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Review Article

Risk in Renal Disorders: A Review

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Abstract

Kidney disease or dysfunction can be due to numerous causes or factors, such as acquired infections, congenital pathologies, or drug abuse. There are multiple complications that can occur in patients with kidney damage and dental procedures must be very careful when paying attention to these patients. Therefore, an exhaustive bibliographical review was carried out about the main risks and complications in kidney disease.

Keywords: Kidney Disease; Surgical Risk; Kidney Disease

Introduction

This chapter will address medical-stomatological aspects of great interest to all dentistry professionals and students, who in their daily work provide assistance to patients with kidney disorders. Currently there is a not inconsiderable number of patients with dissimilar changes in the kidneys, so it is important to delve into the generalities of this organ, so important and essential for the life of human beings, as well as aspects directly linked to the different procedures. stomatological.

This section compiles knowledge ranging from basic sciences to clinical practice. Generalities. The kidneys are two retroperitoneal organs located in the posterior part of the abdomen, on either side of the spinal column. In humans, the upper pole of each of them is at the level of the twelfth thoracic vertebra and the lower one at the level of the third lumbar.

The right kidney usually occupies a more caudal position. On its medial or concave face it presents a slit, called the hilum, through which the pelvis, the renal artery and vein, the lymphatic vessels and the nervous plexus pass into the renal sinus. Irrigation is carried out by the renal artery, it penetrates the hilar region and divides into two branches, the anterior and posterior branches. It has two distinct regions: the cortex (external) and the medulla (internal). In the human being, two or three invaginations, the major calyces, extend outward from its dilated upper end. From each of them arise several smaller calyces directed towards the papillae of the pyramids and drain the urine formed in each pyramidal unit. The lymphatic vessels leave the kidney through two different lymphatic networks: a superficial capsular system and a deeper hilar system. They surround the renal arteries and are distributed mainly through the cortex, following the interlobular and arcuate arteries. The interstitium consists of interstitial cells and a loose and flocculated extracellular matrix-forming substance consisting of sulfated and nonsulfated glycosaminoglycan [1,2].

Objective

Deepen and discuss the main risks of kidney disorder in dental treatments.

Analysis Strategy

The search was based solely on kidney disease conditions of the buccomaxillofacial complex.

Developing

Kidney functions

This organ constitutes the fundamental pillar to maintain adequate homeostasis of body fluids, due to its efficiency in purifying dissimilar substances in the blood plasma. Said function is directly linked to the effectiveness of controlling and regulating the concentration of water, the composition of inorganic ions, and maintaining the acid-base balance.

- Regulate osmolarity and volume of body fluids Regulate normal cell volume of all tissues Quantitatively regulates various inorganic ions electrolyte balance acid-base balance.
- They help regulate the pH of the entire human body.
- Excretion of metabolic products and harmful substances (urea, uric acid, creatinine)
- Eliminate drugs and chemicals hemoglobin metabolism Hormone metabolites production and secretion of hormones
- Degradation of other hormones such as insulin or parathyroid hormone
- Hematopoietic function (erythropoietin) Close relationship with blood pressure Influence cardiac output They purify the plasma

- Synergy with other organ systems (cardiovascular, respiratory, digestive, central nervous system, endocrine)
- Controls the body's water volume
- Maintain proper blood circulation
- Selective purification that avoids the loss of necessary elements for the organism.

When for any reason some of these functions are affected, renal disorders also called nephropathies or kidney diseases occur. The damage can be temporary or permanent, resulting in the loss of normal kidney function. At present, the classification of most nephropathy continues to be based on the nature of the initial lesion that affects the renal parenchyma: the glomeruli, the tubules, and the interstitium or vessels. According to their evolution, nephropathies are classified as acute or chronic. If it is due to an identified disease, it is said to be secondary to [1,3].

When their cause remains unknown, they are said to be primary or idiopathic. glomerular filtration It is an index of kidney function. Its decrease normally means the progression of a nephropathy, while its recovery generally implies a recovery. Therefore, knowledge of the glomerular filtration rate (GFR) is essential to assess the severity and evolution of a renal process.

Creatinine is an intermediate product in skeletal muscle creatinine metabolism, and it can be used to measure GFR. It is freely filtered through the glomerulus into Bowman's space and, to a first approximation, it is not reabsorbed, secreted, or metabolized in nephron cells. Therefore, the amount of creatinine excreted in the urine per minute is equal to the amount filtered by the glomerulus in the same time.

Therefore, creatinine clearance provides a means for the determination of GFR. Clinically, creatinine is used to calculate the GFR, it is synthesized almost constantly and the amount produced is proportional to muscle mass. Not all of the creatinine that enters the kidney with the renal arterial plasma is filtered in the glomerulus. Similarly, not all the plasma that reaches the kidney is filtered. Although almost all of the plasma that arrives with the renal artery passes through the glomerulus, approximately 10% does not.

