



DETERMINATION OF PREGNANCY-RELATED HISTO-TOPOGRAPHIC FEATURES OF THE LIVER OF WHITE PUREBRED RATS IN EXPERIMENTAL CHRONIC RENAL FAILURE

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Annotation

Chronic kidney disease is often clinically and biochemically silent until renal impairment is advanced. [2]. Symptoms are unusual until the glomerular filtration rate declines to <25% of normal, and more than 50% of renal function can be lost before serum creatinine rises above 120 $\mu\text{mol/l}$. [17;18]. Women who become pregnant with serum creatinine values above 124 $\mu\text{mol/l}$ have an increased risk of accelerated decline in renal function and poor outcome of pregnancy (see Scenario box). Several factors must be considered when managing pregnant women with chronic kidney disease to minimise the adverse effects of pregnancy on maternal renal function and the consequent effects on the fetus. [1].

Keywords: function of sodium, morphometric changes, local glomerulosclerosis, membranous, membranousproliferative glomerulonephritis.

Аннотация

Хроническая болезнь почек часто протекает клинически и биохимически бессимптомно до тех пор, пока почечная недостаточность не достигнет прогрессирующей стадии. [5;6]. Симптомы необычны до тех пор, пока скорость клубочковой фильтрации не снизится до <25% от нормы, и более 50% функции почек может быть утрачено до того, как уровень креатинина в сыворотке поднимется выше 120 мкмоль/л. Женщины, которые забеременели при уровне креатинина в сыворотке крови выше 124 мкмоль/л, имеют повышенный риск ускоренного снижения функции почек и неблагоприятного исхода беременности. При ведении беременных женщин с хронической болезнью почек необходимо учитывать несколько факторов, чтобы свести к минимуму неблагоприятное воздействие беременности на функцию почек матери и последующее воздействие на плод. [3;4].





КЛЮЧЕВЫЕ СЛОВА: функция натрия, морфометрические изменения, локальные гломерулосклероз, мембранозный, мембранозно-пролиферативный гломерулонефрит.

Annotatsiya

Surunkali buyrak kasalligi ko'pincha buyrak yetishmovchiligi rivojlanmaguncha klinik va biokimyoviy o'zgarishlarsiz bo'ladi. Simptomlar glomerulyar filtratsiya tezligi me'yordan 25% dan kamayguncha noodatiy bo'ladi va qon zardobida kreatinin 120 mkmol/l dan oshguncha buyrak funksiyasining 50% dan ortig'i yo'qolishi mumkin. Qon zardobidagi kreatinin miqdori 124 mkmol/l dan yuqori bo'lgan homilador bo'lgan ayollarda buyraklar faoliyatining tez pasayishi va homiladorlikning yomon oqibati xavfi ortadi. Surunkali buyrak kasalligi bo'lgan homilador ayollarni davolashda homiladorlikning onaning buyrak funksiyasiga salbiy ta'sirini va natijada homila ta'sirini minimallashtirish uchun bir nechta omillarni hisobga olish kerak.

Kalit So'zlar: natriy funksiyasi, morfometrik o'zgarishlar, local glomeruloskleroz, membranali, membranali proliferativ glomerulonefrit.

Introduction

Chronic kidney disease includes a wide range of different conditions, and monitoring during pregnancy must be tailored to the severity of the disease and its complications. In general, all clinical and biochemical features should be checked more often as pregnancy progresses or if changes suggest deteriorating kidney function. Specialist care should begin early in pregnancy, but much of the monitoring of women with stage 1-2 disease can be done by primary care doctors.

Chronic kidney disease (CKD)—or chronic renal failure (CRF), as it was historically termed—is a term that encompasses all degrees of decreased kidney function, from damaged—at risk through mild, moderate, and severe chronic kidney failure. [9;10]. CKD is a worldwide public health problem. In the United States, there is a rising incidence and prevalence of kidney failure, with poor outcomes and high cost. [15;16]. All women with chronic kidney disease should be referred early in pregnancy to an obstetrician and other specialist as necessary, to plan subsequent antenatal care. However, with a few exceptions, the most important aspects of managing chronic kidney disease in pregnancy relate to managing associated clinical features, rather than the type of kidney disease. [7;8]. Regular monitoring of maternal renal function (serum creatinine and serum urea), blood pressure, midstream urine (for





infection), proteinuria, and when appropriate ultrasound (to detect urological obstruction) should identify pathological changes and allow timely intervention to optimise perinatal outcome and maternal renal outcome.

Materials and methods

The study of changes in the morphological and morphometric parameters of the liver of pregnant white rats after chronic renal failure was investigated separately.

1. Study of morphological and morphometric parameters of the liver of pregnant white rats.
2. Reactive changes in the anatomic and morphological parameters of the liver of pregnant white rats after experimental chronic kidney failure;
3. Determination of pregnancy-related histo-topographic features of the liver of purebred rats in experimental chronic kidney failure;
4. Comparative classification of morphometric changes in the liver of pregnant purebred rats with experimental chronic renal failure after correction with Joyzar waters.

Discussion (conclusions):

Glomerulopathies: focal glomerulosclerosis, membranous and membranous-proliferative glomerulonephritis are primarily damage to the kidney balls. Due to the alteration of the basal membrane of the glomerulus, increased permeability leads to proteinuria. . [10;11]. Prolonged proteinuria results in decreased albumin in the blood plasma (Hypoalbuminemia). The quality of albumin and globulin changes. A decrease in albumin leads to a decrease in osmotic pressure. The passage of fluid from the blood to the tissues increases. The function of sodium and potassium pumps is disturbed. Swelling develops. At the same time, the reduction of albumin leads to an increase in the synthesis of lipoproteins. This has an effect on the oncotic pressure. The release of a large amount of lipids in the urine is a sign of impaired permeability of the glomerular basement membrane. Such changes in the body have also caused complications in the liver. Toxic processes in liver hepatocytes, hypoproteinemia and violation of water-electrolyte balance lead to increased permeability of the hepatocyte membrane, malfunction of sodium and potassium pumps, violation of water homeostasis, violation of osmotic pressure: the fluid in the blood vessel from the sinusoids to the space of Disse (expansion)) and it was determined that parenchymatous water-protein (hydropic) dystrophy was caused by the passage of fluid from the extracellular space to hepatocytes, the shrinking and displacement of the nucleus, the increase in the amount of water with vacuoles of





different sizes in the cytoplasm, and the disintegration of the endoplasmic reticulum. [12]. The passage of hypoxic, ischemic processes, deformation of the wall of the central vein (sclerotic changes), narrowing of the fullness and space (in dimensions), inflammatory infiltrate around it and irregular thickening of delicate pale pink collagen fibers are noticeable. . As a result of chronic kidney failure, intoxication, i.e. poisoning; . [13;14].

1. We can see that the number of Kupffer cells is reduced.

2. Uneven fat filling of hepatocytes located at the edges of liver lobes was found.

This picture is reminiscent of a false nutmeg liver. It is called fatty liver dysentery and long-term healing leads to fatty cirrhosis of the liver. Hepatocytes in the central part of the liver lobes are occupied by the compensatory process; (We can call it compensatory hepatocytes) we can see a small number of hepatocytes with an enlarged nucleus (in size), hyperchromic staining, wide cytoplasm, light pink color.

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