

# Serum Electrolytes Profile During Accidental Acute Poisoning in Children at Charles De Gaulle Pediatric University Hospital of Ouagadougou

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**Abstract:** Accidental acute poisoning (AAP) in children is a significant public health problem worldwide. Their adequate management requires relevant biological elements. The objective of this study was to evaluate ionic disturbances during acute poisoning in children aged from 0 to 15 years admitted to the Charles De Gaulle Pediatric University Hospital (CHUP-CDG) of Ouagadougou, Burkina Faso. A retrospective descriptive and analytical study was therefore conducted over a period of 3 years at CHUP-CDG. It was performed in the pediatric, intensive care and laboratory departments of the CHUP-CDG, and involved children aged from 0 to 15 years with complete clinical records. A total of 193 patients were included, with a mean age of 18.82±24.85 months and M/F sex ratio of 1.38. The hospital incidence of AAP was 2.43% and the mortality rate 16.06%. Phytomedicinal intoxications were the most common (39.38%), followed by caustic products (19.69%). On admission, serum electrolytes showed hypobicarbonatemia (64.23%), hyperchloremia (55.24%), hyponatremia (33.78%), hypoproteinemia (21.47%) and hyperkalemia (12.16%). At control, hypobicarbonatemia (28.57%), hyperchloremia (75%), hypoproteinemia (28.57%), hyperkalemia (12.50%) and here hypernatremia (25%) were found. The deceased patients had significantly lower natremia ( $p=0.0442$ ), chloremia ( $p=0.0007$ ) and proteinemia ( $p=0.0004$ ) than the other patients in the study. The high death rate was related to herbal medicines and caustic intoxications. Many hydroelectric disorders were found in the study. These disorders could be explain par digestive losses. Indeed, the main clinical signs found in the study were vomiting and diarrhea in children. In addition, acute renal failure in patients could also explain these ionic disturbances. AAP are at the root of multiple ionic disorders, which may be responsible for life-threatening complications in patients.

**Keywords:** Electrolytes, Sodium, Bicarbonates, Accidental Acute Poisoning, Child, CHUP-CDG, Burkina Faso

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## 1. Introduction

Accidental acute poisoning (AAP) is a major public health

problem affecting children throughout the world [1–3]. According to a World Health Organization (WHO) report, acute poisoning is the fourth most common cause of childhood morbidity and mortality [4]. It is defined as all the

pathological manifestations following an ingestion of food, the administration of products or drugs behaving like a poison in the body. Poisoning in children is a fortuitous and damaging event that occurs suddenly in the home or in the neighborhoods [5]. Although they are often accidental, these accidents are linked to sociological and economic specificities such as medicines left within the reach of children; clumsy behavior, represented by the decanting of toxic products into containers or bottles for food use (bleach, acids, pesticides, insecticides). In addition to the classic accident, parents can intoxicate their children by making medication errors or by administering dangerous traditional therapies [6].

In Mali, a study in 2019 reported 80 cases of intoxication in pediatric emergencies at the Gabriel Toure University Hospital over two years, representing a hospital frequency of 1.47% and a lethality rate of 17.5% [7]. In 2009, in Burkina Faso, a study conducted in Ouagadougou at the pediatric emergency department of the Charles de Gaulle Pediatric University Hospital (CHUP-CDG) found 103 cases of AAP, representing 1.3% of hospitalizations over a two-year period, with a 3% mortality rate [8]. In 2017, a study in Bobo-Dioulasso conducted at the pediatric emergency unit of the Sourou Sanou University Hospital reported 240 AAP over a five-year period, corresponding to a frequency of 0.87%, and a mortality rate of 2.92 [9].

Despite these available data on pediatric AAP, the biological aspects remain unexplored, as attested to by the limited literature in this area [10].

Indeed, in our context, several studies have explored the epidemiological, clinical, etiological, therapeutic and evolutionary aspects of AAP, but few studies on the biological dimensions. However, biological analysis is essential, as it allows to evaluate the severity of the intoxication and to rapidly correct its effects. It can also represent a useful element for orienting the diagnosis, in the absence of any particular presumption as to the nature of the product in cause [11].

