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CHRONIC RENAL DISEASE AS A CAUSE OF CARDIOVASCULAR PATHOLOGY

Kulik Y. E.[1], Martymianova L. O.[1], Rybchynskyi S. V.[1], Kartvelishviliy H. Yu.[2]
[1] V. N. Karazin Kharkiv National University, Kharkiv, Ukraine
[2] Kharkiv Railway Clinical Hospital № 1 of Brence of «HC» JSC «Ukrzaliznytsia», Kharkiv, Ukraine

The issues of etiology of development of cardiac changes in renal dysfunction, diagnostics and establishment of clinical diagnosis are reviewed as illustrated by a clinical case. Recommendations on lifestyle modification and medicament treatment tactics are described.

KEY WORDS: chronic pyelonephritis, coronary heart disease, arterial hypertension, cardiac failure

ХРОНІЧНА ХВОРОБА НИРОК ЯК ПРИЧИНА ВИНИКНЕННЯ СЕРЦЕВО-СУДИННОЇ ПАТОЛОГІЇ

Кулик Я. Е.[1], Мартим'янова Л. О.[1], Рибчинський С. В.[1], Картвелішвіли А. Ю.[2]
[1] Харківський національний університет імені В. Н. Каразіна, м. Харків, Україна
[2] ХКЛ ЗТ № 1 Філії «ЦОЗ» ПАТ «Укрзалізниця», м. Харків, Україна

На прикладі клінічного випадку розглянуто питання етіології розвитку кардіальних змін при нирковій дисфункції, діагностику і встановлення клінічного діагнозу. Описано рекомендації з модифікації способу життя, а також тактику медикаментозного лікування.

КЛЮЧОВІ СЛОВА: хронічний пієлонефрит, ішемічна хвороба серця, артеріальна гіпертензія, серцева недостатність

ХРОНИЧЕСКАЯ БОЛЕЗНЬ ПОЧЕК КАК ПРИЧИНА ВОЗНИКНОВЕНИЯ СЕРДЕЧНО-СОСУДИСТОЙ ПАТОЛОГИИ

Кулик Я. Э.[1], Мартимьянова Л. А.[1], Рыбчинский С. В.[1], Картвелишвили А. Ю.[2]
[1] Харьковский национальный университет имени В. Н. Каразина, г. Харьков, Украина
[2] ХКБ ЖДТ № 1 Филиала «ЦЗ» ПАО «Укрзализныця», г. Харьков, Украина

На примере клинического случая рассмотрены вопросы этиологии развития кардиальных изменений при почечной дисфункции, диагностику и постановку клинического диагноза. Описано рекомендации по модификации образа жизни, а также тактику медикаментозного лечения.

КЛЮЧЕВЫЕ СЛОВА: хронический пиелонефрит, ишемическая болезнь сердца, артериальная гипертензия, сердечная недостаточность

INTRODUCTION

Multiple studies have proven that renal dysfunction [1] is an independent predictor of cardiovascular morbidity.

Even a mild renal pathology, irrespective of its etiology, considerably increases the risk of arterial hypertension, coronary heart disease (CHD), cardiac failure, and cardiovascular death [2].

The risk of cardiovascular complications occurs not only at renal failure terminal stage, but also at early stages of renal function decrease [3]. This is due to complex effect of hemodynamic, metabolic, and endocrine disorders associated with renal dysfunction on myocardium and vessels [4]. The interrelation between renal function and cardiovascular system condition is obvious, which allows not only combining kidney and heart affection and chronic cardiac failure development into cardiorenal continuum, but also introducing the concept of «cardiorenal syndrome» into clinical practice [5]. Its essence is as follows: kidney or heart dysfunction with acute or chronic development pattern aggravates the failure of

each organ, thus increasing mortality due to cardiac or renal pathology [6].

The renal pathology in the examined clinical case is chronic pyelonephritis. This is a slowly progressing, occasionally exacerbating bacterial inflammation of renal interstitial, which leads to irreversible changes in pelvicalyceal system, as well as to elevated blood pressure and chronic renal failure [7]. Chronic pyelonephritis can be either a consequence of incurred acute pyelonephritis or a primary chronic process [8].

