Nephrologist and Intensive Care Unit

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Abstract

Background. Collaboration between the nephrologist and the intensivist has been the most urgent and necessary in the diagnosis, optimal therapy and in establishing of 21 patients with acute renal failure of traumatic origin, 50 patients after acute intoxication by organophosphate inhibitors of cholinesterases, and 20 patients after acute ethylene glycol poisoning received treatment at the department of anaesthesiology and intensive medicine, intensive care unit of the IVth Medical Clinic, or the Nephrological Clinic of the Faculty Hospital of L. Pasteur in Košice, Slovak Republic.

Methods. In addition to conservative therapy all basic forms of extracorporeal elimination therapy (acetate, and later exclusively bicarbonate haemodialysis, haemoperfusion using charcoal and other sorbents) were used.

Results. Out of 91 patients, 14/21 died from the first group, 4/50 died in the second group, and 3/20 patients died after ethylene glycol intoxication.

Conclusions. The nephrologist was the chief consultant in establishing the diagnosis and treatment of acute renal failure and managing acute intoxication to avoid delay in the use of an early, adequate conservative and extracorporeal elimination therapy.

Key words: nephrologist, intensivist, acute renal failure of trauma etiology, acute intoxication of organophosphate inhibitors of cholinesterases, acute ethylene glycol intoxication

Introduction

Number of hospitalized patients significantly increased in the last 10-15 years. Progress of internal medicine and surgery allowed treating many older patients with various serious acute diseases. Number of departments of anaestesiology and intensive medicine (DAIM) and intensive care unit (ICU) for acute surgical and other patients increased in about 125%. It was a consequence of increased number of serious acute patients (1). This fact led to the specialisation of medical doctors, who are working in ICU of various clinical departments so-called intensivists and to the increased number of consultants in those units. Nephrologists, who are working in those departments spent in ICU until 29% of their working time, in comparison to general internists who are working only 21% of their working time in the same departments (1). According to Smithe et al (2) ICU's patients suffered from three most frequent syndromes: 1. respiratory distress syndrome, angina pectoris, heart attack, 2. sepsis, 3. hypotensia. These three diagnoses occurred in the patients, who were dominated and treated also by nephrologists (patients with chronic renal failure and after renal transplantation). In comparison to general internists, the nephrologists inclined more to a cardiovascular resuscitation in patients with renal diseases and in haemodialysis patients (3). Besides cardiovascular and lung diseases in ICU the patients were treated most often with disturbances of water and electrolyte metabolism, acid-base balance and with various disorders of renal functions. According to De Vita et al (4) 29.6% of the patients in ICU suffered from diluted hyponatriemia, caused by the intravenous administration of hypoosmolal solutions. Acute renal failure (ARF) of internal and surgical origin occurred in ICU from 14.9 - 23.0% (1). The kidneys were the third organ, which were the most frequently damaged (16%), after cardiovascular (33%) and lung damage (33%) (2). Hypotension was the most frequent cause of ARF and occurred in 85.5% (1). Ninety per cents of patients who were hospitalized in ICU suffered from multiorgan failure and 72% of those patients suffered from sepsis (1,5,6,7).

It was shown that nephrologists were the most experienced and the most suitable specialists for the treatment of those disorders, because above mentioned disorders were an integrated part of the daily work and their erudition. Intensivist who is working in DAIM and an ICU is a specialist in the treatment of acute patient with severe cardiovascular and lung diseases and in their monitoring. Only some intensivists have the experiences with the acute treatment in nephrology. In the years 1995 - 1997, 6.875 internists, 1.600 surgeons, 4.620 pulmonologists, 486 cardiologists, and as less as only 190 nephrologists were educated in DAIM and ICU at the appropriate institutions in USA. On the other hand only 2.7% of intensivists were educated in the clinical nephrology. As a consequence of this fact the intensivits in USA who are responsible for the coordination of various aspects of the intensive care in acute patients and who are working in DAIM and ICU, did not have sufficient amount of experiences to establish the diagnosis and to treat various complications of renal dysfunctions. If cases of severely ill patients, renal complication is presented for a consultation by experienced nephrologist is necessary (8). According to Bellomo et al (9) the patient with ARF would be treated exclusively by continuous extracorporeal elimination methods in ICU. The obtained results showed a decrease of oligoanuric period and a shorter hospitalization period and a significantly superior survival of patients with potentialy reversible ARF in comparison to the patients, who were treated in other