The GFR ranges from 90 to 140 ml/min and in women from 80 to 125 ml/min. This means that during 24 hours 180 L of plasma will have been filtered by the glomerulus, this ultrafiltrate lacks formed elements and also some proteins, this ultrafiltration is driven by Starling force through the glomerular capillaries and its changes modify the VFG. Renal disease Renal disease is considered when any of the morphophysiological elements of the kidneys are affected, mainly as a result of the deterioration and destruction of the nephrons that, once lost, will never be recovered. It is classified into acute and chronic. Acute kidney disease (ARD) Acute Renal Failure (ERA) is defined as the decreased ability of the kidneys to eliminate waste nitrogenous products, established in hours to days, with a window of 15 days to 3 months, thus becoming a sub (ERA) acute, if it exceeds 3 months then it becomes Chronic Renal Failure (CKD) Chronic Kidney Disease Because there is a compensatory hypertrophy of the remaining nephrons, renal function remains normal for a while, this is a period of relative renal disease during which it is asymptomatic, homeostasis is preserved, and can only be diagnosed by an abnormality. mild from laboratory tests. However, the damage progressively increases, decreasing the renal capacity to carry out its excretory, endocrine and metabolic functions beyond compensatory mechanisms. [4]

Definition of chronic kidney disease Chronic kidney disease: structural or functional abnormalities of the kidney, present for more than three months with health implications. The "chronicity" criterion defines it when kidney damage is present for more than three months, thus differentiating it from acute kidney disease, which generally lasts less than that period.

Causes of chronic kidney disease

- Primary glomerular disease focal segmental sclerosis Membranoproliferative or mesangiocapillary glomerulonephritis
- Endo and extracapillary proliferative glomerulonephritis
- Nonproliferative extramembranous glomerulonephritis
- Secondary glomerular disease
- lupus nephropathy
- Henoch Schönlein nephropathy
- Sickle cell nephropathy.
- renal amyloidosis
- Mellitus diabetes
- Goodpasture syndrome
- Wegener's granulomatosis
- scleroderma
- Polyatheritis
- AIDS
- obstructive uropathies
- Posterior urethral valves
- ureteroceles neurogenic bladder b) Bilateral polyureteral obstruction with hydronephrosis c) Primary megaureter d) Infections
- Kidney tuberculosis
- Bladder neck obstruction
- Abdominal muscle hypoplasia
- Urethral stricture
- Tumors
- Renal hypoplasias a) Simple bilateral renal hypoplasia b) Renal hypoplasia with oligomeganephrones c) Hypoplasia with dysplasia e) Kidney dysplasia
- Hereditary nephropathies a) Nephronophthisis polycystic kidney c) Chronic tubular acidosis d) Allport syndrome e) Nephrotic syndrome in children f) Neil Patela nail-patella dysgenesis g) Family benign hematuria. thinned basement membrane h) Chronic idiomatic hypercalcemia j) Idiomatic hypercalcemia
- Vascular nephropathies a) Hemolytic uremic syndrome renal artery c) Bilateral renal vein thrombosis d) Renal cortical necrosis

- Interstitial nephritis a) Nephropathies due to analgesics b) Other interstitial nephropathies
- Metabolic diseases a) Amyloidosis
- Etiology Unknown a) Unclassifiable nephropathies

Risk factors for chronic renal failure and its results

Type Definition Examples Susceptibility factors. Factors that increase susceptibility to kidney damage Elderly, family history of chronic renal failure, reduced kidney mass, low birth weight, ethnic or racial minority in the United States, low income or educational level initiation factors Factors that directly initiate kidney damage Diabetes mellitus, arterial hypertension, autoimmune diseases, systemic infections, urinary tract infections, renal lithiasis, lower urinary tract obstruction, drug toxicity Progression factors Factors that accentuate kidney damage and kidney function declines faster after the damage has started Higher levels of proteinuria, higher arterial hypertension, poor glycemic control in diabetes, smoking End-stage factors Factors that increase morbidity and mortality in renal failure Low dialysis dose (Kt/V)*, temporary vascular access, anemia, low serum albumin, late start of dialysis Manual of Nephrology (Spiral Manual), by R. Schrier 6th edition (October 5, 2004) *Kt/V (accepted nomenclature for dialysis doses), "K" stands for urea clearance, "t" represents time, and "V" represents volume of distribution for urea. Clinical manifestations of chronic kidney disease Chronic kidney disease is the stage that occurs when the destruction of nephrons or specific parts of them, such as the glomerulus, renal tubules, and renal vasculature, exceeds 80% of the available organic mass. It is an irreversible and progressive syndrome that reduces glomerular filtration. Its origin can be given by multiple entities, such as primary or secondary glomerulopathy, metabolic diseases, such as diabetes mellitus, immunological diseases such as systemic lupus erythematosus, neoplastic diseases, and hypertension. Initially, the patient may not manifest symptoms, and it is only possible to detect abnormalities in laboratory tests, with a decrease in the glomerular filtration rate [5].

