

**МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ
БУКОВИНСЬКИЙ ДЕРЖАВНИЙ МЕДИЧНИЙ УНІВЕРСИТЕТ»**



МАТЕРІАЛИ

**104-ї підсумкової науково-практичної конференції
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Конференція внесена до Реєстру заходів безперервного професійного розвитку,
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посилюються або тривають більше 14 днів, призначають повторний візит з метою подальшого обстеження. Якщо кров'яні виділення припиняються - надалі застосовується рутинна антенатальна допомога. Вагітним, які мають в анамнезі більше як один викидень і теперішня вагітність ускладнилась загрозливим абортom із кровотечею, рекомендовано призначення вагінального мікронізованого прогестерону (vaginal micronized progesterone) 400 мг двічі на добу. За підтвердженого серцебиття плода прийом прогестерону має тривати до 16 повних тижнів вагітності. Менеджмент вагітних із звичним невиношуванням PRISM [2021] теж є диференційованим. Зокрема, за наявності в анамнезі від до 3 викиднів, при відсутній кровотечі пацієнткам рекомендовано звичайне спостереження вагітності. Якщо в анамнезі 4 і більше викиднів, навіть при відсутності кровотечі, необхідно застосувати вагінальний мікронізований прогестерон 400 мг двічі на день з метою профілактики НВ.

Висновки. Результати дослідження Аппі Кумарасамі (2019) довели, що запропоноване лікування із застосуванням вагінального мікронізованого прогестерону достовірно збільшує частоту живонародження при загрозі викидня у жінок із втратами вагітності в анамнезі, у тому числі, у підгрупі звичного невиношування (72% порівняно із групою плацебо 57%). Відповідно до нової моделі надання допомоги жінкам із невиношуванням, вже після першого викидня необхідно надавати інформаційну підтримку і рекомендації щодо ведення майбутніх вагітностей. При звичному невиношуванні медична допомога повинна надаватися у спеціалізованій клініці. При трьох викиднях обов'язковим є генетичне тестування, виключення антифосфоліпідного синдрому і тромбофілій.

СЕКЦІЯ 12 СУЧАСНА ДІАГНОСТИКА ТА ЛІКУВАННЯ НЕВРОЛОГІЧНИХ ТА ПСИХІЧНИХ ЗАХВОРЮВАНЬ

Filipets O.O.

ACUTE KIDNEY INJURY AND ISCHEMIC STROKE

*Department of Nervous Diseases, Psychiatry and Medical Psychology
Bukovinian State Medical University*

Introduction. Patients with kidney dysfunction have an increased risk of renoparenchymal hypertension and cardiovascular complications (myocardial infarction, stroke, heart failure, cardiovascular death). In general, patients with kidney damage more often die not from end-stage renal failure, but from cardiovascular complications, in particular from stroke. The leading mechanisms for this include activation of the sympathetic and adrenal system, increased atherogenesis, endothelial dysfunction, increased inflammatory factors, changes in the level of lipoproteins, hypertrophy of the left ventricle, and the presence of hypertension.

The aim of the study - to analyze the scientific data highlighted in the available sources of computer databases of evidence-based medicine in order to systematize ideas about the relationship between renal dysfunction and the development and course of acute ischemic stroke.

Material and methods. A systematic approach of searching for the most qualitative information in systematic reviews over the last ten years was used. Information was searched in the Cochrane Library (Cochrane Database of Systematic Reviews, DARE, HTA), the US National Library of Medicine (Medline), computer databases PubMed, Embase.

