Liver and kidney damage in acute poisonings

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Abstract

In the paper the retrospective analysis of simultaneous damage of liver and kidneys after acute carbon tetrachloride poisoning in 60 patients, after mushroom poisoning (Amanita phalloides) in 81 patients and after ethylene glycol poisoning in 20 patients from the view of differential diagnosis, conservative, intra- and extracorporeal elimination therapy is described. Acute toxic hepatitis with the different degree of severity was present in all patients and even acute renal failure was present in some patients. After acute carbon tetrachloride poisoning acute hepatitis developed approximately simultaneously with the development of acute renal failure. After acute Amanita phalloides poisoning acute toxic hepatitis, which was the cause of death in liver coma in 16 patients, quickly developed in the foreground of the clinical picture. Renal damage was less frequent and it was not the cause of death even in one patient. After acute ethylene glycol poisoning acute renal failure dominated with severe metabolic acidosis, oxaluria and leucocytosis, acute toxic hepatitis was less severe. After ethylene glycol poisoning 3 patients died in the period before the use of the bicarbonate haemodialysis was possible. During 30 years the intra- and extracorporeal elimination therapy qualitatively developed (bicarbonate haemodialysis, haemoperfusion through active charcoal and other sorbents, plasmapheresis, haemofiltration), which participates to the significant degree on the recovery and improvement of the prognosis of patients after acute poisonings with simultaneous damage of liver and kidneys.

Key words: acute poisoning, carbon tetrachloride, mushroom (Amanita phalloides), ethylene glycol, hepatic and renal damage, extracorporeal elimination therapy

Introduction

Simultaneous damage of liver and kidneys in acute poisoning occurs very often in present times (Table 1). This fact is very important from the diagnostic, differential diagnostic and therapeutic point of view (1-6). Besides that a damage of other organs after acute poisoning can cause multiorgan failure in patients previously healthy (2,5). This finding is very important in patients with preceding illness (2,7,8).
The aim of the study was the retrospective analysis of 60 patients after carbon tetrachloride poisoning, of 81 patients after mushroom poisoning (Amanita phalloides) and of 20 patients after ethylene glycol poisoning with the liver and kidney damage from the diagnostic, differential diagnostic, conservative, intra- and extracorporeal elimination treatment point of view.

Table 1. Simultaneous damage of liver and kidneys in acute poisonings

Acute poisoning	Damage of Liver	Kidneys
1. Carbon tetrachloride	+	+
2. Dichlorethane	+	+
3. Ethylene glycol	+	+
4. Paraquat, Diquat	+	+
5. Mushroom (Amanita phalloides)	+	+
6. Arsenic hydride	+	+
7. Mercury chloride	-	+
8. Trichloroethylene	+	-+
9. Paracetamol	+	-+
10. Carbamazepine	-+	-
11. Isoniazid	+	-
12. Other	-+	-+

Patients and Methods

Sixty men, mean age was 41±9 years, after carbon tetrachloride poisoning were investigated. Among them 52 patients were poisoned by inhalation way and 8 patients drunk carbon tetrachloride in mistake of other drinks. At the beginning of acute poisoning dyspeptic syndrome was dominated, especially after oral poisoning. All patients used ethylalcohol for „the treatment of acute dyspepsia". Use of ethylalcohol potentiated carbon tetrachloride poisoning (2,7,8). Clinical and laboratory signs after acute carbon tetrachloride poisoning from liver damage were present earlier (several hours or days) in comparison to the signs of kidney damage. From that reason one third of poisoned patients was admitted to the Clinic for Infection Diseases in Faculty Hospital, Košice or to the other Departments of Infection Diseases in Eastern Slovakian region. That diagnostic mistake was caused by insufficient anamnesis, minimization of the contact with carbon tetrachloride by patients or deliberate concealment of the contact with that poison. Carbon tetrachloride is a very good lipid solvent and in the past it was used in home setting and in many workplaces (1,7,8). Except this carbon tetrachloride was used as a filling in fire-extinguishers (2).
Eighty-one patients were investigated after mushroom poisoning (Amanita phalloides), mean age was 30.5±9 years. Among them were 28 children and 53 adult patients. Clinical and laboratory signs after acute poisoning of Amanita phalloides were present first of all during the several hours as an acute dyspepsia with the rapid increase of serum bilirubin and of activity of serum transaminases. Acute renal failure developed later or was not present. All acute poisoning caused by Amanita phalloides were accidental except one

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young lady, who ate Amanita phalloides in attempted suicide (3,9,10).

