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HOMECARE GENTAMICINE TREATMENT FOLLOWED BY SEVERE RENAL INJURY ASOCIATED WITH HIPOALBUMINEMY AND DEHIDRATATION (CLINICAL CASE)

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ABSTRACT. Presenting the case of 83 year old patient A.R., brought to ER for severe dyspnea due to a total left pneumothorax. After mounting the pleural drainage tube, the patient is admiitted into ICU, presenting significantly altered renal probes and showing specific clinical signs of acute dehydration syndrome and systemic uremic syndrome. The patient is given crystalloid and colloid solutions to correct the water deficite and improve renal perfusion, but the clinical condition does not improve, diuresis is absent at the 4 mark, thus it's decided to establish continuous renal support consisting in continuous veno-venous hemodiafiltration.At the same time the patient is receiving additional therapy with hypertonic solution of 20% albumin to correct severe hypoalbuminemia. Patient evolution is ultimatly favorable under diuretic treatment administered continuously, evaluation of hidric balance on a daily basis and with fluid intake adjustment on the basis of that balance.
KEYWORDS: tubular necrosis, sepsis, hypoalbuminemia, renal injury severe dehydration

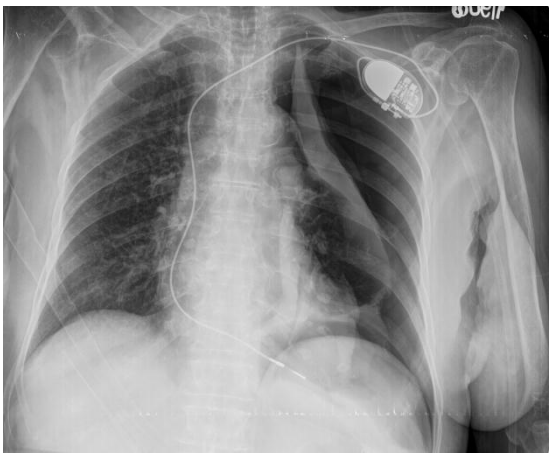
INTRODUCTION

Acute tubular necrosis is the leading cause of severe renal injury seen in clinical practice, it being the renal pathology that often require continuous renal support. The most common cause of this tubular necrosis is by far sepsis, other possible causes include crush syndrome, abdominal compartment syndrome, administration of nephrotoxic substances, severe renal ischemia in conditions of shock regardless of cause, there are many predisposing conditions such as dehydration or severe hypoalbuminemia.

MATERIAL

The case of a patient which after pleural drainage and respiratory improvement is admitted in ICU for severe renal injury. During admission the patient shows constant drowsiness, dry skin and mucous membranes, numerous petechiae on skin, MV is slightly diminished on the left side with bronchial rales superimposed AV 72b/min, BP 125/70 mmHg, slim abdomen without tenderness palpation is afebrile, with diuresis absence.

Laboratory investigations – leukocytes -11 700 thousand/mm 3, HT-41% Hg 12 g/dl, platelet count - 122,000, IQ 64%, 1.25 INR, APTT 34 seconds, TGO-140, ALT-120, -212 mg urea/dL Creatinine 8.2 mg/dl, K-3.6 meq/dl, Na 147 mEq/dl Lactic acid 2.7 mmol/dl, serum albumin 2.4 mg/dl, total protein 4.8 mg/dl, procalcitonin 1.2 ng/ml. Pulmonary x-ray show left pneumothorax with secondary total lung collapse and scleroemfizem in the right lung.



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From history we learn that the patient was hospitalized in cardiology the week before, when she was fitted with a pacemaker for total atrioventricular block, laboratory tests on her discharge papers showing good kidney function and leukocytes being in normal limits. Also we noted that at home the patient presents productive coughing for which she is taking Cefort 1g /12h, Gentamicin 80mg/12h (for four days), fluid intake is below the necessary physiological approximately 1500ml / day, and that the patient is under angiotensin receptor inhibitor chronic treatment (Candersartan 8mg / day) for hypertension

ICU admission diagnosis is:

- left drained iatrogenic pneumotorax
- moderate respiratory failure
- acute bronchitis
- severe renal injury
- acute dehydration syndrome
- uremic encephalopathy
- stage 2arterial hypertension
- severe hypoalbuminaemia
- lactic acidosis

MATERIAL AND METHOD

Consists in following the evolution of the patient, both clinically and paraclinicaly after the start of electrolyte rebalancing treatment and fluids repletion, and then after aproximatively 4 hours, with the initiation of renal support consisting of continuous veno-venous haemodiafiltration (HDFVVC). This type of continuous renal support used in ICU consists in combining the two methods of kidney treatment - filtration and dialysis, filter ST-150, the total dose of flow in both circuits, is 30 ml /kg/h, initially equally divided and for anticoagulation we used APTT systemic control heparin, the target value being 2x the base.