Signs and symptoms of uremia appear when the glomerular filtration rate reaches 5-10 ml/minute. The first symptoms are related to an increase in the amount of nitrogenous products in the blood, a decrease in the concentration of urine, and the beginnings of anemia. Once renal failure has occurred, it manifests with metabolic acidosis, decreased plasma K and P values, which lead to the final state of renal disease, uremic syndrome, as a consequence of the retention and accumulation of toxic products of metabolism and metabolism. decreased metabolic and endocrine functions of the kidney. CKD affects most systems and clinical signs depend on the stage of renal failure and the systems involved. Urea syndrome is a constellation of signs and symptoms in a patient with advanced renal failure, underlined by generalized malaise and fatigue.

These signs and symptoms are described below, ordered by organ systems. integumentary system The result of pallor produced by underlying anemia and the deposition of ill-defined accumulated pigments. The skin is dry and there may be ecchymosis as a result of the tendency to bleed. The patient frequently complains of brittle nails, and onycholysis may be prominent. Cardiovascular system During the course of chronic renal failure, hypertension develops in 90% of patients. It undoubtedly contributes to other problems, including development of cardiomyopathy, accelerated atherogenesis, and progression of renal failure itself. The habitual use of ultrasound also frequently reveals pericardial effusions in patients with chronic renal failure, without symptoms of pericarditis. The leading cause of mortality in the population with chronic renal failure [6].

Pulmonary or Respiratory System Pleurisy may be another representation of the generalized serositis that occurs in uremia. "Uremic lung", a butterfly-shaped pulmonary infiltrate on chest X-ray, associated with dyspnea and hypoxemia, is another entity that is debated whether it is really exclusive to the uremic milieu. Lung calcification, another consequence of impaired soft tissue calcium-potassium homeostasis, can sometimes be seen on chest radiography in patients with chronic renal failure. Gastrointestinal system Anorexia and nausea become progressively more frequent as the patient becomes uremic. Nausea is often more prominent in the morning, and the patient notes a specific aversion to meat. There is an increased incidence of gastrointestinal bleeding in patients with chronic renal failure, and the frequent finding of gastritis, duodenitis, and even colitis may reflect generalized mucosal injury from uremia. Circulating gastrin levels are high. Hematological system. Once the glomerular filtration rate is less than 40-50% of normal, a normocytic normochromic anemia will predictably develop, the severity of which progressively increases in parallel with the course of renal decline. Erythropoietin is not completely absent, however other clinical and *in vitro* evidence suggests a variety of presumed marrow toxins in the uremic environment also reduce the erythropoietic response of the marrow. (However, exogenous erythropoietin can completely reverse the anemia).

Renal osteodystrophy appears in patients with chronic renal failure, both before and after starting dialysis. Subperiosteal penetration of cortical bone, seen in the fingers, clavicles, and lamina dura of the mandible, and "brown tumors" suggest osteitis fibrosa but are not pathognomonic. The "wrinkled jersey" spine is a characteristic finding of patients with chronic renal failure. Neurological system All patients with chronic renal failure develop central and peripheral neurologic signs and symptoms as they become uremic. Characteristically, however, patients complain of cramps, "burning feet" or "restless leg." Subtle central nervous system dysfunction may manifest as restlessness, irritability, and changes in sleep pattern, and the electroencephalogram may show abnormalities consistent with metabolic encephalopathy. Cognitive function deteriorates, with memory loss and inability to perform mental tasks. Finally, confusion, disorientation, and lethargy develop, along with asterixis and myoclonus. Metabolic Endocrine System Chronic renal failure is a state of carbohydrate intolerance, with peripheral insulin resistance, due to impaired insulin binding to ef-

fector tissues, as well as post-receptor defects. Some of the clinical manifestations of uremia suggest a diagnosis of hypothyroidism. In fact, there is a marked incidence of goiter in patients with chronic renal failure [1,2,7].

To summarize, in CKD anemia, adynamia, fatigue, dyspnea, anorexia, nutritional status disorders, metabolism and homeostasis abnormalities, bone disease, decreased vitamin D and hyperglycemia are also affected. So far it has been shown that patients with kidney damage are exposed to multiple complications and/or risks, since all organs and organ systems can be compromised by the disease. Therefore, dentistry professionals must be oriented in the management of these patients. They cannot see the oral cavity as a separate entity, in these patients it is important to know the criteria of their primary care physician, depending on the procedure to be performed and whether a large-scale surgery is conservative.

It is essential to reflect carefully on which drug is going to be prescribed, always assessing the risk and benefit. The kidney plays a preponderant role in the elimination of drugs, the dentist has a great responsibility when it comes to establishing a therapy for these patients with this type of alteration. Variations in the pharmacokinetics and pharmacodynamics of these effects should be analysed. In general, the principles of the prescription are the same as for any patient. (see chapter on risks due to poor medication prescription).

Drugs may have affected the rate of excretion, in patients with renal failure more than in healthy patients. The metabolism of a drug is carried out fundamentally in the liver and to a lesser extent in the membrane of some organs, in fluids up to the intestine, the excretion of said drugs is generally carried out by the kidney and by the liver (first stage), the Uremia affects the liver, decreasing the metabolism of drugs and presenting a high concentration of these in the plasma. Uremia affects the absorption of some drugs that require an acid medium to be absorbed, which cannot be completed due to the alkanization that it performs in ammonia from urea in a cycle that prevents the process from being completed.