The third group of patients were 20 patients after ethylene glycol poisoning. Mean age was 41± 8 years, among them were 2 women and 18 men. Ethylene glycol was the most frequent oral poisoning in a form of antifreeze agent which was used in the cars. That poison was used frequently in the change for ethylalcohol or from „ absence of ethylalcohol drinks“ in three soldiers. Ethylene glycol is a very dangerous poisoning nowadays and it is relatively frequent in our country in present times (3,5,6,11,12).

In three groups of patients with those poisonings all available diagnostic and therapeutic methods were used including various forms of intra-and extracorporeal elimination methods (1-8,10-16). In the treatment of carbon tetrachloride poisoning we did not use a hyperbaric oxygen therapy (17).

Results

Clinical signs and some laboratory parameters, conservative and intra-and extracorporeal elimination treatment in the patients after carbon tetrachloride, mushroom (Amanita phalloides) and ethylene glycol poisonings were described in Tables 2-4.

Table 2. Liver damage in acute poisoning

Poisoning	Number of patients	Hepatomegaly	Bi (µmol/L)	Serum ALT	AST (µkat/L)	Coma
1. Carbon tetrachloride	60	57	78.4±20.5	16.2±3.3	-	4
2. Mushroom (Amanita phalloides)	81	81	76.8±14.1	89.5±12.4	88.7±12.8	16
3. Ethylene glycol	20	16	17.5± 2.0	1.23± 0.45	1.12±0.4	5

Bi-bilirubin

Table 3. Kidney damage in acute poisoning

Poisoning	Number of patients	Proteinuria (g/24h)	K (mmol/L)	Serum Urea	Creatinine (µmol/L)	Metabolic acidosis (pH)
1. Carbon tetrachloride	60	57	78.4±20.5	16.2±3.3	-	4
2. Mushroom (Amanita phalloides)	81	81	76.8±14.1	89.5±12.4	88.7±12.8	16
3. Ethylene glycol	20	16	17.5± 2.0	1.23± 0.45	1.12±0.4	5

Table 4. Intra-and extracorporeal elimination therapy in acute poisoning

Poisoning	Number of patients	Intra-and extracorporeal elimination therapy					Mortality
		PD	HD	ET	HP	PF	
1. Carbon tetrachloride	60	8	81	6	-	4	2 (3.3%)
2. Mushroom (Amanita phalloides)	81	-	20	2	128	23	24 (29.6%)
3. Ethylene glycol	20	-	28Ac+147Bic	-	5	-	3 (15%)

PD-peritoneal dialysis, HD-haemodialysis, Ac-acetate, Bic-bicarbonate, ET-exchange transfuzion, HP-haemoperfusion, PF-plasmapheresis

Discussion

In differential diagnosis of the patients with simultaneous damage of the liver and kidneys, a direct anamnesis of patients is very important, but also an indirect anamnesis from family members or from co-workers. It is necessary to distinguish acute viral hepatitis from acute carbon tetrachloride poisoning, from poisoning by dichloroethane, mushroom poisoning (Amanita phalloides), arsenic hydride, ethylene glycol and from acute leptospirosis (1-3, 5,6,8-10,12,16,18). After acute paraquat poisoning and simultaneously with a damage of the liver and kidneys, the damage of lung and heart is developed. The patient who is insufficiently treated after paraquat poisoning is dying during the following 10-14 days. Cause of death after that poisoning is alveolocapillary block in the lung (19,20). Acute arsenic hydride poisoning in our department was not present during the last 35 years (5,21-23). Acute mercury chloride poisoning leads to acute renal failure without liver damage (5). Acute trichloroethylene poisoning is manifested by cerebral and liver damage. Damage of the kidneys is very rare but it was described (24). Acute paracetamol poisoning leads to liver damage, but kidney damage in some patients was also described (25-27). After oral carbamazepine poisoning important liver damage was observed (28). Liver damage after isoniazid poisoning was also observed. High intravenous dose of pyridoxine (5g/24 h) as an antidote in that poisoning was recommended (5). We successfully used this therapy in one our patient after severe isoniazid poisoning.

