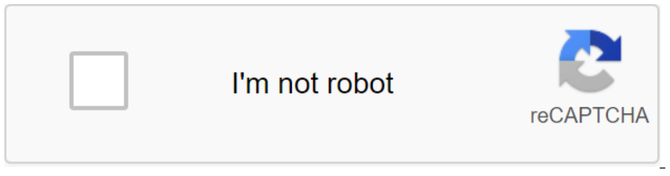

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Anemia megaloblastik pdf

Most megaloblast anemia is caused by vitamin B12 or folic acid deficiency. It is important to determine what shortcomings are the cause in each case. In an emergency where it can be dangerous, it is sometimes important to give both medications after a bone marrow examination in anticipation of the results of a plasma examination. In general, however, treatment should be done only when the results are already there. The cause of meloblast anemia (pernical anemia) is the absence of gastric internal factors due to autoimmune gastritis, which causes vitamin B12 malabosorbys. Vitamin B12 is also needed to treat megaloblastosis due to prolonged anesthesia of nitric oxide, which activates this vitamin, and the treatment of congenital transcobalamin deficiency II, a rare syndrome. Vitamin B12 should be administered as a preventive measure after a general gastrectomy or total underlying resection (or after a partial gastrectomy indicating vitamin B12 malabosortion). In addition to dietary deficiencies, another cause of vitamin B12 deficiency is malabsorption so there is very little reason to use low doses of oral vitamin B12 and there is absolutely no reason to use complex internal vitamin B12 oral factors. Hydroxyhobalamin replaced cyanokobomalamine as a selected form of vitamin therapy; This substance persists in the body longer than cyanokobomalmine and can therefore be administered every 3 months for supporting therapy. Usually treatment begins with intramuscular injection to replace the lost reserves of the body. Maintenance can then be carried out. There is no evidence that large doses provide additional benefits of vitamin B12 neuropathy. Folic acid for long-term treatment is only marginally necessary because most of the causes of folic acid deficiency end on their own, or can be solved with short-term treatment. This vitamin should not be used for undiagnosed megaloblastic anemia if vitamin B12 is administered simultaneously because otherwise it may cause neuropathy (see above). In meloblast anemia, folic acid deficiency (e.g. due to poor nutrition, pregnancy, antiepileptic) standard treatment to cause henotological remission and replace the body reserve, is the oral administration of folic acid 5 mg daily for 4 months; Malabsorbtion may require up to 15 mg per day. During pregnancy, folic acid at a dose of 5 mg is transmitted before birth. For prevention in chronic hemolyte or renal dialysis, simply inject folic acid 5 mg daily or even weekly, depending on the consumption and rate of hemolite. For the prevention of folic acid doses during pregnancy 200-500 micrograms per day (see iron and folic acid, see See also Neural Tube Defect Prevention below. Folic acid is actively excreted mother's milk and well-breasted baby. Folic acid is also found in cow's milk and milk formula, but it does not withstand the heat. Levels of folic acid in the baby's serum and a drop in blood after childbirth process and in large amounts through urine, especially in babies born with low weight. Folic acid is also effective in treating megaloblast anaemia deficiency of folic acid, but the drug is usually used in connection with cytotoxic drugs (see 8.1); as calcium folic acid. WARNING NEURAL TUBE DEFECT To prevent recurrence of neural tube defects (in children of men or women with spina bifida or history of neural tube defects in previous children), women who want pregnancy (or who risk becoming pregnant) should be advised to use folic acid supplements at a dose of 5 mg daily (reduced to 4 mg daily when appropriate doses are available); supplements should continue until a week of pregnancy. For women receiving antiepileptic drugs, you should see a doctor before you start eating folic acid. To prevent neural tube defects for the first time in women planning pregnancy, it should be recommended to eat folic acid as a medicine or food additive at a dose of 400 micrograms daily before conception and in the first twelve weeks of pregnancy. women who have not received the supplement and believe that they are pregnant should immediately start eating supplements and continue until the t11th week of pregnancy. The daily need for comalamine is about 5-7 micrograms/. Dietary deficiency of comalamine rarely causes megaloblast anemia, except for strict vegetarians who avoid meat, eggs and dairy products. Atrophic gastritis and acchlorhydria are commonly found in the elderly. These conditions are the cause of the disruption of the release of mosquitoes associated with food and, consequently, the presence of cobalamines. This is a common problem in older people. There is a failure in the production of internal factor (IF) in pernicious anemia, due to autoimmune destruction of the gastric parietal cell. Pernicious anaemia is the most known cause of