I'm not a bot

مبادئ توجيهية للسكتة الدماغية الحادة pdf

Cerebrovascular accident: The sudden death of some brain cells due to lack of oxygen when the blood flow to the brain is impaired by blockage or rupture of an artery to the brain. A CVA is also referred to as a stroke. Symptoms of a stroke depend on the area of the brain affected. The most common symptom is weakness or paralysis of one side of the body with partial or complete loss of voluntary movement or sensation in a leg or arm. There can be speech problems and weak face muscles, causing drooling. Numbness or tingling is very common. A stroke involving the base of the brain can affect balance, vision, swallowing, breathing, and even unconsciousness. A stroke is a medical emergency. Anyone suspected of having a stroke should be taken immediately to a medical facility for diagnosis and treatment. The causes of stroke: An artery to the brain may be blocked by a clot (thrombosis) which typically occurs in a blood vessel that has previously been narrowed due to atherosclerosis ("hardening of the artery"). When a blood clot or a piece of an atherosclerotic plaque (a cholesterol and calcium deposit on the wall of the artery) breaks loose, it can travel through the circulation and lodge in an artery of the brain, plugging it up and stopping the flow of blood; this is referred to as an embolic stroke. A blood clot can form in a chamber of the heart when the heart beats irregularly, as in atrial fibrillation; such clots usually stay attached to the inner lining of the heart but they may break off, travel through the bloodstream, form a plug (embolus) in a brain artery and cause a stroke. A cerebral hemorrhage (bleeding in the brain), as from an aneurysm (a widening and weakening) of a blood vessel in the brain, also causes a stroke. The diagnosis of stroke involves a medical history and a physical examination. Tests are done to search for treatable causes of a stroke and help prevent further brain damage. A CAT scan (a special X-ray study) of the brain is often done to show bleeding into the brain; this is treated differently than a stroke caused by lack of blood supply. A CAT scan also can rule out some other conditions that may mimic a stroke. A soundwave of the heart (echocardiogram) may be done to look for a source of blood clots in the heart. Narrowing of the carotid artery (the main artery that supplies blood to each side of the brain) in the neck can be seen with a soundwave test called a carotid ultrasound. Blood tests are done to look for signs of inflammation which can suggest inflamed arteries. Certain blood proteins are tested that can increase the chance of stroke by thickening the blood. Stroke look-alikes: Just because a person has slurred speech or weakness on one side of the body does not necessarily mean that person has had a stroke. There are many other nervous system disorders that can mimic a stroke including a brain tumor, a subdural hematoma (a collection of blood between the brain and the skull) or a brain abscess (a pool of pus in the brain caused by bacteria or a fungus). Virus infection of the brain (viral encephalitis) can cause symptoms similar to those of a stroke, as can an overdose of certain medications. Dehydration or an imbalance of sodium, calcium, or glucose can cause neurologic abnormalities similar to a stroke. Treatment of a stroke: Early use of anticoagulants to minimize blood clotting has value in some patients. Treatment of blood pressure that is too high or too low may be necessary. The blood sugar (glucose) in diabetics is often quite high after a stroke; controlling the glucose level may minimize the size of a stroke. Drugs that can dissolve blood clots may be useful in stroke treatment. Clot retrieving interventions can now be performed in some stroke centers. Oxygen is given as needed. New medications that can help oxygen-starved brain cells survive while circulation is reestablished are being developed. Rehabilitation: When a patient is no longer acutely ill after a stroke, the aim turns to maximizing the patient's functional abilities. This can be done in an inpatient rehabilitation hospital or in a special area of a general hospital and in a nursing facility. The rehabilitation process can involve speech therapy to relearn talking and swallowing, occupational therapy for regaining dexterity of the arms and hands, physical therapy for improving strength and walking, etc. The goal is for the patient to resume as many of their pre-stroke activities as possible. Footnote: The term "stroke" reflects the belief among the ancient Greeks and Romans that someone suffering a stroke (or any sudden incapacity) had been struck down by the gods. The Motorsport Images