Miroslav Mydlik, Nephrological Clinic of the Medical Faculty of P.J. Šafárik University and Faculty Hospital of L.Pasteur, Logman a.s., Rastislavova 43, 04190 Kosice, Slovak Republic; E-mail: k.derzsiova@fnlp.sk departments. These authors supposed that nephrologists and intensivists should much more colaborate and a new specialisation "critical care nephrology" should originate in the near future.

Aim of the study was the restrospective analysis the three groups of seriously ill acute patients who were treated by nephrologists and intensivists in DAIM and ICU of the IVth

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Internal Clinic and in Nephrological Clinic of the Faculty Hospital, L. Pasteur, Košice, Slovak Republic.

Patients and methods

The first group of patients consisted of 21 patients with ARF of traumatic origin, among whom 18 were men and 3 women.

| Table 1. A | Table 1. Acute renal failure of traumatic origin | | | | | | | | |
|--------------|--|---------------------|----------------------|---------------|---------------------|---|-----------------|-----------|--|
| Number of | Mean age | Serum K (mmol/L) | Serum Na (mmol/L) | Serum urea | Serum creatinine | Number of days before the 1 st HD | Number of HD | Mortality | |
| patients | (year) | | | (mmol/L) | (µmol/L) | after polytrauma | | | |
| 21 | 32 | 7.1 | 130 | 49.5 | 892.6 | 5 | 74 | 14 | |
| 18 m | ± | ± | ± | ± | ± | ± | | (66.7%) | |
| 3 w | 6 | 0.6 | 4.5 | 13.5 | 185.7 | 2 | | | |

HD-haemodialysis

Table 2. Basic data and anamnesis in 50 patients after acute poisoning by organophosphate inhibitors of cholinesterases

| Organophosphate | Number | Mean | S | ex | Poising | | Poising Way of | | ning |
|----------------------------------|----------------|---------------|----|----|---------|------------|----------------|----|-------|
| inhibitors of cholinesterases | of patients | age (year) | М | W | S | accidental | Ι | 0 | other |
| 1. Soldep | | | | | | | | | |
| (trichlorfon) | 27 | 34 | 19 | 8 | 20 | 7 | - | 27 | - |
| Arpalit | | | | | | | | | |
| (trichlorfon) | 1 | 28 | - | 1 | - | 1 | - | - | 1 |
| 3. Phosdrin 24 EC | | | | | | | | | |
| (mevinphos) | 6 | 47 | 4 | 2 | 3 | 3 | 2 | 3 | 1 |
| 4. Metathion E 50 | | | | | | | | | |
| (fenitrothion) | 14 | 44 | 12 | 2 | 9 | 5 | - | 14 | - |
| 5. Decemption EK | | | | | | | | | |
| 20 (phosmet) | 1 | 39 | - | 1 | 1 | - | - | 1 | - |
| 6. Unknown | 1 | 39 | 1 | - | - | 1 | - | 1 | - |
| Total: | 50 | 38 | 36 | 14 | 33 | 17 | 2 | 46 | 2 |