cobalt deficiency. Significant amounts of comalamine are not absorbed in the absence of IF. In 1% of people over 60 years of age is diagnosed with harmful anemia. The incidence in women is slightly higher than in men. It should be noted that H2 antagonists may interfere with IF secretion. If the pancreas is insufficiency, the alkaline environment in the small intestine is not enough to release comalamine from R-proteins and bind to internal factors. In Sollinger-Ellison syndrome, the acidic environment also prevents comalamine from binding to the internal factor. In both conditions, a decrease in connection with the internal factor prevents the absorption of comalamine. Terminal ilay violations can lead to deficits Because that Ileum is a place of absorption of comalamine-IF complexes, tropical lithium, inflammatory bowel disease, lymphoma, and ileal resection can lead to a deficiency of comalamine. Tropical lithium is more serious than non-tropical spru (coeliac disease) and can be associated with both comalamine and folic acid deficiencies. It will take several years for the cobaltmine deficiency to develop after the onset of these disorders due to the time it takes to deplete the stocks of comalamine. In Imerslund-Grasbebe syndrome, there is an autoimmune destruction of the ileal receptor, cubulin, to absorb comalamine associated with an internal factor. Blind Loop Syndrome can lead to comalamine deficiency. Bacterial colonization can occur in the intestine, deformed by strict requirements, surgical blind loops, scleroderma, inflammatory bowel disease or amyloidosis. The bacteria then compete with the host for comalamine. Fish tapeworm Diphyllobothrium latum can compete with the owner for swallowed comalamine. This organism is most commonly found in Canada, Alaska and the Baltic Sea. Exposure to nitrous oxide can cause megaloblastosis by oxidative inactivation of comalamine. Prolonged exposure to nitrous oxide can lead to severe mental and neurological disorders. Details of hereditary disorders are beyond the scope of this review, but information can be found in other links. A partial list of medications that can cause comalamine deficiency includes purine analogues (6-mercaptopurine, 6-thioguanin, acyclovir), pyrimidine analogues (5-fluorouracil, 5-asazcitydin, zidovudine), inhibitors of ribonucletide redukase (hydroxyouarea, cytarabina arabinoside) and drugs that affect the metabolism of cobalamin (p-aminesalic acid, fenformin, metformin). The daily need of adults is about 0.4 mg/d. Storage is limited, and folic acid deficiency develops approximately 3-4 weeks after cessation of folic acid intake. Folic acid content in food and food preparation are the main causes of folic acid deficiency, especially in older people. The folates are very thermolabous. Thus, excessive heating can lead to inactivation, especially when foods are over-diluted in water. In the United States, most people get enough folic acid from fortified foods. However, alternative diets may contain little folic acid. Increased demand can lead to shortages. There is an increased need for folic acid in hemolysis, pregnancy, lactation, rapid growth, hyperalimentation, renal dialysis, psoriasis and exfoliating dermatitis. Intestinal disorders that prevent folic acid absorption include tropical spry, ingots (coeliac disease or sensitivity to gluten), amyloidosis, and inflammatory bowel disease. In alcoholism, the bioavailability of folic and folic biochemical reactions may be impaired. A partial list of drugs that can cause folic acid deficiency includes phenytoin, metformin, phenobarbital, dihydrofolate redukase (trimethoprem, pyrimethamine), methotrexate and other antifolates, sulfonamides (competitive inhibitors of 4-aminobenzoic acid) and valproic acid. Details of hereditary disorders that cause folic acid deficiency go beyond this review, but information can be found in other references). Megaloblast disease in HIV and myelodysplastic disorders is caused by direct effects on DNA synthesis in hemathopetic and other cells. Megaloblast anemia is a type of anemia characterized by abnormal red blood cell pieces and larger sizes. Normal red blood cells should be in the form of flat round discs that are slightly styed in the middle. However, in the case of this anemia, pieces of red blood cells are oval-shaped. This abnormal shape and size is because red blood cells do not experience cleavage and do not develop perfectly. As a result, a normal and healthy amount of red blood cells is not enough. This blood disorder also causes the bone marrow to produce fewer cells. Normal red blood cells can usually last about 90-120 days before finally being destroyed by the body to be replaced by a new one. However, in these cases, red blood cells sometimes break down or die earlier than they should. How common is this condition? Megaloblast anemia is a condition that can