Collections captures events from 1895 to today’s most recent coverage.Discover The CollectionCurated, compelling, and worth your time. Explore our latest gallery of Editors’ Picks.Browse Editors' FavoritesExperience AI-Powered CreativityThe Motorsport Images Collections captures events from 1895 to today’s most recent coverage.Discover The CollectionCurated, compelling, and worth your time. Explore our latest gallery of Editors’ Picks.Browse Editors' FavoritesExperience AI-Powered CreativityThe Motorsport Images Collections captures events from 1895 to today’s most recent coverage.Discover The CollectionCurated, compelling, and worth your time. Explore our latest gallery of Editors’ Picks.Browse Editors' FavoritesExperience AI-Powered Creativity Share — copy and redistribute the material in any medium or format for any purpose, even commercially. Adapt — remix, transform, and build upon the material for any purpose, even commercially. The licensor cannot revoke these freedoms as long as you follow the license terms. Attribution — You must give appropriate credit , provide a link to the license, and indicate if changes were made . You may do so in any reasonable manner, but not in any way that suggests the licensor endorses you or your use. ShareAlike — If you remix, transform, or build upon the material, you must distribute your contributions under the same license as the original. No additional restrictions — You may not apply legal terms or technological measures that legally restrict others from doing anything the license permits. You do not have to comply with the license for elements of the material in the public domain or where your use is permitted by an applicable exception or limitation . No warranties are given. The license may not give you all of the permissions necessary for your intended use. For example, other rights such as publicity, privacy, or moral rights may limit how you use the material. Abnormal kidney structure or gradual loss of kidney function This article is about CKD in general. For the end-stage of the disease, see kidney failure. Medical conditionChronic kidney diseaseOther namesChronic renal disease, kidney failure, impaired kidney function[1]Illustration of a kidney from a person with chronic renal failureSpecialtyNephrologySymptomsEarly: None[2]Later: Leg swelling, feeling tired, vomiting, foamy urine, loss of appetite, confusion[2]ComplicationsHeart disease, high blood pressure, anemia[3][4]DurationLong-term[5]CausesDiabetes, heart failure, high blood pressure, glomerulonephritis, polycystic kidney disease[5][6]Risk factorsSmoking, genetic predisposition,low socioeconomic status[7]Diagnostic methodBlood tests, urine tests[8]TreatmentMedications to manage blood pressure, blood sugar, and lower cholesterol, renal replacement therapy, kidney transplant[9][10]Frequency753 million (2016)[1]Deaths1.2 million (2015)[6] Chronic kidney disease (CKD) is a type of long-term kidney disease, defined by the sustained presence of abnormal kidney function and/or abnormal kidney structure.[2][5] To meet criteria for CKD, the abnormalities must be present for at least three months.[11] Early in the course of CKD, patients are usually asymptomatic, but later symptoms may include leg swelling, feeling tired, vomiting, loss of appetite, and confusion.[2] Complications can relate to hormonal dysfunction of the kidneys and include (in chronological order) high blood pressure (often related to activation of the renin–angiotensin system), bone disease, and anemia.[3][4][12] Additionally CKD patients have markedly increased cardiovascular complications with increased risks of death and hospitalization.[13] CKD can lead to end-stage kidney failure requiring kidney dialysis or kidney transplantation.[9] Causes of chronic kidney disease include diabetes, high blood pressure, glomerulonephritis, and polycystic kidney disease.[5][6] Risk factors include a family history of chronic kidney disease.[2] Diagnosis is by blood tests to measure the estimated glomerular filtration rate (eGFR), and a urine test to measure albumin.[8] Ultrasound or kidney biopsy may be performed to determine the underlying cause.[5] Several severity-based staging systems are in use.[14][15] Testing people with risk factors (case-finding) is recommended.[16][8] Initial treatments may include medications to lower blood pressure, blood sugar, and cholesterol.[10] Angiotensin converting enzyme inhibitors (ACEIs) or angiotensin II receptor antagonists (ARBs) are generally first-line agents for blood pressure control, as they slow progression of the kidney disease and the risk of heart disease.[17] Loop diuretics may be used to control edema and, if needed, to further lower blood pressure.