A study conducted in 2013 in Morocco in the intensive care unit of the Sidi Mohamed Ben Abdellah University Hospital found that 48% of patients presented hyperleukocytosis, 14% hepatic cytolysis, 5% respiratory acidosis, 7% renal insufficiency, 23% hydroelectrolytic disorders and finally 5% of them had an elevated lactate dehydrogenase [6]. AAP are a daily concern in the pediatric setting because of their frequency, diversity and severity. The lack of studies on biological aspects, mainly on ionic parameters, led us to ask the following question: what ionic disorders are present during AAP? Secondly, the potential severity of these disorders must be considered. Thus, the study was conducted to evaluate the impact of AAP on the child's organism, particularly the impact on ionic parameters, in order to ensure better patient management. The main objective was therefore to study the ionic disorders associated with AAP in patients aged from 0 to 15 years, admitted to CHUP-CDG.

## 2. Material and Methods

### 2.1. Framework and Period of Study

The study was performed at the Charles de Gaulle University Pediatric Hospital (CHUP-CDG) in Ouagadougou, in the laboratory, intensive care and medical pediatrics departments, over a 3-year period, from January 1, 2018, to December 31, 2020.

### 2.2. Patients

The study involved patients aged from 0 to 15 years, hospitalized for AAP during the study period in the selected CHUP-CDG clinical services (intensive care and medical pediatrics). Were included those patients having performed serum electrolytes on admission and having a usable clinical record. Thus, any patient record with a diagnosis of AAP and in whom an assay of at least one ionic parameter was performed on the patient's admission to CHUP-CDG was selected. Patients seen as outpatients and cases of envenoming were not included.

### 2.3. Methods

A retrospective study was performed for descriptive and analytical purposes. Data were collected from the clinical records of the hospitalized children, from the hospitalization registers and from the laboratory service. Following variables were studied: epidemiological variables (age and sex of patients), clinical variables (time of consultation, type of toxicant, mode of discharge of patients) and biological variables (natremia, kalemia, chloremia, bicarbonatemia and proteinemia). Epidemiological data of the patients and the diagnosis of intoxication were obtained from the hospitalization records. Clinical records were used to extract the nature of the product absorbed. Biological data of the patients were obtained from the records of the medical analysis laboratory. Complete serum electrolytes results were extracted for all patients in the study on admission and for some on follow-up. These results were obtained by assaying the various parameters of serum electrolytes on a venous blood sample in a dry tube, centrifuged at 3500 rpm for five minutes and from which the serum was taken [12].

Serum sodium, potassium, and chlorine were determined by potentiometry [13], with respective normal values of 130-145 mmol/L, 3.5-7 mmol/L, and 95-110 mmol/L in neonates and infants; 135-145 mmol/L, 3.5-5.5 mmol/L, and 98-105 mmol/L in children older than 30 months. For bicarbonate ions, the phosphoenolpyruvate carboxylase enzymatic method [14] was used with the following normal values: 14-27 mmol/L in newborns and infants; 20-28 mmol/L in children over 30 months. The biuret method [15] was used to determine total protein, with normal values of 45-70 g/L in newborns and infants and 60-80 g/L in children over 30 months of age.

Descriptive statistics were performed using Excel version 2019. Analyses were performed with Stata software version 16.0, including Student's T test and ANOVA test to compare the means of the parameters between the different

variable modalities; with a 95% confidence interval and a significance level of  $p < 5\%$ .

The study obtained authorization from the CHUP-CDG executive management and the data were treated with confidentiality.

### 3. Results

#### 3.1. Epidemiological Data

##### 3.1.1. Frequency of AAP

During the study, 9023 patients, all medical pathologies

included, were hospitalized in clinical services concerned by the study and 219 of them were diagnosed with AAP; representing a hospital incidence of intoxication of 2.43%. Among them, 193 patients, whose clinical records fulfilled the criteria, were included.

##### 3.1.2. Age Distribution of Patients

All pediatric age groups were represented (Table 1). Infants represented the largest number of cases with 72.02%, followed by neonates (17.10%). The mean age was  $18.82 \pm 24.85$  months.

