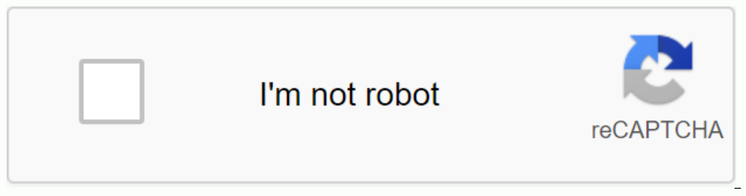

Continue

Hypertension emergency and urgency pdf

Up to 1% of patients with essential hypertension develop malignant hypertension, but the reason why some patients develop malignant hypertension, while others do not, is unknown. A characteristic vascular lesion is fibriroid necrosis of arteriol and small arteries, which causes clinical manifestations of damage to the end organs. Red blood cells are damaged because they flow through blood vessels, which are blocked by the deposition of fibrian, which leads to microangiopathic hemolytic anaemic. In a retrospective study evaluating hospitalization data from a nationwide hospitalization sample for malignant hypertension, hypertensive encephalopathy and essential hypertension, polgreen et al found a growing trend of malignant hypertension and hypertensive encephalopathy from 2000 to 2011, with a dramatic increase after 2007. [4] However, there was no corresponding dramatic increase in morbidity for both diseases, which investigators thought could be the result of changes in coding in diagnostic-related groups in 2007. Mortality decreased significantly in patients with malignant hypertension, but not in patients with hypertensive encephalopathy. [4] Another pathological process is the dilation of the cerebral arteries after the breakthrough of normal self-regulation of blood flow by the brain. Under normal conditions, blood flow through the brain is maintained by constant cerebral vasoconstriction in response to an increase in BP. In patients without hypertension, the flow rate is kept constant at an average pressure of 60-120 mm Hg. In patients with hypertension, the flow rate is constant above the average pressure of 110-180 mm Hg due to arteriolar thickening. When BP is raised above the upper limit of self-regulation, arterioles expand. This results in hyperperfusion and edema of the brain, which cause clinical manifestations of hypertensive encephalopathy. Other causes of malignant hypertension include any form of secondary hypertension; complications of pregnancy, i.e. cocaine use, monoamnooxidaze inhibitors (IMAO) or oral contraceptives; and the collection of alcohol, beta-blockers or alpha stimulants such as clonidine. Stenosis of the renal arteries, feochromocytoma (most feochromocytoma can be localized with the help of computed tomography [CT] of the adrenal glands), aortic coarctation and hyperaldosteronism are also secondary causes of hypertension. In addition, both hyperthyroidism and hypothyroidism can cause hypertension. When diagnosing, the following conditions should also be taken into account: ischemic or hemorrhagic stroke, intracranial mass, head injury, epilepsy or postictal condition, connective tissue disease (especially lupus with cerebral vasculitis), drug overdose or withdrawal, ingestion of cocaine or amphetamine, acute anxiety and thrombocytopenic purpura. [5, 6] Since malignant hypertension induced by thrombotic microanbiopathy may mimic thrombocytopenic there is a possibility that patients may initially be given plasma replacement rather than antihypertensive agents. In the khanal et al literature review, that the factors more likely to be present in malignant hypertension-induced thrombotic microanbiopathy are (1) previous history of hypertension, (2) high mean arterial pressure, (3) significant kidney damage, but relatively modest thrombocytopenia, and (4) deficiency of the severe adamts-13 gene (activity < 10%) diagnosis. [6] For more information, see Hypertension. Condition of significantly elevated blood pressure with diastolic pressure usually greater than 120 mm Hg Hypertensive emergencyNew namesMalignant hypertension, hypertensive crisisCT scan showing intracranial bleeding, possible complication of hypertensive emergency. Patients with spontaneous intracranial bleeding experience a newfound headache and neurological deficits. Special Cardiology Hypertensive emergency is high blood pressure with potentially life-threatening symptoms and signs indicating acute damage to one or more organ systems (brain, eyes, heart, aorta or kidneys). [1] Hypertensive urgency is defined as systolic blood pressure above 180 mmHg or diastolic