ELECTROLYTE DISTURBANCES IN PATIENTS WITH CHRONIC HEART FAILURE – CLINICAL, EVOLUTIVE AND THERAPEUTIC IMPLICATIONS

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ELECTROLYTE DISTURBANCES IN PATIENTS WITH CHRONIC HEART FAILURE – CLINICAL, EVOLUTIVE AND THERAPEUTIC IMPLICATIONS (Abstract): Patients with heart failure are, by definition, hemodynamic unstable. Often, this condition may be accentuated by medication (digitalis, diuretics, antiarrhythmics), so that they become more sensitive to electrolyte disturbances, reacting sometimes violently with severe and life threatening rhythm troubles. Aim: the evaluation of the incidence of electrolyte disturbances in patients diagnosed with chronic heart failure in order to establish a correlation with the evolution, prognosis and therapeutic implications. Material and methods: We analyzed retrospectively 100 patients diagnosed with chronic heart failure NYHA II-IV classes, admitted in Cardiology Clinic during 2009 – 2011. We analyzed electrolytic disturbances occurred during different strategies of therapy. Results: 100 patients with heart failure were admitted in Cardiology Clinic during 2009-2011, 75 males and 25 females. Diagnosis was established by classical criteria. Evaluation was very complex and included: complete clinical examination, electrocardiogram, echocardiography, chest ray examination and biochemical analyses especially hepatic, renal function and electrolyte status. Conclusions: The obtained data showed that electrolyte disturbances are frequent in patients with chronic heart failure, irrespective of NYHA class. Hyperkalaemia, hypokalaemia and hyponatraemia are associated with diuretic therapy and may play a very important role in subsequent development of life-threatening arrhythmias. Key words: ELECTROLYTE DISTURBANCES, HYPOKALAEMIA, HYPERKALAEMIA, HYponatraemia.

The patient with heart failure is by definition a hemodynamically unstable patient in which any therapeutic gesture initially beneficial, may have unexpected side effects, some of them life-threatening (1). This type of patient usually has electrical instability related to the underlying disease and potentiated under medication (diuretics, digitalis, antiarrhythmics etc.) (1, 2, 3) In these conditions, they become more sensitive to even minor electrolyte balance disturbances, sometimes reacting violently through serious arrhythmias or conduction disturbances. The medication of the patient with heart failure involves complex therapeutic schedule, adapted to the etiopatho-
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genic and hemodynamic features of each case. In these schedules the diuretic medication is essential and it is pathophysiologically addressed to the salt and water retention (3). Although well tolerated by most patients, diuretic medication acting at renal level disrupts the electrolyte balance so that, depending on the type of mechanism, on the patients’ condition at the time, on the associated medication and diet, there may appear other electrolyte and fluid disorders some of which could be life-threatening (1, 2, 3). Hyponatraemia is the most common electrolyte disorder encountered in intensive care services (its frequency is estimated at 30-40% of the hospitalized patients) (4, 5).

The aim of the study is to evaluate the incidence of electrolyte disorders encountered in patients with heart failure development and to establish a correlation between these disorders and the apparition of certain complications, and the entailed prognosis and therapeutic implications.

MATERIAL AND METHODS

The study is retrospective and included a group of 100 patients, 75 men and 25 women, aged 48-85 years, hospitalized in the 1st Cardiology Clinic between the years 2009-2011 and diagnosed with chronic heart failure of the NYHA II-IV classes.

The classification of patients in the NYHA class of severity of heart failure was as follows (fig. 1):

- NYHA Class II - 15 patients: 10 men, 5 women;
- NYHA class III - 72 patients: 55 men, 17 women
- NYHA Class IV - 13 patients: 8 men, 5 women.

Fig. 1. Distribution of patients according to the NYHA class of heart failure severity.

The diagnosis of heart failure was based on the known and classical clinical and paraclinical data:
- Symptoms and signs of left ventricular failure (shortness of breath of different degrees, cardiac cough, tachycardia with / without gallop rhythm, presence of organic heart breaths and pulmonary rales stasis);
- Signs of right myocardial failure: hepatomegaly, edema in the legs (cardiac type), turgid jugular (level I-III), hepatojugular reflux, with or without ascites.

Paraclinical exploration of the patients included:
- Chest radiography - objectified cardiomegaly (ICT over 0, 55) with or without
signs of pulmonary stasis or pleural effusions.

- Echocardiography (2D, M mode, Doppler) objectified cardiac cavities dilation with decreased contractile function parameters (ejection fraction, shortening fraction), and other aspects: valvular regurgitation, intracardiac thrombus, presence of spontaneous contrast and signs of hypertension pulmonary artery. Echocardiography was essential for identifying the etiology of heart failure.

- Peripheral venous pressure was elevated in most patients with right heart failure component.

- 12 leads surface ECG. There were sought: rhythm or management disturbances, ischemo-lesional changes, overstress of certain cavities, changes due to electrolyte disturbances.

- Biochemical balance included: hepatic function (transaminases, bilirubin, GGT), renal function (urea, creatinine, uric acid, creatinine clearance, RA), serum electrolytes (Na +, K +, Ca ++, Mg ++);

- Paraclinical investigation was complemented with complete blood count, spirogram and determination of blood gases and pH.

Patient evaluation was performed first at the time of admission and subsequently, depending on each particular case, was performed daily during hospitalization.