S-suicidal attempt, I-inhalation, O-oral, M-man, W-woman

 Table 3. Clinical and laboratory signs in 50 patients after acute poisoning by organophosphate inhibitors of cholinesterases

| Organophosphate | | Clinical signs | | | Serun activity |
|-------------------------------|------------------|-----------------|-------------------|------|-------------------------------|
| inhibitors of cholinesterases | Muscarine effect | Nicotine effect | Central effect | Coma | of cholinesterase (µkat/L) |
| 1. Soldep | | | | | |
| (trichlorfon) | 27 | 8 | 6 | 3 | 3.8 |
| 2. Arpalit | | | | | |
| (trichlorfon) | 1 | - | - | - | 0 |
| 3. Phosdrin 24 EC | | | | | |
| (mevinphos) | 6 | - | - | - | 17.0 |
| 4. Metathion E 50 | | | | | |
| (fenitrothion) | 14 | 2 | 1 | 1 | 15.7 |
| 5. Decemption EK | | | | | |
| 20 (phosmet) | 1 | - | 1 | 1 | 18.5 |
| 6. Unknown | 1 | 1 | 1 | 1 | 6.2 |
| Total: | 50 | 11 | 9 | 6 | 8.1 ± 2.4 |

Table 4. Treatment in 50 patients after acute poisoning by organophosphate inhibitors of cholinesterases

| Organophosphate | Mean number | Drug | Surgical | Number of Hp | | Mortality |
|----------------------------------|--|-------------------------------------|------------------------|--------------|------------------|-----------|
| inhibitors of cholinesterases | of hours before the beginning of Hp | treatment Atropine, TMB-4 | lavage of intestine | 800 C | 800 A2 800 A4 | |
| 1. Soldep | | Comp., Serum | | | | |
| (trichlorfon) | 10.5 | cholinesterase Behring | 1 | 33 | - | 1 |
| Arpalit | | | | | | |
| (trichlorfon) | 48.0 | -,,- | - | 2 | - | - |
| 3. Phosdrin 24 EC | | | | | | |
| (mevinphos) | 16.0 | -,,- | - | 6 | - | 1 |
| 4. Metathion E 50 | | | | | | |
| (fenitrothion) | 25.0 | -,,- | 5 | 16 | 2 | 1 |
| 5. Decemption EK | | | | | | |
| 20 (phosmet) | 38.0 | -,,- | - | 1 | 1 | 1 |
| 6. Unknown | 10.0 | -,,- | - | - | 2 | - |
| Total: | 16.5 | 50 | 6 | 58 | 5 | 4 |

Hp-haemoperfusion, C-Active charcoal, A-Amberlite

Mean age was 32 years. Fifty patients after acute oral poisoning by organophosphate inhibitors of cholinesterases were included in the second group of patients. Among them there were 36 men and 14 women, with mean age of 38 years. In the third group of patients were 20 patients suffered from acute oral ethylene glycol poisoning. There were 18 men and 2 women, mean age was 41 years in the last group. In all patients the present vital clinical sings and laboratory parameters showed an acute disease. Each patient obtained a

conservative medical treatment and various forms of extracorporeal elimination therapy (6,10,11).

Results

The most important clinical signs, laboratory parameters, used treatment and mortality of the patients are described in Tables 1 - 7.

| Number of patients | Mean age (years) sex | Accidental oral poisoning | Metabolic acidosis (pH) | Blood leucocyte count (x.10 ⁹ /L) | Oxaluria (n) |
|--------------------------|-------------------------|---------------------------|----------------------------|---|-----------------|
| 20 | 41 ± 8 | Syntol 190 - 3 | 7.06 | 26.4 | 17 |
| | | Fridex - 10 | ± | ± | |
| | 18 M 2 W | Ethylene - 7 glycol | 0.14 | 5.5 | |

M-man, W-woman

Table 6. Clinical and laboratory parameters in 20 patients after acute ethylene glycol poisoning

| Oligoanuria (days) | Serum urea (mmol/L) | Serum creatinine (µmol/L) | Serum K (mmol/L) | Serum Na (mmol/L) | Serum ALT (µkat/L) |
|-----------------------|---------------------------------------|---------------------------------|---------------------|----------------------|--------------------------|
| 6.5 | 27.6 | 447.4 | 5.0 | 135.9 | 1.23 |
| ± | ± | ± | ± | ± | ± |
| 2.5 | 11.5 | 85.2 | 0.8 | 5.4 | 0.45 |
| ALT IL | · · · · · · · · · · · · · · · · · · · | | | | |