blood pressure above 110 mmHg. Hypertensive alertness is defined as elevated blood pressure corresponding to hypertensive urgency, plus evidence of impending irreversible hypertension mediated organ damage (HMOD). Signs of organ damage will be described below. Signs and symptoms fundoscopic view of the eye with diabetic retinopathy. Similar to hypertensive retinopathy, evidence of infarction of nerve fibers due to ischemia (stains of cotton wool) can be seen on physical examination. Symptoms may include headache, nausea or vomiting. Chest pain can occur due to increased workload on the heart, resulting in insufficient oxygen supply to meet the metabolic needs of the heart muscle. The kidneys can be affected, resulting in blood or protein in the urine, and acute renal failure. People may have reduced urine production, fluid retention, and confusion. Other symptoms may include: Chest pain Abnormal heart rhythm Headache Nosebleeds that are difficult to stop shortness of breath fainting or feeling the world spinning around them (dizziness) Severe anxiety Agitation Altered mental state Abnormal sensations The most common presentation of hypertensive emergencies are cerebral infarctions (24.5%), pulmonary edema (22.5%), hypertensive encephalopathy (16.3%) congestive heart failure (12%). 2] Uncommon symptoms include intracranial bleeding, aortic dissection and preeclampsia or eclampsia. [3] A massive, rapid increase in blood pressure can trigger any of these symptoms, and require additional work-ups by doctors. Physical examination findings would be performed to measure blood pressure in both arms. Laboratory tests to be carried out include urine toxicology, blood glucose levels, a basic metabolic panel evaluating kidney function or a complete metabolic panel evaluating liver function, ECG, chest X-ray and pregnancy screening. [4] The eyes may show retina bleeding, exudation, cotton wool stains, diffuse chip haemorrhage, or swelling of an optical disc called papilledema. Causes Many factors and causes contribute to hypertensive crises. The most common cause is patients diagnosed with chronic hypertension who have discontinued treatment with hypertension. [5] Other common causes of hypertensive crises are autonomic hyperactivity, such as feochromocytoma, collagen-vascular disease, drug use, in particular stimulants, cocaine and amphetamines and their substituted analogues, monoaminexidase inhibitors or interactions between food and pharmaceuticals, spinal cord disorders, glomerulonephritis, head injuries, neoplasia, preeclampsia and eclampsia, hyperthyroidism and rescululonogenicity. [4] [5] People who withdrew from drugs such as clonidine or beta-blockers often found hypertensive crises. [6] It is important to note that these conditions exist outside the hypertensive emergency room, in that patients diagnosed with these conditions are at increased risk of hypertensive emergencies or failure of the final organs. Pathophysiology Kidney biopsy showing thrombotic microanbiopathy, histomorphological finding observed in malignant hypertension Pathophysiology hypertensive emergency is not well understood. Failure of normal self-regulation and a sudden increase in systemic vascular resistance are typical initial components of the disease process. [3] Hypertensive

emergency pathophysiology includes: sudden increase in systemic vascular resistance, Probably related to humoral vasoconstrictive injuries Endothelial injury and dysfunction Fibrinoid necrosis of arteriols Platelet deposition and fibrin The breakdown of normal autoregulatory function Resulting ischemia leads to further release of vasoactive substances including prostaglandins, free radicals and thrombotic/mitotic growth factors that end the vicious cycle of inflammatory changes. [3] If the process is not stopped, homeostatic failure begins, leading to loss of brain and local self-regulation, ischemia and dysfunction of the organ system and myocardial infarction. The involvement of one organ occurs in approximately 83% of hypertensive patients in need, the involvement of two organs in approximately 14% of patients, and multiorgan failure (failure of at least 3 organ systems) in approximately 