Meanwhile the patient received 20% hypertonic albumin solution to correct hypoalbuminemia, antibiotic therapy consisting in cefoperazone / Sulbactam 2g/12h, and Ciprofloxacin 200mg/12h, collection of tracheobronchial secretions, Acetylcysteine 300mg/12h, gastric protection performed with Ranitidine 50mg/12hr, 10mg/8h metoclopramide was added to prevent nausea.

During the treatment session the patient is given extrarenal 100Oml crystalloid solutions of Nacl 0.9%, nutritional support is provided by the solution of 10% glucose 1000ml corrected with insulin and Nefrotect Fl 1/12h amino acid solution, which corresponds to 50 g AA, KCL added 7.4% each 15ml/fl glucose to maintain potassium levels in the normal range. It is important to note that the removal of liquids during the session which lasted 30 h was 0 ml / hr initially and then 10 ml /h in the last 6 h, and the patient's spontaneous diuresis was only 500ml / day. It is important to mention that during and after the session the patient presents phenomena of hyperactive delirium for which neuroleptic (haloperidol), is administered the loading dose of 15 mg iv slowly administered over 1 hour followed by maintenance dose of 5 mg/6h.

At the end of the HDFVVC session, kidney samples indicate urea 60 mg/dl, creatinine 3 mg/dl, ionogram serum- normal levels, procedure is stopped, a matter of vascular access, due to significant bleeding from the puncture site, a needed 3 pg PPC transfusion is administration (dose 10 ml/kg) to correct the clotting factor deficiency and MER 2 pg for anemia correction.

Subsequently, the intensive treatment consists in continuously maintaining the intake of 1500ml/day, crystalloid solutions and parenteral nutrition (10% glucose and nefrotec, the patient showing at this moment good digestive tolerance for liquids, thus along side the liquid intake continuous administration of 20% albumin solution is provided for three days until its value becomes 3.4g /dl. After the HDVVC session, considering that diuresis is only 500ml / day and the hidric balance is positive, the furosemide diuretic is inserted into the treatment - continuously syringe administered at a dose of 10mg /h.

Evolution of the patients is still slow but favorable, diuresis was taken up gradually, thus 2 days after starting treatment with Furosemide, the dose is initially decresed to 5mg /h, and then stopped at a diuresis level of 3.300ml / day. The patient enters the polyuria phase of renal injury, with spontaneous diuresis approx. 10.000ml for 2 days, requiring supplementation of fluid intake though balanced crystalloid solution, 10% glucose solutions, amino acid solutions, as well as verification of the ionogramelor series. 10 days since admission to ICU, the patient is transferred to the department of nephrology, clinical status at discharge is good, and laboratory analysis indicates improving kidney function, urea 56 mg / dl, creatinine 2.6, diuresis spontaneous 6000ml / day .

RESULTS

Regarding the intensive treatment of renal support which consisted as mentioned, first in restoring circulating volume and maintain it with crystalloid

electrolyte rebalancing and colloids solutions, as well as use of albumin solution 20% for Hypoalbuminemia correction and later undertaking the continuous extrarenal treatment procedure (based on criteria established internationally - RIFLE classification), and finally the introduction of the diuretic in treatment, due to increasing nitrogen retention and fluid overload, we present the results in table 1.

DAY	1	2	3	4	5	6	7	8	9	10
DIURESIS/ml	100	500	1900	2200	3300	3600	6300	10000	10400	6200
ALBUMIN	2,4	2,9	3,2	3,4	3,4	3,4	3,4	3,6	3,7	3,6
CREATININE	8,3	3,	4,3	5,4	6,1	6,2	6,2	4,7	3,4	2,6
UREA	212	70	100	108	120	121	120	101	72	56

Table no.1 Daily parameters evolution presented in graphs nr.1,2,3,4, .based on intensive administered treatment.