There are also some complications such as the interaction with other drugs that patients with renal failure consume, such as antacids that bind phosphates to the diet, preventing CKD patients from absorbing some available drugs. In patients with edema, the volume of distribution of a drug will show a little concentration due to the fluid retention shown. On the other hand, in patients with kidney disease, the concentration of some proteins is low, mainly due to the diet and the treatment that the patient receives, such as serum albumin, which binds to some drugs in the liver or in the liver. kidney for excretion. therapeutic norms.

The selection of a method depends on the clinical state of the patient and the characteristics of the drug, in addition, it is necessary to know if a target level, peak or trough concentration of the drug is desired or if a constant level of the drug is desirable. between doses. Both the extension of the interval and the reduction of the dose cause similar mean plasma concentrations of the drug. The interval extension method causes larger fluctuations between peak and trough concentrations and is recommended when a constant serum concentration of the drug (e.g., aminoglycosides) is not required [1,2].

This method is obviously more convenient, since it promotes compliance with the therapy by the patient with fewer doses and is cheaper. The dose reduction method is recommended when more constant levels of drug serum concentration are desired (for example: beta-lactam antibiotics). The adjustment of the dose based on the serum levels of the drug and the clinical response may recommend a combination of both methods. a group of commonly used drugs that do not require readjustment in patients with renal failure are cited. Most used drugs that do not need readjustment Antibiotics - Amoxicillin - Azithromycin - Ceftriaxone.

Non-steroidal anti-inflammatory drugs • Diclofenac • Ibuprofen • Indomethacin • Naproxen • Piroxicam Muscle relaxants • Methocarbamol

Anticoagulants and antiplatelet agents • Heparin • Warfarin Anticonvulsants

• Carbamazepine

Tricyclic antidepressants • Amitriptyline Antihistamines • Chlorpheniramine

• Diphenhydramine.

Drugs with special considerations aminoglycosides

- Cause of acute renal failure
- They should only be prescribed to patients with renal insufficiency when clearly indicated.
- The therapeutic response is related to its concentration
- The risk of nephrotoxicity is related to high trough levels nonsteroidal anti-inflammatories
- May have a negative effect on renal blood flow, glomerular filtration rate, and renal sodium reabsorption.
- Nephrotoxicity
- In patients with arterial hypertension they can increase blood pressure by increasing renal sodium retention.
- Significant reduction in glomerular filtration rate and simultaneously increase serum potassium concentrations.
- Its use should be confined to short periods of time to treat acute conditions.
- No dose adjustment is necessary since they have a very important hepatic metabolism (except ketorolac whose dose must be reduced in patients with chronic kidney disease).
- Large doses of these drugs should be avoided in patients with chronic kidney disease due to the possibility of developing gastrointestinal bleeding. Benzodiazepines
- Are prescribed without any type of dose adjustment Antifungals

- Should be administered only if there is no alternative such as Amphotericin and Fluconazole. Antivirals
- Reduce dose

Chronic kidney disease and stomatology

The practice of Stomatology must be included within the concept of "total health"; This is possible if the interdisciplinarity between medicine-stomatology is carried out properly. The oral cavity and its annexed structures cannot be considered as independent compartments from the rest of the organism. The changes or alterations associated with diseases, organic or systemic, that can be observed in the oral cavity are numerous and quickly observable, which highlights the importance of knowing them for the diagnosis and prognosis of different pathologies. Dentistry should aim to integrate into interdisciplinary health teams.

The dentist must have knowledge of the basic processes of diseases and establish whether dental treatment will affect or be affected by the patient's condition. More than 90% of patients with kidney disease present oral signs and symptoms of the disease, which are not pathognomonic or decisive in the diagnosis. Oral complications can occur as a result of CKD or its treatment. According to the patient's clinical condition, an unavoidable dental and periodontal maintenance program must be established every 3 months. It is of fundamental importance to know the different stages of kidney disease and the specific treatment that the patient receives, therefore, permanent consultation with the treating physician, before any dental procedure that requires modification of the usual medication is necessary [9].

Oral manifestations

Patients with CKD are considered special patients because we call special patients those who present signs and symptoms that deviate from normality, be they physical, mental or sensory, as well as behavioral, who require maneuvers, dental and stomatological care. concepts, equipment and special help people, with the capacity to meet the needs that these generate in the office. Dental treatment of these patients requires an understanding of the systemic complications of chronic renal failure, which have been studied before.

Direct contact with the nephrologist can alert the dentist to important problems present in the patient and facilitate the development of a comprehensive dental treatment plan. It is important to bear in mind that the clinical manifestations of kidney disease (CKD) rarely come from an isolated kidney problem, since all organs and systems of the human body are affected to a greater or lesser extent. Among the systemic manifestations, arterial hypertension is a frequent complication.