Acute toxic hepatitis dominated in the first group of patients who were poisoned by carbon tetrachloride, in four of them developed liver coma. Two patients suffering from preceding chronic hepatitis died in liver coma despite of the use of repeated haemodialyses and exchange transfusions. Anamnestic serum titres against Leptospira grippotyphosa were found in one patient suffering from acute carbon tetrachloride poisoning and in another patient with the same poisoning acute leptospirosis developed caused by Leptospira sejrő. Dynamics of titres testified to acute illness (2). Determination of serum titres against leptospirosis was very important especially in the patients from agricultural region. Uremic syndrome as a sign of acute renal failure in acute carbon tetrachloride poisoning developed relatively slowly in comparison to other acute poisonings (2). Duration of acute carbon tetrachloride poisoning until to restore to normal of hepatic tests and of renal functions was in average 39±9 days. The treatment of carbon tetrachloride poisoning included: use

of hepatotropic drugs, forced diuresis, repeated haemodialyses, peritoneal dialysis and in patients in liver coma we used exchange transfusions and later plasmaphereses (2,8). Occurrence of acute tetrachloride poisoning is very rare in the last years, because it was due to prohibition to use of carbon tetrachloride in fire-extinguishers, in industry and in various chemical laboratories.

In the second group of 81 patients, who were poisoned by mushroom (Amanita phalloides), acute toxic hepatitis developed rapidly until to liver insufficiency and coma. From this group of patients 24 patients (29,6%) died in liver coma. Damage of the kidneys after Amanita phalloides poisoning was less frequent, despite that in 16 patients acute tubular necrosis with kidney failure developed. No one patient died caused by acute renal failure. That dissociation of liver and kidney damage is characteristic for hepatotoxic type of acute mushroom poisoning. Conservative treatment including of hepatic drugs and of forced diuresis and especially of extracorporeal elimination therapy we used relatively late for the late admission of the patients to our renal unit (in average 53 hours after poisoning).

During many years we tried to use early haemodialysis, charcoal haemoperfusion, Amberlite XAD-2 and XAD-4 haemoperfusions and in the last years plasmapheresis which is the bridge to liver transplantation. Haemodialysis was used on the influence of uremic syndrome (3,5,9,10,13,14).

In the third group of patients with acute ethylene glycol poisoning dominated severe metabolic acidosis, leucocytosis, oxaluria and various neurological symptoms. Leucocytosis was present from the onset of acute poisoning caused by ethylene glycol irritation of the bone marrow and duration of that laboratory sign was in average 7 days. Damage of the kidneys with relatively slow development of acute renal failure showed for the important disturbances of renal functions. Damage of the liver with hepatomegaly and increase of serum activity of alanine aminotransferase were less important and occurred in 16 patients (11,12). Dissociation of the liver and kidney damage was present after ethylene glycol poisoning in disadvantage for the kidney damage. In the medical treatment of ethylene glycol poisoning were applicated hepatic drugs including of the group of vitamin B. The therapeutic method of choice in ethylene glycol poisoning was in the last years bicarbonate haemodialysis using 100 mg% ethylalcohol in dialysis solution. Ethylalcohol was used as a blocker of alcoholdehydrogenase in metabolic transformation from ethylene glycol to glycol aldehyde. Two patients died after ethylene glycol poisoning before the possibility to use bicarbonate haemodialysis and one patient died for the late admission to our dialysis unit (11,12).

At the end of this retrospective study it is neccessary to say, that the review of simultaneous damage of the liver and kidneys after carbon tetrachloride poisoning, mushroom poisoning (Amanita phalloides) and ethylene glycol poisoning contributed to diagnosis, differential diagnosis and also to the use of various forms of extracorporeal elimination therapy. In the near future several new forms of extracorporeal elimination therapy will be used in the treatment of acute poisoning i.e. albumin dialysis and the use of new sorbents for toxins removal mainly for toxins with high albumin and other proteins binding in plasma (29-31).

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