Renal parenchyma infection occurs via hematogenous pathway from remote foci (in furunculosis, carbunculosis, abscess etc.) or via ascending urinogenous pathway (in cystitis, urethritis, prostatitis).

OBJECTIVE

The aim of the research is demonstration of particulars of managing a patient with complex cardiac pathology by an example of the represented clinical case.

OUR CASE

Case history of a man 54 years old.

Complaints. Thoracalgias of pressing nature (lasting about 3–5 minutes) appear at walk up to 200 m. They have predominantly retrosternal location. They are arrested by nitroglycerine after 2–3 minutes. Dyspnea appears both after walk and at rest. Headaches develop mainly at blood pressure elevation. They are arrested by citramonum after 20 minutes. Weakness. Palpitation attacks lasting for a few minutes to an hour, which develop upon blood pressure elevation. Cough with expectoration of a little sputum. Polyuria up to 2 l a day. The patient presents no complaints from other organs and systems.

Disease history. In 1999, the patient underwent surgery due to the left kidney carbuncle. Later, the patient was diagnosed with chronic pyelonephritis. The patient reports episodes of blood pressure increase since 2005; the maximum blood pressure was 220/140 mm Hg. He did not receive any treatment. Since June 2007, the patient has noticed the appearance of palpitations episodes associated with blood pressure elevation, which are accompanied with weakness, shortness of breath, and cough with expectoration of a little sputum. In August 2007, the patient received treatment in cardiologic department of the Central Clinical Hospital, where he was diagnosed with: persistent form of auricular flutter. Acute left ventricular insufficiency. Pulmonary edema. Cardiac failure degree 2A. The patient's condition was improved as of the moment of discharge, and sinus rhythm was restored as a result of urgent defibrillation. In 2010, the patient was hospitalized to the Central Clinical Hospital with complaints about pain in cardiac area, which he could not arrest on his own, and palpitations. He underwent a treadmill test with a positive result. Coronaro-ventriculography (CVG) was performed, and multivessels disease of coronary bed has been identified. Coronary artery bypass graft (CABG) surgery (3 bypasses) was performed. In July 2015, a new palpitation attack took place. The patient was urgently hospitalized to the Department of Intensive Care. The patient was diagnosed with cardiac rhythm disorders – persistent atrial fibrillation (AF) has been identified, for which catheter ablation was performed. The patient's condition has improved subsequent to the treatment performed. It was recommended to continue intake of anti-arrhythmic drugs (metoprolol 100 mg in the morning, losartan + hydro-chlorothiazide 50 mg twice daily, nifedipine 40 mg in the evening).

Life history. The patient's living conditions are satisfactory. He denies any pernicious habits. The patient's medication history and history of allergies are not aggravated. The patient denies a history of tuberculosis, viral hepatitis A, diabetes mellitus, psychic and venereal diseases. His heredity is aggravated in terms of cardiovascular diseases – coronary heart disease and arterial hypertension.

Objective status. The patient's condition is of moderate severity, his consciousness is clear, and his position is active. The patient's constitutional type is normosthenic. His height is 185 cm, his body weight is 78 kg, and the body mass index is 22.9. His cutaneous coverings are typical, pale pink. The lymph nodes available for palpation are not enlarged. The thyroid gland is not clearly identifiable. Palpation is painless. The locomotor system is unremarkable. Pastosity of lower extremities is at the ankle level.

Respiratory system. The chest is normosthenic. Condition after sternotomy. Percussion: dullness of lung sound in posterior lower lung portions along the scapular line at the level of rib IX on the left and along the paraspinal line at the level of rib X. Auscultation: rales in lower lung portions

associated with decreased vesicular respiration. Respiratory frequency – 23 per minute.

Cardiovascular system. The apex beat is located in intercostal space V along the left midclavicular line, diffuse (up to 3 cm). At topographic percussion, the left border of relative heart dullness is located in intercostal space v along the midclavicular line, and the right one and the upper one are unaltered. The cardiac activity is rhythmic. The heart sounds are muffled. Heart rate = pulse – 110/min. Blood pressure 180/120 mm Hg.

The abdomen has typical dimensions, it is soft and painless. The liver is located at the costal arch margin, it is painless. Costovertebral angle tenderness is negative on the both sides.