We can also mention as complications the ischemic diseases of the patient with a high frequency of ischemic heart disease, cardiovascular accidents, mesenteric ischemia and peripheral ischemic disorders, especially in elderly patients. 90% of patients with CKD present oral signs and symptoms, which present in hard tissues, soft tissues and salivary glands. In the soft tissues it can be found: gingival enlargement, gingivitis, gingival bleeding, paleness of the mucosa, petechiae and ecchymosis, coated tongue, mouth ulcers, candidiasis, angular cheilitis. In hard tissues it can be observed: bacterial plaque and dental calculus, dental caries, dental erosion, enamel hypoplasia, loss of insertion and dental mobility and temporomandibular dysfunction. At the level of salivary glands it can be observed: xerostomia, dysgeusia and halitosis.

Soft tissue manifestations: Within the soft tissue alterations we have paleness in the oral mucosa due to anemia, which can be divided into two components: 1) The minor component can be corrected with dialysis, so it is likely that it can be attributed to the retention of some substance in the plasma as a consequence of the failure of the renal excretory function. 2) The main cause of normocytic and normochromic anemia is the inability of the marrow to respond to the magnitude, usually small, of hemorrhages and moderate hemolysis. Paleness is a typical sign in patients with chronic renal failure, this is due to decreased production of the hormone erythropoietin. Petechiae, ecchymosis and gingival bleeding can be caused by platelet dysfunction, thrombocytopenia and thrombasthenia or both, due to a qualitative alteration of platelets, which lose their ability to aggregate and adhere or due to the accumulation of uremic toxins. [10]

On the other hand, we have the influence of anticoagulant drugs such as heparin that is used in patients who will undergo hemodialysis therapy, which produce platelet dysfunction, coagulation inhibition, and increased capillary fragility. spontaneous gum bleeding Individuals with CKD generally suffer from thrombocytopenia, capillary fragility, and decreased platelet adhesion. Said alteration is caused by uremic intoxication and when undergoing hemodialysis, these symptoms are increased by the application of heparin and by the damage suffered by platelets when hitting against the internal walls of the ducts of the apparatus that performs the exchange of liquids, thus reducing the coagulation capacity, generating gingival bleeding as an obstacle to dental care. As a consequence of the alteration in the level of coagulation factors, the soft tissue of the oral cavity, such as the gums and mucous membranes, can take on a purple color.

Petechiae and ecchymosis Petechiae and ecchymosis may be seen as a result of platelet dysfunction and anticoagulants used during hemodialysis Alterations in the salivary glands Some authors suggest the possible reasons why the salivary glands are altered, presenting an atrophy in their parenchyma, this is due to the chemical inflammation that occurs due to the high levels of ammonia in the saliva and results in a decrease in the flow and what worsens the state of secretion of the salivary glands even more is the restriction of fluids that these patients present in their diet or due to the medications they take (antihypertensives). Parotitis CKD patients are usually predisposed to retrograde parotitis,

which is believed to be the result of a combination involving the gland, adverse effects of drug treatment, chemical inflammation, dehydration, and oral respiration. Glossitis.

The various alterations at the blood level present in the renal disorder, such as thrombocytopenia and thrombasthenia, generate changes in the tongue that trigger glossitis, considered inflammation of the tongue accompanied by a burning sensation and increased sensitivity to food. Gingival enlargement or hyperplasia occurs as a consequence of medications (calcium channel blockers) taken by patients with CKD who have associated high blood pressure. Gingivitis can also be caused by drugs as in the cases of gingival enlargement, the difference is that it can also be influenced by inflammatory and irritative phenomena that is produced by the high concentration of urea in various body fluids, this situation favors bacterial development , which acts as a lesion agent by colonization or by degradation of ammonium with the release of irritative factors. Gingivitis due to systemic factors is characterized by being modified in its evolutionary course by various general effects such as the endocrine system or blood dyscrasia. If gingivitis is not controlled, it can evolve into periodontitis, spreading to deeper areas, such as the periodontal ligament and alveolar bone [11,12].

Both physical and mental or sensory handicaps are associated with poor oral hygiene with greater oral and dental impairment in general, and periodontal impairment in particular. Within the great variety of oral conditions that exist in this group, periodontal disease is the most important problem at the stomatological level. Periodontal disease, in addition to bacterial plaque, is influenced by many other factors, both systemic and local. mucosal lesions.

White lesions with or without ulcerations can be observed. It is common to observe lichenoid lesions associated with the medication received by CKD patients. White plaques can be seen on the skin and occasionally on the oral mucosa called uremic frost resulting from urea crystals remaining as a result of sweat and saliva evaporation. Patients with chronic renal failure due to their general condition due to the disease and the high concentrations of ammonia in their saliva may present irritation of the oral mucosa and ulcerations, which may be accompanied by symptoms of pain and burning. A typical oral manifestation of this disease is uremic stomatitis. Uremic stomatitis: It is a rare complication of uremia, which can occur as a result of advanced renal failure. The etiology of uremic stomatitis remains unknown, although it has been suggested that it may be the consequence of elevated levels of ammonium compounds. Ammonia is formed through the action of bacterial ureases modifying salivary urea, which may be elevated in affected patients. The loss of local defense systems facilitates the proliferation and colonization of the usual bacteria in the mouth, but especially the fusospirochetal association. Barles classification of uremic stomatitis