Results. Acute kidney injury (AKI) is a sudden onset of renal dysfunction accompanied by structural and functional abnormalities that develop over several hours/days. AKI can result from decreased renal perfusion, nephrotoxicity, or damage to the glomeruli, tubules, interstitium, or renal vasculature. AKI usually occurs in critically ill patients and is defined by a decrease in the glomerular filtration rate (GFR), albuminuria, a sharp increase in the level of creatinine and cystatin in the blood plasma, and oliguria. Creatinine levels in the blood plasma do not accurately reflect acute changes in kidney function and can vary widely depending on age, sex, initial functional status of the kidneys, etc., which makes it difficult to determine the degree of renal damage. A meta-analysis of eight studies showed that AKI is a common complication of acute ischemic stroke

with an overall prevalence of 12.9% of patients developing AKI, leading to increased mortality. In a cohort of 40 stroke patients (mean age 69.1 years, 90% of patients had ischemic stroke), 62.5% of patients developed AKI. A follow-up 10-year study of a large cohort of patients with first-ever acute stroke found that approximately 27% of patients developed AKI and had a higher mortality rate than stroke patients without AKI. The incidence of AKI was not significantly different between stroke patients treated with and without tissue plasminogen activator (tPA) (35.5% of patients treated with tPA compared with 33.9% of patients not treated with tPA developed AKI), but in patients with AKI, the in-hospital mortality rate was significantly higher (50.0% mortality in patients with AKI vs. 3.4% in patients without AKI). In a 7-year follow-up study, creatinine clearance was calculated in patients with acute stroke and found elevated plasma creatinine and urea concentrations, as well as high urea/creatinine ratio, which was reflected by increased short- and long-term mortality rates. Symptoms of AKI, such as decreased GFR and elevated plasma uric acid, have been reported to occur within 72 hours of acute stroke. To a large extent, stroke patients with severe neurologic deficits, cardiac abnormalities such as heart failure, atrial fibrillation and coronary heart disease, hyperglycemia, hypertension, low GFR, or advanced age were more susceptible to developing AKI. Although the incidence of AKI after acute ischemic stroke varies widely depending on AKI assessment markers, an increased mortality risk has been reported among patients who develop AKI after stroke.

Conclusions. Taking into account the functional and metabolic interrelationship of the brain and kidneys, timely analysis of indicators of the functional state of the kidneys, detection and urgent therapy of renal dysfunction is of important practical importance for pharmacotherapy, respectively, the course of acute ischemic stroke and the results of treatment, in particular, rehabilitation and recovery after stroke and will also be reflected in reduced mortality rates.

Herasymiuk I.G.

PREREQUISITES OF RESPONSE TO ANTIDEPRESSANT TREATMENT IN PATIENTS WITH RECURRENT DEPRESSIVE DISORDER

*Department of Nervous Diseases, Psychiatry and Medical Psychology
Bukovinian State Medical University*

Introduction. Depressive disorders encounter in the population in 3.2% of patients without concomitant somatic diseases and from 9.3% to 23.0% in patients with chronic diseases. It is the fourth leading cause of disability worldwide and is likely to become the second leading cause of disability after cardiovascular disease over the next decade.

The aim of the study. The task of the study was to form the criteria of therapeutic response to antidepressant treatment, a detailed structured study of specific ways of the influence of clinical-psychopathological and pathopsychological criteria on clinical features and the treatment process of a depressive disorder.

Material and methods. All patients were examined somatically and neurologically, consulted by a therapist and other specialist doctors. When taking an anamnesis, much attention was paid to objective data: a detailed survey of the patient's relatives and close friends, an analysis of the available medical documentation made it possible to verify the diagnosis of recurrent depressive disorder more accurately. Identification and description of significant premorbid factors was carried out by comparing the complete list of factors in the literature with the anamnesis and status of each patient.

Results. No significant differences were found according to the gender of the patients, as depression prevailed in women in 83.3%. This result rather reflects the peculiarities of the formation of the research sample and is not representative of the entire population's depression. Heredity is not a mandatory risk factor for depression. Mental disorders and peculiarities were found in the family history of 53.3% of patients. The education of majority of patients most often belonged to the higher level (43.3%), another 23.3 participants had not completed higher education. The trend toward a higher frequency of incomplete higher education at a young age was due to the fact that several patients had interrupted their university studies during the current depressive