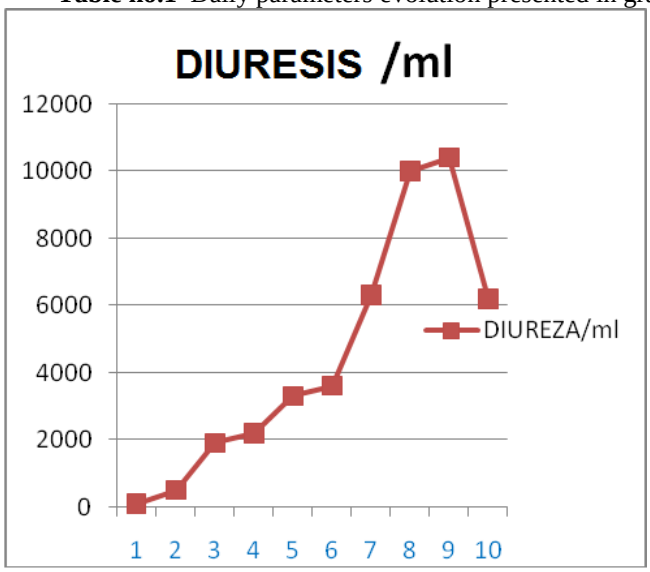

Fig. 1

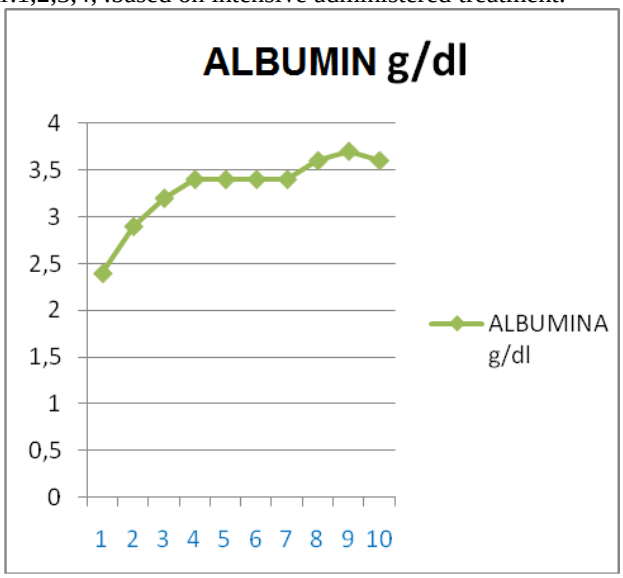

Fig.2

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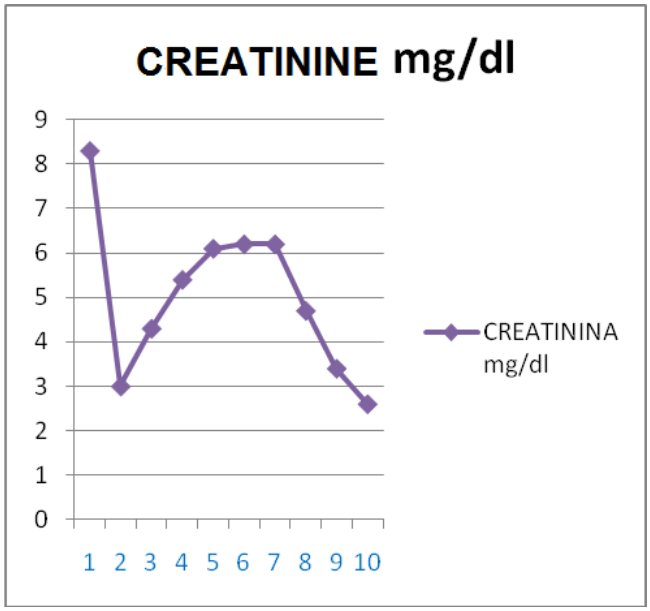