- **Type I:** it has an erythematous-papulaceous form that initially manifests as a reddish thickening of the buccal mucosa, then with a thick, pasty, sticky gray exudate; and finally the appearance of pseudomembranes that cover the mucous membranes of the oral cavity. This can be accompanied by pain, halitosis, xerostomia, burning sensation and pain, yeast infection and dysgeusia.
- **Type II:** It is similar to type I, but includes a loss of mucosal integrity with extensive ulcerations. Ulcers can be superficial or deep and frequently affect the gums. These alterations disappear spontaneously when medical treatment decreases the urea concentration (48 hours after dialysis). Oral candidiasis in patients with chronic renal failure can be caused by various factors that can immunosuppress them, such as ingesting certain medications that make them sensitive to infection, such as folic acid that is given to patients when they are suffering from anemia, corticosteroids, among others. Another factor that predisposes them to contracting this infection is the low salivary flow that they present and the degree of stress that these patients undergo due to their disease. The lesions will look reddish ulcerative that are often accompanied by pseudoplaques that can be detached. [12, 13]

Hard tissue injuries. The hard tissues can also be affected, it can be observed that a high percentage of patients with renal failure have a greater accumulation of bacterial plaque, when the patient does not have an optimal oral cleanliness and this plaque is not removed, it accumulates and calcifies, thus forming dental calculus that is composed of 70-90% of inorganic elements and the difference in organic. These patients are more predisposed to form salivary stones because they have increased levels of urea in saliva, phosphorus and calcium carbonate, which they ingest for treatment of the disease (inorganic elements). Dental erosion occurs in patients with chronic renal failure due to frequent acid regurgitation such as urea-induced vomiting, medications, and dialysis.

Mostly dental erosion will be observed in the lingual areas of the teeth. Studies of oral disorders in children with chronic renal failure and kidney transplants have been carried out and observed that many of the patients examined with the disease presented enamel hypoplasia. This alteration in the enamel occurs during the formation and development of the tooth and when the patient is in nephrocalcinosis, which is the precipitation of calcium phosphate in the kidney tubules. It can be said that enamel hypoplasia is a constant and pathognomonic sign in pediatric patients presenting with chronic renal failure. There is a high percentage of children with chronic renal failure who present rash problems, secondary to oral administration of iron to treat anemia. Renal osteodystrophy, are lesions presented by patients with chronic renal failure, course as part of the evolution of their metabolic bone disease before the total deterioration of renal function. Patients are more predisposed to fractures, joint pain, etc. Among the oral alterations we can see that it can produce alterations in the shape of the jaws, making them prominent, repercussions on the temporomandibular joint, predisposing to jaw fractures, bone resorption, among others [14].

Loss of insertion In patients with chronic renal failure, an alteration in bone metabolism and mineralization will be observed, which could in turn alter periodontal bone surfaces, causing dental mobility and thus favoring periodontal disease. Some authors associate loss of attachment with renal osteodystrophy. Possible involvement of the temporomandibular joint has been documented, including decreased bone density, subchondral cysts, and irregularities of the condyle head or glenoid fossa, or both, and in the most severe cases. Complete resorption of the condylar head and coronoid process. Other manifestations Halitosis The first oral sign of renal failure is "uremic fetor." Halitosis is caused by microbial degradation in the oral cavity that leads to the production of volatile sulfur compounds. These compounds are mainly produced by Grand negative anaerobic bacteria. It also occurs secondary to uremia, an ammonia odor can be detected on the patient's breath. All this occurs due to the high concentrations of ammonium in the saliva and due to the decrease in salivary flow. metallic taste The metallic taste reported by these patients is the result of the high concentration of urea in the saliva and its subsequent transformation into ammonia. Metallic taste and odor occurs in one third of hemodialysis patients.

The change in taste can also be caused by the use of medications, a decrease in the number of taste buds, and changes in salivary flow. Additionally, these patients report sensory alterations, that is, a dysesthesia or altered sensation of flavors, especially with sweet and sour tastes due to high levels of urea and the presence of dimethyl and trimethyl amines or low levels of zinc due to absorption. derived from gastrointestinal disorders sensory disturbances Patients with CKD may report dysesthesias such as burning mouth syndrome of the lips and tongue of neuropathic origin, as well as a sensation of enlargement of the tongue xerostomia. It can develop for various reasons: decreased salivary flow due to involvement of the salivary glands, chemical inflammation, dehydration, diabetes mellitus, advanced age, mouth breathing, due to restricted fluid intake.

The other conditions that cause xerostomia in uremic patients are: mumps, metabolic abnormalities, and the consumption of medications such as antihypertensives and diuretics, fibrosis, and atrophy of the parenchyma of the minor salivary glands. Secondary to xerostomia, glossitis, cervical caries, ulcerated or atrophic lips, and candidiasis may occur.

Oral lesions associated with transplant immunosuppression It has been warned that CKD patients who have received transplants and are receiving immunosuppressive therapy may present hairy leukoplakia associated with immunosuppressive drugs. These patients have a greater susceptibility to developing epithelial dysplasia and carcinoma of the lip [5].