3% of patients. In the brain, hypertensive encephalopathy - characterized by hypertension, altered mental state and swelling of the optical disc - is a manifestation of dysfunction of cerebral self-regulation. Cerebral self-regulation is the ability of blood vessels in the brain to maintain constant blood flow. People who suffer from chronic can tolerate higher arterial pressure before disrupting their self-regulatory system. Hypertension also has increased cerebrovascular resistance, which puts them at greater risk of developing cerebral ischemia if blood flow decreases to the normotensive range. On the other hand, a sudden or rapid increase in blood pressure can cause hyperperfusion and increased blood flow through the brain, causing increased intracranial pressure and cerebral edema, with an increased risk of intracranial bleeding. [4] Increased arterial stiffness, increased systolic blood pressure and enlarged pulse pressures, all due to chronic hypertension, can cause significant damage in the heart. Coronary perfusion pressures are reduced by these factors, which also increase the oxygen consumption of the myocardium, which can lead to hypertrophy of the left ventricle. Since the left ventricle is unable to compensate for the acute increase in systemic vascular resistance, left ventricular failure and pulmonary edema or myocardial ischemia may occur. [3] In the kidneys, chronic hypertension has a large effect on the vasculature of the kidneys, which leads to pathological changes in the small arteries of the kidneys. In the affected arteries, endothelial dysfunction and deterioration of normal vasodilatation develop, which changes the self-regulation of the kidneys. When the self-regulatory system of the kidneys is disturbed, intraglomerular pressure begins to change directly with systemic arterial pressure, thereby offering no protection of the kidneys during fluctuations in blood pressure. The Renin-aldosterone-angiotensin system can be activated, leading to further vasoconstrition and damage. During hypertensive crisis, this can lead to acute kidney ischemia, hypoperfusion, involvement of other organs and subsequent dysfunction. After an acute event, this endothelial dysfunction persists for years. [3] Diagnosis The term hypertensive emergency is mainly used as a specific term for hypertensive crisis with diastolic blood pressure higher than or equal to 120 mmHg or systolic blood pressure higher than or equal to 180 mmHg. [7] Hypertensive alertness differs from hypertensive urgency in that in the first case there is evidence of acute organ damage. [7] Both of these definitions were collectively known as malignant hypertension, although this medical term is replaced. In a pregnant patient, the definition of hypertensive emergency (probably secondary to pre-eclampsia or eclampsia) is only blood pressure higher than 240 mmHg of systolic blood pressure or diastolic blood pressure of 140 mmHg. [8] Treatment In case of hypertensive emergency, treatment should first stabilize the patient's airways, breathing and blood circulation as directed by the ACLS. Patients should have their blood pressure slowly lowered for several minutes to hours with an antihypertensive agent. Documented blood pressure targets include a reduction in the mean pressure by less than or equal to 25% during the first 8 hours of emergency. [4] If blood pressure is lowered aggressively, patients are at increased risk of complications including stroke, blindness, or kidney failure. [3] Several classes of antihypertensive agents are recommended, the choice depending on the cause of the hypertensive crisis, the severity of the increase in blood pressure and the patient's underlying blood pressure before hypertensive emergency. Doctors will try to identify the cause of hypertension of the patient, including chest X-ray examination, serum laboratory studies evaluating kidney function, urine analysis, as this will change the treatment approach for a more patient-driven regimen. Hypertensive emergencies differ from hypertensive urgency in that they are treated parenterally, while in an emergency it is recommended to use oral antihypertenves to reduce the risk of hypotensive