[18][10][19] NSAIDs should be avoided.[10] Other recommended measures include staying active,[20] and "to adopt healthy and diverse diets with a higher consumption of plant-based foods compared to animal-based foods and a lower consumption of ultraprocessed foods."[20][10][21] Plant-based diets are feasible and are associated with improved intermediate outcomes and biomarkers.[22] An example of a general, healthy diet, suitable for people with CKD who do not require restrictions, is the Canada Food Guide Diet.[23] People with CKD who require dietary restrictions or who have other specific nutritional problems should be referred to a dietitian.[20] Treatments for anemia and bone disease may also be required.[24][25] Severe disease requires hemodialysis, peritoneal dialysis, or a kidney transplant for survival.[9] Chronic kidney disease affected 753 million people globally in 2016 (417 million females and 336 million males.)[1][26] In 2015, it caused 1.2 million deaths, up from 409,000 in 1990.[6][27] The causes that contribute to the greatest number of deaths are high blood pressure at 550,000, followed by diabetes at 418,000, and glomerulonephritis at 238,000.[6] Uremic frost on the head in someone with chronic kidney disease CKD is initially without symptoms and is usually detected on routine screening blood work by either an increase in serum creatinine, or protein in the urine. As the kidney function decreases, more unpleasant symptoms may emerge:[28] Blood pressure is increased due to fluid overload and the production of vasoactive hormones created by the kidney via the renin-angiotensin system, increasing the risk of developing hypertension and heart failure. People with CKD are more likely than the general population to develop atherosclerosis with consequent cardiovascular disease, an effect that may be at least partly mediated by uremic toxins.[29][unreliable medical source?] People with both CKD and cardiovascular disease have significantly worse prognoses than those with only cardiovascular disease.[30] Urea accumulates, leading to azotemia and ultimately uremia (symptoms ranging from lethargy to pericarditis and encephalopathy). Due to its high systemic concentration, urea is excreted in eccrine sweat at high concentrations and crystallizes on the skin as the sweat evaporates ("uremic frost"). Potassium accumulates in the blood (hyperkalemia with a range of symptoms including malaise and potentially fatal cardiac arrhythmias). Hyperkalemia usually does not develop until the glomerular filtration rate falls to less than 20–25 mL/min/1.73 m2, when the kidneys have decreased ability to excrete potassium. Hyperkalemia in CKD can be exacerbated by acidemia (triggering the cells to release potassium into the bloodstream to neutralize the acid) and from lack of insulin.[31] Fluid overload symptoms may range from mild edema to life-threatening pulmonary edema. Hyperphosphatemia results from poor phosphate elimination in the kidney, and contributes to increased cardiovascular risk by causing vascular calcification.[32] Circulating concentrations of fibroblast growth factor-23 (FGF-23) increase progressively as the kidney capacity for phosphate excretion declines, which may contribute to left ventricular hypertrophy and increased mortality in people with CKD .[33][34] Hypocalcemia results from 1,25 dihydroxyvitamin D3 deficiency (caused by high FGF-23 and reduced kidney mass)[35] and the skeletal resistance to the calcemic action of parathyroid hormone.[36] Osteocytes are responsible for the increased production of FGF-23, which is a potent inhibitor of the enzyme 1-alpha-hydroxylase (responsible for the conversion of 25-hydroxycholecalciferol into 1,25 dihydroxyvitamin D3).[37] Later, this progresses to secondary hyperparathyroidism, kidney osteodystrophy, and vascular calcification that further impairs cardiac function. An extreme consequence is the occurrence of the rare condition named calciphylaxis.[38] Changes in mineral and bone metabolism that may cause 1) abnormalities of calcium, phosphorus (phosphate), parathyroid hormone, or vitamin D metabolism; 2) abnormalities in bone turnover, mineralization, volume, linear growth, or strength (kidney osteodystrophy); and 3) vascular or other soft-tissue calcification.[12] CKD–mineral and bone disorders have been associated with poor outcomes.[12][26] Metabolic acidosis may result from decreased capacity to generate enough ammonia from the cells of the proximal tubule.[31] Acidemia affects the function of enzymes and increases the excitability of cardiac and neuronal membranes by the promotion of hyperkalemia.