The risk of malignancy is probably a consequence of the effects of iatrogenic immunosuppression, which makes the mucosa more susceptible to developing tumors related to oncogenic viruses, such as Kaposi's sarcoma, non-Hodgkin's lymphoma. From all of the above, it can be deduced that chronic kidney patients are at risk of suffering from infections, bleeding episodes, risk of fractures, risk of suffering from periodontal diseases, predisposition to tumor diseases and of infecting health personnel with hepatitis and HIV. infections It is important to take into account that the main causes of death in CKD and kidney transplant recipients are cardiovascular disease and infections. The patient with a kidney transplant must take immunosuppressants, such as corticosteroids or cyclosporine, to avoid kidney rejection; however, they have increased infections as a side effect, so one must be very careful in the use of antibiotics and keep the patient with optimal dental hygiene. Septicemia is common in CKD due to depressed immune response.

The number and function of lymphocytes are reduced, as are chemotaxis and phagocytosis of neutrophils. Another peculiar aspect of the hemodialysis procedure is that an arteriovenous fistula is performed in these patients, which allows multiple punctures to "connect" the patient to the dialysis machine; Since any type of implant, such as those that have direct contact with the bloodstream, can retain microorganisms and create a situation that causes endoarteritis or endocarditis or both, therefore antimicrobial prophylaxis should be considered. For this reason, it is very important to carry out an evaluation of the oral status of these patients to eliminate possible infectious dental foci and indicate antibiotic prophylaxis (2 g of amoxicillin orally in adults or 50 mg/kg in children, 1 hour before the intervention). in any procedure that causes bacteraemia (periodontal treatment, conventional or surgical extraction, etc.).

In case of allergy to penicillin, clindamycin 600 mg in adults or 20 mg/kg in children will be indicated, 1 hour before the intervention, other therapeutic variants in a prophylactic way would be ampicillin, 500 mg every 8 h, or clindamycin, 300 mg every 8 h, in elective cases. When these patients are treated dentally, the use of antibiotics is extremely important, since it is known that a high percentage of cases of bacterial endocarditis can be caused by dental treatment due to microorganisms from the oral cavity. Oral candidiasis infections in transplant patients and those receiving dialysis are common. A prevalence between 20-30% has been reported. Candida infections present as angular cheilitis, pseudomembranous candidiasis, erythematous ulceration, or chronic atrophic candidiasis. Prevention, which is the only effective method in newly transplanted patients, consists of oral medication and antifungal solutions. Cytomegalovirus and herpes virus infection have also been found in these patient [16].

For a patient on peritoneal dialysis, there are not as many requirements as one on hemodialysis for dental treatment, given that he does not have to face "heparinization" or the risks of viral infection. However, it should not be neglected for the purposes of infection prevention that the patient carries an external catheter, so antimicrobial therapy will always be advisable due to the high risk of developing peritonitis due to the following.

- The catheter in the peritoneal cavity is a foreign body.
- Dialysis fluid, due to its high glucose content c) Nitrogenous waste products, due to their high risk of developing the disease.
- Dental treatment, especially invasive procedures
- Periodontal scaling and extractions, since surgery can send bacteria into the bloodstream, which are deposited in the peritoneal cavity and cause peritonitis. For all these reasons, the use of prophylactic antibiotics is indicated. risk of bleeding It is common for patients with CKD to have hematological problems, especially normochromic and normocytic anemia that usually appears when the glomerular filtration rate is less than 60ml/min.

This anemia is multifactorial and will be caused by the inability of the kidney to produce erythropoietin, due to toxins. uremics that shorten the half-life of the red blood cell, decreasing the affinity of the erythropoietin receptors and the binding capacity of transferrin to iron, due to blood loss, an example of this is the blood that remains residual in the hemodialysis circuits or during the disconnection, due to iron and vitamin deficiency, due to fibrosis of the bone marrow secondary to hyperparathyroidism. Other factors to take into account and that affect the risk of bleeding is the qualitative alteration of platelets, which is a platelet dysfunction influenced by uremic thrombopathy, a consequence of the inhibitory effect of uremic toxins on platelet function. It has been seen that uremic patients present higher levels of prostacyclin and nitric oxide, both potent agents that inhibit platelet adhesion, which lose their capacity for aggregation and adhesion; another platelet dysfunction is the defective interaction between von-Willebrand factor and platelet glycoprotein IIb-IIIa receptors and the use of drugs such as heparin In "heparinized" patients, tests should be indicated to measure the coagulation capacity of plasma, such as prothrombin time and partial thromboplastin time. Dental management should be carried out when the figures are preferably normal or as close to normal. Surgical procedures, debridements, or punctures should not be attempted with platelet counts less than 80,000. Considering that multiple factors are involved in the pathophysiology of hemorrhage in uremia, an approach to all of them should be included in its prevention and treatment [14].

