary tract.

Disorders of urine flow

If little or no urine appears, it may be because the kidneys are forming little urine (oliguria) or none (anuria); or it may represent a holdup in the bladder or urethra affecting the outflow from both kidneys. About one person in 500 is born with only one kidney, and loss of a kidney from disease or accident is not rare. The loss of a single kidney does not substantially affect an individual's ability to eliminate wastes, as long as the other kidney functions normally. In cases in which complete obstruction of the remaining ureter occurs, patients will experience effects similar to obstruction of the entire lower urinary tract. Partial or complete failure to form urine is treated in the section on acute renal failure, obstructive conditions in the section on diseases of the urinary tract.

In instances of damage to nervous control, certain typical clinical situations may be differentiated, corresponding to different modes of disordered urinary flow:

(1) Lack of conscious inhibition of micturition because of damage to the cerebral cortex or, more commonly, from psychological causes results in a need to micturate that cannot be suppressed even though the bladder volume may be quite small; micturition is precipitate and continues until the bladder is empty.

(2) Transverse lesions or other damage to the spinal cord above the sacral reflex centres that cause paralysis of the lower half of the body produce at first a bladder that is atonic (lacking in

physiological tone). This bladder becomes greatly distended; the detrusor relaxes and reflex micturition is abolished. Pressure finally rises sufficiently to overcome the spasm of the sphincters and urine is voided in small amounts. Further accumulation and partial voiding of the overflow recur (overflow incontinence). Under these conditions the bladder readily becomes inflamed, which may cause disability or death from chronic ascending urinary infection. Intermittent drainage of the bladder with a catheter may be necessary, or firm pressure on the lower abdominal wall may be used to avoid overdistension and to develop an "automatic" bladder after some time. This is a small capacity organ (around 150 millilitres) with frequent emptying; there is reflex control mediated through the sacral segments of the spinal cord; the higher centres do not restrain the detrusor, and the internal sphincter relaxes more readily. Voluntary assistance from the abdominal muscles helps in this situation if these too have not been paralyzed. There is, however, always some residual urine from incomplete emptying and a risk of infection. In some cases, pressure building within the bladder can be transmitted to the kidneys; without medications or more frequent bladder emptying to relieve the pressure, the kidneys will incur damage.

(3) In contrast, there is the isolated, or "autonomous," bladder resulting from damage to the central nervous system below the sacral cord reflex centres or to the nerves supplying the bladder and urethra. The bladder becomes tense but contracts only weakly so that, while small amounts of urine are voided, the residual urine may be as high as 200–300 millilitres. This condition is known as active incontinence as opposed to the overflow incontinence of the automatic bladder. Here again, more effective emptying of the bladder by catheter drainage may be helpful.

Pain associated with urination (dysuria) can arise from bladder distension, which is then relieved by effective micturition; from inflammation of the lower urinary tract, commonly due to infection but rarely caused by chemical irritants in the urine; and from mechanical irritation by tumour or during the passage of stones. Dysuria is commonly, but not necessarily, associated with frequency of urination. This in turn may represent either an irritable or contracted bladder; or the actual amount of urine formed may be unusually large (polyuria), in which case voiding is likely to be painless. Sometimes polyuria may not be noticed by day but may manifest itself in the need to micturate on several occasions during the night (nocturia). The acute onset of dysuria and frequency suggests urinary infection; sustained polyuria is more likely to be due to renal failure (defective concentrating power) or to diabetes. In those who drink beverages into the night, nocturia is physiological.

Incontinence, the involuntary passage of urine (or feces), may be due to a faulty nerve supply, which either leaves the sphincters relaxed or allows them to be overcome by distension of the bladder. Comatose and disturbed patients, especially among the elderly, are commonly incontinent. Apart from nerve lesions, the sphincters that normally prevent the escape of urine may be damaged by repeated childbirth, by the growth of the prostate, or by other distortions of the bladder neck. Medications to relax the bladder and increase its capacity may be helpful. Alternately, more complete bladder emptying by intermittent catheterization may limit the amount of urine leakage. Procedures have been devised to stimulate the sphincters electrically, when their nerve supply is damaged; or to stimulate the bladder to empty itself at set times. For

chronic incontinence, however, devices to catch the urine and prevent soiling of clothing are the most practical.