ALT-alanin aminotransferase

Table 7. Extracorporeal elimination treatment in 20 patients after ethylene glycol poisoning

| Treatment by ethylalcohol (100 mg | Alkaline treatment | Numb | er of HD | Duration ofillness | Mortality | |
|--------------------------------------|-----------------------|------|----------|-----------------------|-----------|--|
| % in dialysis solution) | | Ac | Bi | (days) | | |
| 12 | 19 | 28 | 197 | 62 ± 25 | 3 (15.0%) | |

HD-haemodialysis, Ac-acetate, Bi-bicarbonate

Discussion

Collaboration of the nephrologist and the intensivist is a very urgent and necessary issue during 24 hours a day, regardless of the fact whether the patient has been hospitalized in DAIM or ICU of the IVth Internal Clinic or Nephrological Clinic of Faculty Hospital of L. Pasteur, Košice. However, the patients suffered from ARF after trauma or with multiorgan failure were hospitalized only in DAIM. The most effective treatment in this group of patients from the nephrological point of view, besides the antibiotic and surgical treatment, was undergoing haemodialysis because the uremic syndrome with hyperkaliemia developed during several hours or days after the polytrauma occured. Two patients died due to hyperkaliemia as a consequence of late admission to dialysis centre, 6 patients died due to sepsis (2) and 4 patients died after repeated haemorrhage in the gastrointestinal tract. In the treatment of ARF of traumatic origin with multiorgan failure, a complex collaboration of intensivist, nephrologist, traumatologist, general surgeon, general internist, biochemist and other specialists was necessary (6).

Patients after acute organophosphate inhibitors of cholinesterases poisoning underwent an adequate conservative medical treatment and haemoperfusion until 24 hours after the poisoning, despite of the big difference between a relatively small troubles of patients and serious anamnestic data and clinical symptoms. Gastrointestinal lavage was performed in each patient, atropine was repeatedly injected subcutaneously and Serum cholinesterase - Behring was intravenously administered and a single reactivator of cholineasterase was used at the onset of acute poisoning. Besides charcoal haemoperfusion, amberlite XAD-4 haemoperfusion was used in the later years. In 5 patients after acute metathione poisoning and in 1 patient after trichlorfon poisoning, surgical lavage of intestine was performed. An intensive collaborative treatment of nephrologists and intensivists, in 6 patients also with the surgeon, led to a recovery in 46 patients (92%), while only 4 patients died. The cause of death in two of them was pneumonia, preceding chronic obstructive а disease. In bronchopulmonary other two patients hypokaliemia participated as a cause of death (12,10).

In the treatment of acute ethylene glycol poisoning early diagnosis and treatment of severe acidosis by bicarbonate dialysis with 100 mg% concentration of ethylalcohol in dialysis solution were the most important issues. Ethylalcohol was used as an antidote. Combined treatment of ethylene glycol poisoning led to the improvement of neurological status, to the disappearance of metabolic acidosis, leucocytosis and oxaluria. Acute renal failure was reversible in all patients except one in whom there were two simultaneous causes of ARF, ethylene glycol poisoning and polytrauma. In this patient we performed 111 haemodialyses and thereafter he underwent a successful renal transplantation. From the total number of 20 patients after ethylene glycol poisoning three patients died in the period

before the possibility to use bicarbonate haemodialysis was available (11).

Conclusion

Clinical analysis of the three groups of patients suffering from acute serious diseases (ARF of traumatic origin, acute intoxication by organophosphate inhibitors of cholinesterases and acute intoxication caused by ethylene glycol) showed the despaired need for collaboration between nephrologists, intensivists and other specialists in establishing an early diagnosis and securing either an effective conservative medical treatment, or surgical procedures and extracorporeal elimination therapy. This fact led to the significant improvement of the prognosis in those patients.

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