occur in men and women of any racial or ethnic background. However, it is not yet known exactly how many people in the world have this type of anemia. Characteristics of megaloblast anemia are similar to those of anemia in general, such as weakness and fatigue, dizziness and sickening skin. On the other hand, some people may not show clear symptoms. A quote from the Mayo Clinic, the common symptoms of megaloblastic anemia are: Numbness of breathing at each end of the body; for example, the tips of the fingers and toes of the swollen tongue Of Muscle Skin Cramps looks like a pale loss of appetite and a dramatic weight loss. Heartbeat Hands and Leg Tremor Some symptoms of megaloblast anemia associated with digestive problems can cause nerve damage. If left for a long time, this condition can cause a decrease in bone density and the development of stomach cancer. One of the things that distinguishes this type of anemia is the variety of causes. The main cause of megaloblast anemia is a lack of intake of vitamin B12 and folic acid (vitamin B9). Vitamin B12 and folic acid are included as the main ingredients of red blood cell solutions. Deficiency of these two nutrients can lead to the fact that the bone marrow will not be able to produce healthy and normal blood components in sufficient quantities. This also results in red blood cells being produced abnormally in shape and size. These are damaged or formed red blood cells will die earlier healthy environment. The absence of red blood cells leads to a decrease in the level of haemoglobin in the blood. In fact, hemoglobin plays an important role in binding oxygen and nutrients to blood cells to then drain them throughout the body. Not only red blood cells, megaloblastic anemia also makes granulocytes (white blood cells that have pellets in the cytoplasm) and platelets also decreases. Reported from the National Center for Biotechnology Information, in rare cases, this type of anemia occurs due to hereditary, such as: Megaloblastic anemia syndrome, which responds to thiamine (vitamin B1), a disease characterized by megaloblastic anemia associated with hearing loss and diabetes. Imerslund-Grasbeck syndrome, i.e. the absence of factors that come from within or receptors in the intestine. Errors in the absorption of folic acid are transmitted in infants. One of the things that causes megaloblast anemia is vitamin B12 deficiency and folic acid. In addition, there are several factors that make you more at risk of megaloblast anemia, namely: 1. Eating less foods high in vitamin B12 vitamin B12 deficiency can cause your spinal cord not to be able to produce healthy blood cells. People who rarely eat red meat, chicken, fish, eggs and milk, or vegetarians, are at risk of megaloblast anemia. 2. Less consumption of folic acid Eating green vegetables such as spinach or mustard, or animal products can cause the body to experience folic acid deficiency. Inappropriate cooking, such as boiling vegetables for too long with overheating temperatures can damage folic acid content. 3. Nutritional absorption disorders Absorption disorder can make you malnourished even if you ate foods containing vitamin B12 and folic acid and cause megaloblastic anemia. This is because your body cannot absorb vitamins properly. Usually this condition can be caused by a decrease in protein in the stomach, which helps to absorb vitamin B12. Autoimmune diseases, bacterial infections and parasitic worm infections will also make vitamin B12 levels more difficult to absorb. In particular, megaloblast anemia due to vitamin B12 deficiency is known as pernicia anemia. Meanwhile, folic acid can usually be more difficult for the body to absorb due to certain factors. For example because you have a history of drinking excessive alcohol, or pregnant. 4. Medical conditions there are several other diseases that may be triggers for megaloblast anemia. Among others: Leukemia HIV Infection Myelodisplasia Myelofibrosis Syndrome Using Epilepsy Anti-Addiction Drugs Using Chemotherapy Drugs Symptoms can worsen the quality of life and health of patients over time. Therefore, anemia should not immediate appeal. There are different ways of diagnosing the type of anemia. Here's how doctors treat and diagnose megaloblast anemia: 1. Full blood tests can be used to diagnose different types of anemia. This test will measure the different components and the amount of your blood. In addition, the doctor can check the number and appearance of your red blood cells. Blood cells that seem larger and less developed may be a sign that you have megaloblast anemia. Your doctor will also collect your medical history and perform a physical examination to rule out other causes of your symptoms. 