The etiology of heart failure was as follows (fig. 2):

- Ischemic dilated cardiomyopathy - in 20% of cases;
- Ethanoletic dilated cardiomyopathy - in 45% of cases;
- Mixed dilated cardiomyopathy (ischemic, toxic, diabetic) - 20% of cases.
- Valvular heart disease - 15% of cases.

Etiology was based on history, physical examination and echocardiographic data resulted from examination. In some cases, to these was added the presence of the biochemical markers that supported chronic alcoholism.

Patients’ treatment was strictly individualized according to the haemodynamic status at the time of hospitalization, the etiological features, the presence or absence of arrhythmias and/or conduction disturbances and included, besides the
appropriate hygienic-dietary measures, the following (fig. 3):
- Diuretic: loop - 15 patients (15%), spironolactone + loop diuretic combination - 85% of patients;
- Nitrate type vasodilator - 75% of patients;
- Converting enzyme inhibitor (ACEI) - adjusted doses of TA was administered to 85% of patients (65% received perindopril, 20% received ramipril);
- Beta blocker - 90% of patients (55% carvedilol, bisoprolol 35%);
- Digoxin - 70% of patients;
- Amiodarone - 30% of patients;
- Anticoagulant - 60% of patients;
- Antiplatelet - 75% of patients received aspirin (in 50% of the cases it was associated with anticoagulant), 25% of patients receiving clopidogrel.

Patients received therapeutic regimens that included combinations tailored to each individual case, as follows:
- Loop diuretic + vasodilator (nitrate) + beta blocker + ACE inhibitor - 10% of cases;
- Loop diuretic + spironolactone + ACE inhibitor + beta blocker + vasodilator (nitrate) - 40% of cases;
- Loop diuretic + spironolactone + ACE inhibitor + beta blocker + digoxin + vasodilator - 20% of cases;
- Loop diuretic + ACE inhibitor + amiodarone + vasodilator - 30% of cases.

All patients received an antiplatelet regimen and in 60% of the cases an anticoagulant administration was necessary.
Introducing the anticoagulant regimen was imposed by the presence of echocardiographic intracavitary thrombus or spontaneous contrast, by the presence of dilated right cavities and other clinical signs suggestive of thromboembolism associated phenomena. Throughout the immobilization in bed there were also injected prophylactic doses of low molecular weight heparin.

The nitrate type vasodilator was used with preference in patients with signs of left ventricular failure considered to be due to ischemia and in patients with evident pulmonary stasis.

**RESULTS AND DISCUSSION**

The electrolyte disturbances encountered in patients of the group were the following:

*Hyperkalaemia* (serum K values between 5.8 to 6.5 mmol/l) - present in 65% of patients receiving loop diuretic combination with spironolactone associated with ACE inhibitors. 51% of patients with hyperkalaemia showed electrocardiographic changes suggestive of this type of electrolyte disorder: high T beams, in precordial (5%), ventricular extrasystoles systematized in bi- form and trigeminy ventricular doublets (45% of cases). 35% of these cases were associated with elevated urea and serum creatinine with functional significance of renal failure. The therapeutic conduct consisted of stopping potassium saving diuretic, replacing it only with loop diuretic, infusion of glucose 5% buffered with serum insulin to normalize serum potassium. In patients receiving concomitant digoxin, presence of ventricular extrasystoles imposed reconsidering the therapeutic regimen and removing digoxin where present. Lidocaine or beta blocker was used to control arrhythmias.

*Hyponatraemia* (serum sodium values between 128-110 mmol/l) was present in all patients with NYHA IV class heart failure receiving diuretic therapy. Clinically, these patients presented generalized edema at admission. In 5% of the patients hyponatraemia persisted after stopping diuretic, so it was considered dilution hyponatremia and significantly correlated with increased mortality in this population. Correction of hyponatraemia was difficult, with partial response and limited to fluid restriction and administration of sodium chloride, while continuing the diuretic.

In 15% of cases, these electrolyte disturbances have been associated with inflection of the renal function, that is, increased urea and creatinine values understood as renal functional failure and whose main cause was the diuretic abuse with subsequent reduction in pressure renal perfusion. In 10% of the cases, increased levels of uric acid could be seen (between 9-11 mg%), correlated with the loop diuretic doses. In 7% of cases, renal failure was corrected by
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temporary withdrawal of the diuretic and proper hydration. In 8% of cases, it was necessary the administration of vasodilator dose dopamine (2-3 mcg/kg/min).

CONCLUSIONS

Electrolyte disturbances are common in patients with heart failure development, regardless of NYHA functional class. In most cases this is due to medication, especially to the diuretic therapy. From this perspective, certain drug combinations as loop diuretics with digoxin, potassium saver diuretics with ACE inhibitors present a more important risk. Correcting these imbalances is a major emergency as they often are life-threatening, representing the substrate for different arrhythmias including the ventricular which can represent a vital threat.

Hyponatraemia is an independent negative prognostic factor especially in patients enrolled in the NYHA IV functional class and it is often associated with increased mortality.

Knowing these electrolyte disturbances that can occur at any time in the evolution of patients with heart failure imposes attention to the choice of therapeutic regimen, caution in dosing and combination of drugs, which are, in fact, absolutely necessary.

REFERENCES