[39] Anemia is common and is especially prevalent in those requiring hemodialysis. It is multifactorial in cause but includes increased inflammation, reduction in erythropoietin, and hyperuricemia leading to bone marrow suppression. Hypoproliferative anemia occurs due to inadequate production of erythropoietin by the kidneys.[40] In later stages, cachexia may develop, leading to unintentional weight loss, muscle wasting, weakness, and anorexia.[41] Cognitive decline in patients experiencing CKD is an emerging symptom revealed in research literature.[42][43][44][45] Research suggests that patients with CKD face a 35–40% higher likelihood of cognitive decline and or dementia.[42][43] This relation is dependent on the severity of CKD in each patient; although emerging literature indicates that patients at all stages of CKD will have a higher risk of developing these cognitive issues.[45][46][43] Sexual dysfunction is very common in both men and women with CKD. A majority of men have a reduced sex drive, difficulty obtaining an erection, and reaching orgasm, and the problems get worse with age. Most women have trouble with sexual arousal, and painful menstruation and problems with performing and enjoying sex are common.[47] The most common causes of CKD are diabetes mellitus, hypertension, and glomerulonephritis.[48][49] About one of five adults with hypertension and one of three adults with diabetes have CKD. If the cause is unknown, it is called idiopathic.[50] Vascular disease includes large-vessel disease such as bilateral kidney artery stenosis and small-vessel disease such as ischemic nephropathy, hemolytic–uremic syndrome, and vasculitis. Glomerular disease comprises a diverse group and is classified into:[citation needed] Primary glomerular disease such as focal segmental glomerulosclerosis and IgA nephropathy (or nephritis) Secondary glomerular disease such as diabetic nephropathy and lupus nephritis Tubulointerstitial disease includes drug- and toxin-induced chronic tubulointerstitial nephritis, and reflux nephropathy Obstructive nephropathy, as exemplified by bilateral kidney stones and benign prostatic hyperplasia of the prostate gland; rarely, pinworms infecting the kidney can cause obstructive nephropathy. Genetic congenital disease such as polycystic kidney disease or 17q12 microdeletion syndrome. Mesoamerican nephropathy, is "a new form of kidney disease that could be called agricultural nephropathy".[51] A high and so-far unexplained number of new cases of CKD, referred to as the Mesoamerican nephropathy, has been noted among male workers in Central America, mainly in sugarcane fields in the lowlands of El Salvador and Nicaragua. Heat stress from long hours of piece-rate work at high average temperatures[52][53][54][55] of about 36 °C (96 °F) is suspected,[56] as are agricultural chemicals[57] Chronic lead exposure A 12-lead ECG of a person with CKD and a severe electrolyte imbalance: hyperkalemia (7.4 mmol/L) with hypocalcemia (1.6 mmol/L). The T-waves are peaked and the QT interval is prolonged. Diagnosis of CKD is largely based on history, examination, and urine dipstick combined with the measurement of the serum creatinine level. Differentiating CKD from acute kidney injury (AKI) is important because AKI can be reversible. One diagnostic clue that helps differentiate CKD from AKI is a gradual rise in serum creatinine (over several months or years) as opposed to a sudden increase in the serum creatinine (several days to weeks). In many people with CKD, previous kidney disease or other underlying diseases are already known. A significant number present with CKD of unknown cause.[citation needed] Screening those who have neither symptoms nor risk factors for CKD is not recommended.[58][59] Those who should be screened include: those with hypertension or history of cardiovascular disease, those with diabetes or marked obesity, those aged > 60 years, subjects with African American ancestry, those with a history of kidney disease in the past, and subjects who have relatives who had kidney disease requiring dialysis.[citation needed] Screening should include calculation of the estimated GFR (eGFR) from the serum creatinine level, and measurement of urine albumin-to-creatinine ratio (ACR) in a first-morning urine specimen (this reflects the amount of a protein called albumin in the urine), as well as a urine dipstick screen for hematuria.[60] The GFR is derived from the serum creatinine and is proportional to 1/creatinine, i.e. it is a reciprocal relationship; the higher the creatinine, the lower the GFR. It reflects one aspect of kidney function, how efficiently the glomeruli – the filtering units – work. The normal GFR is >90 ml/min. The units of creatinine vary from country to country, but since the glomeruli comprise