2. Your doctor's vitamin level test should do more blood tests to find out what is causing your anemia. This additional blood test will also help your doctor find out if your anemia is caused because it is a deficiency of vitamin B12 or folic acid. 3. Schilling test Is a Schilling blood test that evaluates your ability to absorb vitamin B-12. First, it is recommended to take radioactive supplements of vitamin B12. You will then be asked to take a urine sample for analysis. You will again be asked to take the same radioactive supplement in combination with the protein internal factor. This factor requires your body to be able to absorb vitamin B-12. The doctor will again ask that your urine sample be compared to the first sample. If urine does not contain internal factors, it indicates that your body absorbs B12 only when consumed with internal factor proteins. This means that your body cannot absorb vitamin B12 naturally. Knowing the symptoms and causes of these types of anemia can make it easier for you to get the right treatment. The goal of treating megaloblast anemia is to prevent symptoms from getting worse, avoid complications due to anemia, and addressing the most basic causes, namely vitamin B12 deficiency, as well as folic acid. Some of the treatment options for megaloblast anemia that you can go through, among other things: 1. Adding vitamin B-12 intake In the case of meloblast anemia caused by vitamin B-12 deficiency, you may need vitamin B-12 injections each month. Based on a doctor's review, as well as the condition of anemia, injections can be administered for up to a full year. In addition, you can also take vitamin B-12

supplements in a dose that has been prescribed by your doctor. You can also multiply eating foods containing vitamin B-12 on your daily menu, such as: Chicken Eggs Cereal enriched with vitamin B12 Red meat (especially beef) Milk Molluscs Some people have genetic mutations in the MTHFR gene (methylentetrahydrofolate reductase). This gene is responsible for the processing of certain B vitamins, B12 and folate become usable forms in the body. People who have a mutation of the MTHFR gene are advised to take additional methylcobalamin supplements to prevent anemia from getting worse. 2. Adding folic acid intake to megaloblast anemia due to lack of folic acid can be treated by regularly taking folic acid supplements or getting infusion of folic fluid. Foods high in folic acid can also help prevent anemia from getting worse. Here are some foods that are well consumed to overcome megaloblast anemia due to folic acid deficiency: Citrus fruits Dark green leafy vegetables Whole grains equal to vitamin B12 deficiency, people who experience mthfr gene mutations are advised to use additional methylfolates to prevent folic acid deficiency and its risk. You can take precautions to avoid anemia or prevent symptoms from recurring. People with megaloblast anemia due to vitamin B12 or folic acid deficiency can overcome their symptoms and feel better with the following current treatments: Many eat iron-rich foods such as toy, green vegetables, lean red meat, lentils, legumes, iron-enriched cereals and bread. Eat and drink foods and beverages rich in vitamin C. Avoid drinking tea or coffee along with food as it may affect the absorption of iron and other vitamins Get enough vitamin B12 and folic acid in your diet. Vitamin B12 and folic acid deficiency can cause not only anemia, but also health problems such as nerve damage, neurological problems and gastrointestinal problems. These complications can be avoided if you are diagnosed and treated at an early stage. Genetic tests can also be performed to detect possible mutations in the MTHFR gene. This can be done as a first step towards early detection of megaloblast anemia. Talk to your doctor if you see signs of anemia so that you and your doctor can make a treatment plan and help prevent irreversible damage. Hello Health Group and Hello Sehat do not provide medical advice, diagnosis or treatment. Please check our editorial policy page for more information. Does this article help you? Fasting can exacerbate symptoms that often experience anemia. If so, should anaemia patients take iron supplements during fasting? Review: Dr. Patricia Lucas Goentoro Raya, Ramadan May 6, 2020. Read the time of 5 minutes One of the common efforts to prevent anemia is iron consumption. However, not only that, there are vitamins and other ways that can also prevent anemia. Review: Dr. Michael Yosia Author: Fajarina Nurin Anemia, Medical Centers February 26, 2020 . Read the time of 7 minutes Gravis anemia a type of severe anemia that can cause complications in the form of organ damage if left untreated Review: Dr. Michael Josiah Author: Novita Joseph A-I Disease, A-I Health January 16, 2020. Reading time 7 minutes anemia megaloblastik pdf. anemia megaloblastik adalah pdf. anemia megaloblastik mcv. anemia megaloblastik jurnal. anemia megaloblastik pada ibu hamil. anemia megaloblastik dan non megaloblastik. anemia megaloblastik shqip. anemia megaloblastike eshte