RESULTS OF THE SURVEY

Clinical blood count (20.02.17*)*: Hb – 162 g/l; erythrocytes – $5.12*10^{12}$/l; leukocytes – $12.2*10^{9}$/l; ESR – 7 mm/h; eosinophils – 2 %; neutrophils: stab – 11 %, segmented – 78 %; lymphocytes – 7 %; monocytes – 4 %; platelets – 344 g/l; hematocrit – 48 %.

Urinalysis (20.02.17): Relative density – 1.007; protein – not identified; glucose – not identified; leukocytes 5–7 in the field of vision; pH – 6.0.

Blood chemistry panel (20.02.17): Total bilirubin – 16.5 μmol/l; AST – 22 U/l; ALT – 13 U/l; creatinine – 111.98 μmol/l; urea – 7.7 mmol/l; glucose – 7.5 mmol/l. Glomerular filtration rate measured by Cockcroft-Gault method – 75.5 ml/min.

Chest X-ray examination results (23.02.17): No focal or infiltrative changes are identified in lungs. Pleuropericardial cords are seen on the left. Venous hyperplasia signs are identified. The roots are structured and not enlarged. The sinuses are patent. The diaphragm is clearly delineated. The heart is expanded on the left. The aorta is unremarkable. Condition after sternotomia.

ECG results (22.02.17): The rhythm is sinus, regular. The heart rate is 73 bpm. Complete left bundle-branch block.

EchoKG results (23.02.17): Sclerotic changes of aorta walls, aortic and mitral valve cusps. Left ventricular myocardial hypertrophy. Dilation of cavities of both atriums. Regurgitation on pulmonary artery valve, degree I-II. Ejection fraction – 65 %.

Daily ECG and BP monitoring results (26.02.17): ECG: predominant rhythm is sinus, with average heart rate 73 bpm at daytime and 71 bpm at night. The circadian index is 1.02 %. Rigid circadian heart rate profile, vegetative denervation signs. Individual ventricular extrasystoles (1072) were registered during observation period. No ischemic changes are identified via ECG. Blood pressure: average daily BP is 133/83 mm Hg (min 105/56 mm Hg, max 160/90 mm Hg). Circadian index of systolic BP is 14 %. Circadian index of diastolic BP is 17 %. The patient belongs to the group with normal nocturnal blood pressure decrease.

Ultrasonic examination of kidneys (24.02.17): Contracted left kidney. Cyst of the left kidney. Microcalculosis of kidneys.

CLINICAL DIAGNOSIS

Coronary heart disease: stable effort angina of functional class III. Atherosclerosis of coronary arteries (coronaroventriculography dated 09.12.10). Coronary artery bypass graft – 3 bypasses (26.12.10). Renal arterial hypertension of III stage, 3 degree. Hypertensive heart. Persistent form of atrial fibrillation. Catheter ablation (2015). Cardiac failure stage II A, functional class II. Chronic renal disease of stage II. Carbuncle of the left kidney (1999). Urolithiasis. Secondary pyelonephritis, remission stage. Secondary contracted kidney. Cyst of the left kidney.

TREATMENT PLAN

1) Lifestyle modification:

– Change of daily routine (sleep duration not less than 8 hours a day).

– Dieting and following of recommendations on tolerable physical activity for this angina pectoris functional class. The main training technique in this case is slow walking without acceleration, at the rate below the pain threshold; after improvement of the patient's condition, achievement of walking rate 3–3.5 km/h can be deemed quite satisfactory. Duration of such exercise may comprise 20 to 60 min depending on the patient's condition severity. At home setting, the patient is recommended to perform respiratory and mild physical exercise 1–2 times daily.

2) Medicament therapy: Aspirin 75 mg; Valsartan 80 mg in the morning and 80 mg in the evening under blood pressure monitoring on a long-term basis; Hydrochlorothiazide 12,5 mg in the morning under blood pressure monitoring on a long-term basis; Atorvastatin 20 mg in the

evening; Amlodipine 5 mg twice daily under blood under blood pressure monitoring.

CONCLUSIONS

This clinical case reflects the peculiarities of incessant progression of combined cardio-vascular pathology developed in association with chronic renal disease, as well as diagnostics and treatment methods.

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