RENAL FAILURE

Renal failure refers to failure of excretory functions of kidney. It is usually, characterized by decrease in glomerular filtration rate (GFR). So GFR is considered as the best index of renal failure. However, decrease in GFR is not affected much during the initial stages of renal failure. If 50% of the nephrons are affected, GFR decreases only by 20% to 30%. It is because of the compensatory mechanism by the unaffected nephrons.

The renal failure may be either acute or chronic. Renal failure is always accompanied by other complications such as:

1. Deficiency of calcitriol (activated vitamin D) resulting in reduction of calcium absorption from intestine and hypocalcemia (Chapter 72). Deficiency of calcitriol and hypocalcemia may cause secondary hyperparathyroidism in some patients
2. Deficiency of erythropoietin resulting in anemia
3. Disturbances in acidbase balance.

ACUTE RENAL FAILURE

Acute renal failure is the abrupt or sudden stoppage of renal functions. It is often reversible within few days to few weeks. Acute renal failure may result in sudden **life-threatening reactions** in the body with the need for EMERGENCY TREATMENT **CAUSES**

1. Acute **nephritis** (inflammation of kidneys), which usually develops by immune reaction
2. Damage of renal tissues by poisons like lead, mercury and carbon tetrachloride
3. **Renal ischemia,** which develops during circulatory shock
4. Acute **tubular necrosis** (necrosis of tubular cells in kidney) caused by burns, hemorrhage, snake bite, toxins (like insecticides, heavy metals and carbon tetrachloride) and drugs (like diuretics, aminoglycosides and platinum derivatives)
5. Severe **transfusion reactions**
6. Sudden fall in blood pressure during hemorrhage, diarrhea, severe burns and cholera
7. Blockage of ureter due to the formation of calculi (renal stone) or tumor.

FEATURES

1. **Oliguria** (decreased urinary output)

2. **Anuria** (cessation of urine formation) in severe cases

3. **Proteinuria** (appearance of proteins in urine) including albuminuria (excretion of albumin in urine)

4. **Hematuria** (presence of blood in urine)

5. **Edema** due to increased volume of extracellular fluid (ECF) caused by retention of sodium and water

6. **Hypertension** within few days because of increased ECF volume

7. **Acidosis** due to the retention of metabolic end products

8. **Coma** due to severe acidosis (if the patient is not
treated in time) resulting in death within 10 to 14 days.

CHRONIC RENAL FAILURE

Chronic renal failure is the progressive, long standing and irreversible impairment of renal functions. When some of the nephrons loose the function, the unaffected nephrons can compensate it. However, when more and more nephrons start losing the function over the months or years, the compensatory mechanism fails

CAUSES

1. Chronic nephritis
2. Polycystic kidney disease
3. Renal calculi (kidney stones)
4. Urethral constriction
5. Hypertension
6. Atherosclerosis
7. Tuberculosis
8. Slow poisoning by drugs or metals.

„ **FEATURES**

1. *Uremia*

Uremia is the condition characterized by excess accumulation of end products of protein metabolism such as urea, nitrogen and creatinine in blood. There is also accumulation of some toxic substances like organic acids and phenols. Uremia occurs because of the failure of kidney to excrete the metabolic end products and toxic substances.

Common features of uremia

i. Anorexia (loss of appetite)

ii. Lethargy

iii. Drowsiness

iv. Nausea and vomiting

v. Pigmentation of skin

vi. Muscular twitching, tetany and convulsion

vii. Confusion and mental deterioration

viii. Coma.

2. *Acidosis*

Uremia results in acidosis, which leads to coma and death.

3. *Edema*

Failure of kidney to excrete sodium and electrolytes causes increase in extracellular fluid volume resulting in development of edema.

4. *Blood Loss*

Gastrointestinal bleeding accompanied by platelet dysfunction leads to heavy loss of blood.

5. *Anemia*

Since, erythropoietin is not secreted in the kidney during renal failure, the production of RBC decreases resulting in normocytic normochromic anemia.

6. *Hyperparathyroidism*

Secondary hyperparathyroidism is developed due to the deficiency of calcitriol (1,25dihydroxycholecalciferol).

It increases the removal of calcium from bones resulting in **osteomalacia.**