complications or ischemia. [3] Parenteral agents are classified into beta-blockers, calcium channel blockers, systemic vasodinators or others (phenoldopam, phentolamine, clonidine). Drugs include Labetalol, Nicardipine, Hydralazine, Nitroprusside sodium, Esmolol, Nifedipine, Minoxidil, Isradipine, and Clonidine. These drugs work through various mechanisms. Labetalol is a beta-blocker with mild alpha antagonism, reducing the ability of catecholamine activity to increase systemic vascular resistance, while reducing heart rate and myocardial oxygen demand. Nifedipine, Nifedipine and Isradipine are calcium channel blockers that reduce systemic vascular resistance and subsequently lower blood pressure. Hydralazine and sodium nitropruside are systemic vasodinators, thereby reducing recharging, however, reflex tachycardia can be found, which is probably the second or third line of choice. Sodium nitropruside was previously the first choice due to its rapid onset, although now it is less often used due to side effects, drastic drops in blood pressure and cyanide toxicity. Sodium nitropruside is also contraindicated in patients with myocardial infarction due to coronary steal. [6] Again, it is important that blood pressure decreases slowly. The initial goal in hypertensive emergency situations is to reduce the pressure by no more than 25% of the average arterial pressure. Excessive lowering of blood pressure can expel coronary, cerebral, or kidney ischemia and, perhaps, a heart attack. Hypertensive alertness is not only based on the absolute level of blood pressure, but also on the patient's basic blood pressure before hypertensive crisis. Individuals with a history of chronic hypertension may not tolerate normal blood pressure and therefore may pose symptomatically with hypotension, including fatigue, headache, nausea, vomiting, or syncope. Blood pressure targets[4] <1 hr 25% at average arterial pressure, diastolic blood pressure above 100-2-6 hours Systolic BP < 160 mmHg or Diastolic BP <110 mmHg 6-24 hrs to ensure that bp does not drop quickly below 160 SBP or 100 DBP 1-2 d, unless the end organs have been damaged, amcient monitoring and JNC8 guidelines to maintain BP control Prognosis Severe hypertension is a serious and potentially life-threatening medical condition. It is estimated that people who do not receive appropriate treatment live on average about three years after the event. [8] Morbidity and mortality of hypertensive emergencies depend on the extent of end organ dysfunction at the time of presentation and the extent to which blood pressure is controlled later. With good blood pressure control and drug adherence, the 10-year survival rate for patients with hypertensive crises is close to 70%. [1] The risks of developing life-threatening diseases affecting the heart or brain increase as blood flow increases. Usually, ischemic heart attack and stroke are causes that lead to death in patients with severe hypertension. It is estimated that for every 20 mm Hg systolic or 10 mm Hg diastolic increase in blood pressure above 115/75 mm Hg, mortality from both coronary heart disease and stroke doubles. Consequences of hypertensive emergency result after prolonged increase in blood pressure and associated dysfunction of the end organs. Acute damage to the end organs may occur, affecting neurological, cardiovascular, renal or other organ systems. Some examples of neurological damage include hypertensive encephalopathy, cerebral vascular accident/cerebral infarction, subarachnoid hemorrhage, and intracranial bleeding. Damage to the cardiovascular system may include ischemia/myocardial infarction, acute left ventricular dysfunction, acute pulmonary edema and aortic dissection. Other end organ damage may include acute renal failure or insufficiency, retinopathy, eclampsia, and microandiopathic haemolytic anemia. Epidemiology In 2000, it was estimated that approximately 1 billion people worldwide have hypertension, making it the most wide-used condition in the world. [2] Approximately 60 million Americans suffer from chronic hypertension, with 1% of these individuals having an episode of hypertensive urgency. In emergency departments and clinics throughout