For its simplification we could structure them into general and specific measures: General measures: 1. Eliminate uremic toxins: toxins accumulated in uremia contribute to platelet dysfunction. Its elimination through renal function replacement therapy contributes to improving platelet function and shortening bleeding time. Both hemodialysis and peritoneal dialysis are equally effective in removing toxins and therefore in preventing perioperative bleeding. For this reason, those patients who are in the phase of advanced CKD and the patient requires scheduled surgery could consider starting dialysis before performing the surgical procedure. 2. In patients who are on a hemodialysis program, this will be done before the intervention. To prevent bleeding, hemodialysis without heparin will be carried out, performing frequent system washes. In the event that hemodialysis with heparin has been performed, it is advisable to wait a prudent time before going to the operating room in order to manage it as well as possible; This attention should not be within the first four hours of receiving dialysis, since the anticoagulant effect of heparin will still be present, which can expose you to hemorrhage or prolonged bleeding. Anesthetic punctures are included in this type of observation. The best option is to plan the treatment the day after hemodialysis has been performed.

Correction of anemia: The use of recombinant human Erythropoietin (EPO) and its derivatives allows improvement of anemia associated with chronic renal failure. Its use requires having adequate iron stores and may require the administration of oral or intravenous iron. With these measures we can correct the anemia associated with CKD and shorten the bleeding time. From the administration of EPO to obtaining an adequate response in hematocrit levels, a period of 2-6 weeks can pass, so the use of these agents should be started early, before the patient goes to the operating room. Blood transfusions are not recommended in a chronic way, since it is possible to immunize a patient who may require a kidney implant in the future. Blood transfusions are indicated in cases of acute bleeding and urgent surgical interventions. 3. Withdrawal of drugs (antiplatelet agents and anticoagulants): The withdrawal of antiplatelet drugs must be individualized according to the type of surgery to be performed and the assessment of the risk benefit of their withdrawal (thrombosis/bleeding). In general, it is advisable to suspend antiaggregants at least 72 hours before surgery.

It is also advisable to withdraw oral anticoagulation and start it with heparin and avoid the use of drugs frequently used in surgical services such as NSAIDs, which reversibly inhibit COX and their use carries a high risk of bleeding. Specific measures: 1. Desmopressin: This agent is a synthetic derivative of vasopressin-ADH with a lower pressor effect. Its mechanism of action, in the prevention and treatment of uremic bleeding, is related to the release of factor VIII and vf W multimers from the endothelium. Desmopressin is administered at a dose of 0.3 mcg/kg iv/sc over 15-30 minutes. Its activity appears after 30-60 minutes, with a duration of its effect of 6-12 hours. For this reason, its main indication is in the prevention and treatment of acute bleeding and before performing invasive tests (biopsy). In a recent review of evidence-based recommendations for the treatment of uremic bleeding, the use of desmopressin would be included with a recommendation grade I and evidence A, avoiding the administration of a second dose (tachyphylaxis). 2. Cryoprecipitates: Upon thawing of the plasma, the presence of a supernatant was observed [18-20].

The use of these cryoprecipitates shortens the bleeding time in uremia. Its mechanism of action is to provide the content of these factors. 10 U iv are administered, beginning its effect in the first hour and maintaining it between 12-24 hours. The fact that it is a potential source for transmitting infections has limited its use to those cases refractory to desmopressin and frequent blood transfusions. 3. Estrogens: The observation that patients with von

Willebrand disease and hereditary telangiectasia improved during pregnancy led to the use of estrogens to prevent bleeding. Its mechanism of action to prevent bleeding is not entirely known. It is believed that it inhibits L-arginine, a precursor of nitric oxide, improving platelet aggregation. Most of the studies carried out with estrogens in the prevention of uremic bleeding have been intravenously (0.6 mg/kg/day), although their use is also described orally (50 mg/day) or transdermally (50 -100 mcg/2 times/week). Its effect appears after 24 hours, with a maximum peak of action after 5-7 days, maintaining its effect for 2 weeks. This slower onset of action means that its main indication is elective (chronic) preparation before surgery. 4. Antifibrinolytic drugs: the systemic inflammation present in uremia is associated with the activation of the fibrinolytic system, therefore, the use of these agents (tranexamic acid) could shorten the bleeding time in uremia [18-20].

Management of perioperative bleeding in the uremic patient General measures + desmopressin. Minor surgery, dental extractions, biopsies and emergency surgery Local bleeding control measures Oral and periodontal surgical procedures, intraoperatively and postoperatively Tranexamic acid In the form of a mouthwash, it acts as an antifibrinolytic element Meticulous surgical technique Closure of wounds by primary intention, the help of traditional elements such as oxidized regenerated cellulose, as well as cold thermotherapy will help prevent bleeding complications General measures + conjugated estrogens 5 days before the procedure + desmopressin 1 hour before the intervention Scheduled major surgery Protamine sulphate to block the anticoagulant effect of heparin If immediate major surgical treatment is necessary It can be said that these patients can be at high surgical risk, which does not imply that the professional treats the back, on the contrary, it is necessary to prepare every day to provide them with better care and give them a better quality of life [18-20].

Conclusion

The main complications and risks of kidney disease in dental care are the risk of infection, intraoperative and postoperative bleeding.

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