Fig. 3

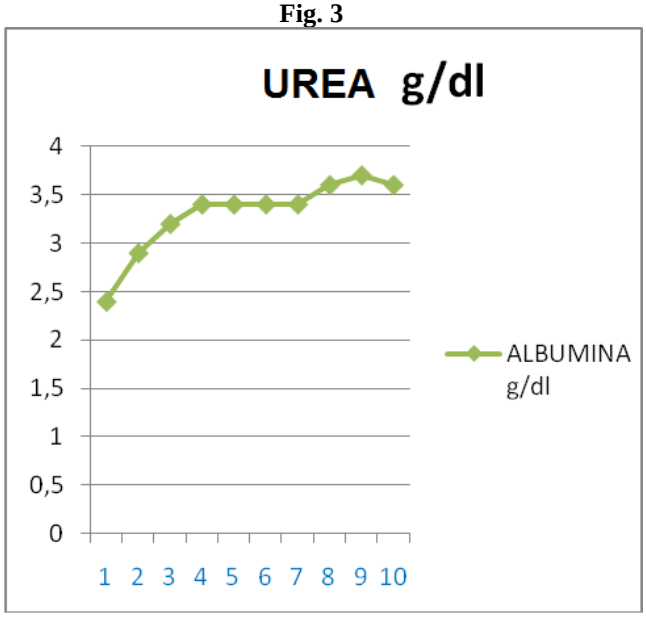


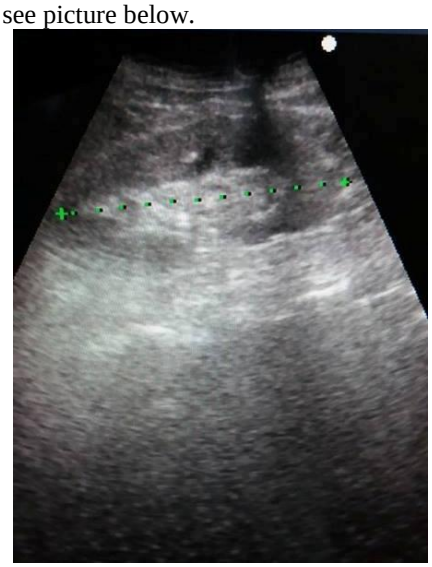
Fig.4

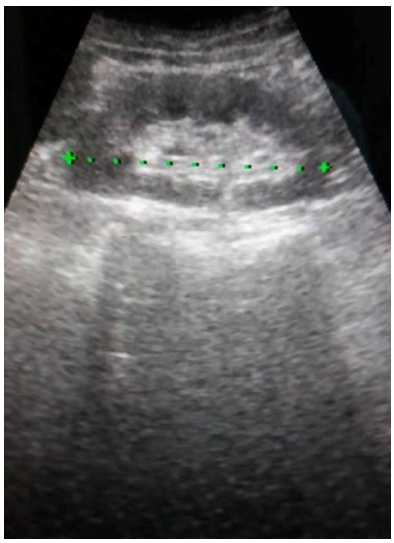
Fig.1,2,3,4 - DAY 1-3 Treatment with albumin 20% 50 ml fl
Fig.1,2,3,4 - DAY1-2 CRRT Treatment
Fig.1,2,3,4 - DAY 3-6 Diuretic furosemide 10mg/h initially, then 5mg/h
Fig.1,2,3,4 - DAY 1-10 Volemic treatment to restore and maintain renal perfusion

CONCLUSIONS

The peculiarity of this case is based on the factors involved in the severe renal injury, with severe hypoalbuminemia from admission we can turn to glomerular pathology (eg. Glomerulonephritis rapidly progressive) for confirmation Esbach test was performed - quantitative analysis of protenuriei 24 hours which was only 680mg/day and therefore excludes this type of pathology.

To eliminate a possible chronic renal failure (patient being elderly and hypertensive), besides the test results which were within normal limits from the previous admission, an ultrasound was performed which highlights the normal size of the kidneys and thus excludes a possible obstruction of renal ducts (postrenal cause) - see picture below.





The possible cause that led to this severe kidney injury is acute tubular necrosis, in many cases a diagnosis chosen by excluding the other possible diagnoses. Acute tubular necrosis is produced, in this case, from administration of nephrotoxic substances - Gentamicin administered in therapeutic doses of 80mg/12 hour for 4 days, plus the predisposing factors of dehydration due to lack of fluid intake, the administration of angiotensin receptor inhibitor at home, the patient's age and severe

hypoalbuminemia whose cause is not clarified due to the lack of any known liver or kidney existing pathology, severe diarrheal disease or protein-calorie malnutrition.

To confirm the diagnosis of acute tubular necrosis urinary tract ionogram must be determined - calculating FENA = NauxCrpl / NaplxCru × 100, whose value must be above 2%, possibly calculating Fe urea, as well as checking the urine osmolarity, the value of which must be less than 500mosm/l, these laboratory tests indicate a functional tube deficit (being used in practice as a method of differentiating between the functional and intrinsic ARF), to which the renal biopsy can be added to, followed by microscopic examination.

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