the United Years, it is suspected that the prevalence of hypertensive urgency varies between 3-5%. [6] It was found that 25% of hypertensive crises are hypertensive emergency situations versus urgency in the presentation of er. [8] Risk factors for hypertensive emergency include age, obesity, non-compliance with hypertensive drugs, female sex, caucasian race, pre-existing diabetes or coronary artery disease, mental illness and sedentary lifestyle. [2] Several studies have concluded that African-Americans have a higher incidence of hypertension and hypertensive disease than non-Hispanic caucasian people, but hypertensive crises are more common in caucasian people. [9] Although severe hypertension is more common in the elderly, it may occur in children (although very rarely), probably due to metabolic or hormonal dysfunction. In 2014, a systematic review identified women who had a slightly higher risk of developing hypertensive crises than men. [2] With use against hypertension, the rate of hypertensive emergencies decreased from 7% to 1% in patients with hypertensive urgency. [2] 16% of patients with hypertensive emergency may have no known history of hypertension. [3] See also Hypertensive Retinopathy Hypertensive Encephalopathy Preeclampsia Eclampsy Aortic Dissection Intracerial Bleeding Reference ^ a b Thomas L (October 2011). Coping with hypertensive emergencies in ED. Canadian family physician. 57 (10): 1137-97. PMC 3192077. PMID 21998228. ↑ a b c d e Pak KJ, Hu T, Fee C, Wang R, Smith M, Bazzano LA (2014). Acute hypertension: systematic review and evaluation of the guidelines. Ochsner's journal. 14 (4): 655-63. PMC 4295743. PMID 25598731. A summary of the recommendations from the selected guidelines is given in Table 2. ↑ a b c d e f g h Cline, David M.; John Ma, O.; Meckler, Garth D.; Tintinalli, Judith E.; Stephan Stapczynski, J.; Yealy, Donald (2015-11-10). Tintinalli emergency medicine : a comprehensive study guide. Tintinalli, Judith E.,, Stapczynski, J. Stephan,, Ma, O. John,, Yealy, Donald M.,, Meckler, Garth D.,, Cline, David, 1956- (eighth ed.). New York. ISBN 9780071794763. OCLC 915775025. ↑ a b c d e Larry Jameson, J.; Fauci, Anthony S.; Kasper, Dennis L.; Hauser, Stephen L.; Longo, Dan L.; Loscalzo, Joseph (2018-08-13). Harrison's principles of internal medicine. Jameson, J. Larry,, Kasper, Dennis L.,, Longo, Dan L. (Dan Louis), 1949-, Fauci, Anthony S., 1940-, Hauser, Stephen L.,, Loscalzo, Joseph (20. ed.). New York. ISBN 9781259644030. OCLC 1029074059. ↑ a b Saguner AM, Dür S, Perrig M, Schiemann U, Stuck AE, Bürgi U, Erne P, Schoenenberger AW (July 2010). Risk factors supporting hypertensive crises: evidence from a longitudinal study. American Journal of Hypertension. 23 (7): 775-80. doi:10.1038/ajh.2010.71. PMID 20395943. ↑ a b c Campos CL, Herring CT, Ali AN, Jones DN, Wofford JL, Caine AL, Bloomfield RL, Tillett J, Oles KS (April 2018). Pharmacological treatment of hypertensive urgency in an outpatient environment: a systematic review. Journal of General Internal Medicine. 33 (4): 539-550. doi:10.1007/s11606-017-4277-6. PMC 5880769. PMID 29340938. ↑ a b Whelton PK, Carey RM, Aronow WS, Casey DE, Collins KJ, Dennison Himmelfarb C, et al. (June 2018). 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APHA/ASH/ASPC/NMA/PCNA Guidelines for The Prevention, Detection, Evaluation and Management of High Blood Pressure in Adults: A American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Hypertension. 71 (6): e13-e115. doi:10.1161/HYP.0000000000000000000065. PMID 29133356. ↑ a b c Walls, Ron; Hockberger, Robert; Gausche-Hill, Marianne (2017-03-09). Rosen Emergency Medicine : concepts and clinical practice. Walls, Ron M.,, Hockberger, Robert S.,, Gausche-Hill, Marianne (ninth ed.). Philadelphia, Pa. ISBN 9780323390163. OCLC 989157341. ↑ Howard J (1965). Racial Differences in Hypertension Mortality Trends: Differential Exposure to The Drug as a Theory. Systemic hypertension. Milbank Fund Mem Q. 43 (2): 202-218. JSTOR 3349030. External Links ClassificationDICD-10: I10ICD-9-CM: 401-405MeSH: D006974DiseasesDB: 7788External resourcesMedlinePlus: 000491eMedicine: article/241640Pacient UK: Hypertensive Emergency Retrieved from