**Table 1.** Distribution of patients by age group.

Age ranges	Number	Frequency (%)
Newborns [0-28 days]	33	17.10
Infants [28 days-30 months]	139	72.02
Toddlers [30 months-5 years]	12	6.22
Older children / Teenagers [5-15 years]	9	4.66
Total	193	100

##### 3.1.3. Gender Distribution of Patients

In the study, male patients represented 58.03% and female patients 41.97%. The sex ratio M/F was 1.38.

#### 3.2. Clinical Data

##### 3.2.1. Distribution of Patients According to Time from Accident to Consultation at CHUP-CDG

Of the patients, only 2.59% ( $n=5$ ) were treated at a health facility within 1 hour of intoxication ( $\leq 1$  hour); 33.68% ( $n=65$ ) were treated between 1 and 6 hours; and 13.47% ( $n=26$ ) between 6 and 12 hours. The great majority of patients, 50.26% ( $n=97$ ), were managed beyond 12 hours.

##### 3.2.2. Distribution of Patients by Intoxication Route and Type of Toxicant

AAP occurred by the oral route in the largest majority of cases (96.89%,  $n=187$ ). The respiratory (2.07%,  $n=4$ ), percutaneous (0.52%,  $n=1$ ) and ocular (0.52%,  $n=1$ ) routes represented the other routes of poisoning.

Regarding the type of toxicant, intoxications by phytomedicines were the main type with 39.38% of cases. Poisoning by caustic products was 19.69% (bleach, caustic soda and acid) and by pesticides 7.77% (insecticides, herbicides and rat poisoners). Poisonings by other types of toxicants represented 33.16%. These included intoxications by spoiled food products (yogurt, meat, juice, cashew fruit), drug intoxications (paracetamol, amoxicillin, polyvitamins, antihypertensives, antihistamines, antimycotics, corticosteroids, estrogen-progester, and antivitamin K). There were also intoxications by hydrocarbons (gasoline, drain oil, petroleum), foaming products (detergents, soaps), psychoactive substances (alcohol, tobacco, psychotropic drugs), cosmetic products (relaxers, solvents, perfumes, ointments), gaseous products (carbon monoxide, butane gas) and battery powder.

##### 3.2.3. Distribution of Patients by Mode of Discharge

The outcome of the patients was recovery in 81.35% of

cases ( $n=157$ ), escape or discharge against medical advice in 2.59% of patients ( $n=5$ ) and death in 16.06% of cases ( $n=31$ ).

The mortality rate in the study was therefore 16.06%. The types of toxicants involved in these cases of death were pesticides (93.55% of cases of death) and caustics (6.45%).

#### 3.3. Biological Data

##### 3.3.1. Distribution of Patients According to Serum Electrolytes Results at Admission

Figure 1 shows the distribution of patients according to serum electrolytes on admission to CHUP-CDG.

Hypobicarbonatemia was found in 64.23% of cases (bicarbonatemia performed in 137 patients), hyponatremia 33.78% ( $N=148$ ) and hypoproteinemia 21.47% ( $N=163$ ). In addition, hyperchloremia was present in 55.24% of patients ( $N=143$ ) and hyperkalemia in 12.16% ( $N=148$ ).

##### 3.3.2. Variations in Serum Electrolytes Parameters on Admission According to Epidemiological and Clinical Characteristics

On admission, kalemia was significantly higher in males ( $p=0.0311$ ) and neonates ( $p=0.000$ ); proteinemia significantly lower in older children and adolescents ( $p=0.0003$ ) (Table 2).

According to the time from accident to consultation at CHUP-CDG (Table 3), natremia was significantly lower in children received within one hour of intoxication ( $p=0.0113$ ) and proteinemia was lower in those received after 12 hours ( $p=0.000$ ). Regarding the administration route of the toxicant (Table 3), routes other than oral were associated with hyponatremia ( $p=0.0366$ ) and hyperkalemia ( $p=0.0172$ ). Depending on the type of toxicant, kalemia was significantly higher ( $p=0.0048$ ) and proteinemia lower ( $p=0.0048$ ) with phytomedicines (Table 3).

Deceased patients had significantly lower natremia ( $p=0.0442$ ), chloremia ( $p=0.0007$ ) and proteinemia

( $p=0.0004$ ) than other patients (Table 3).

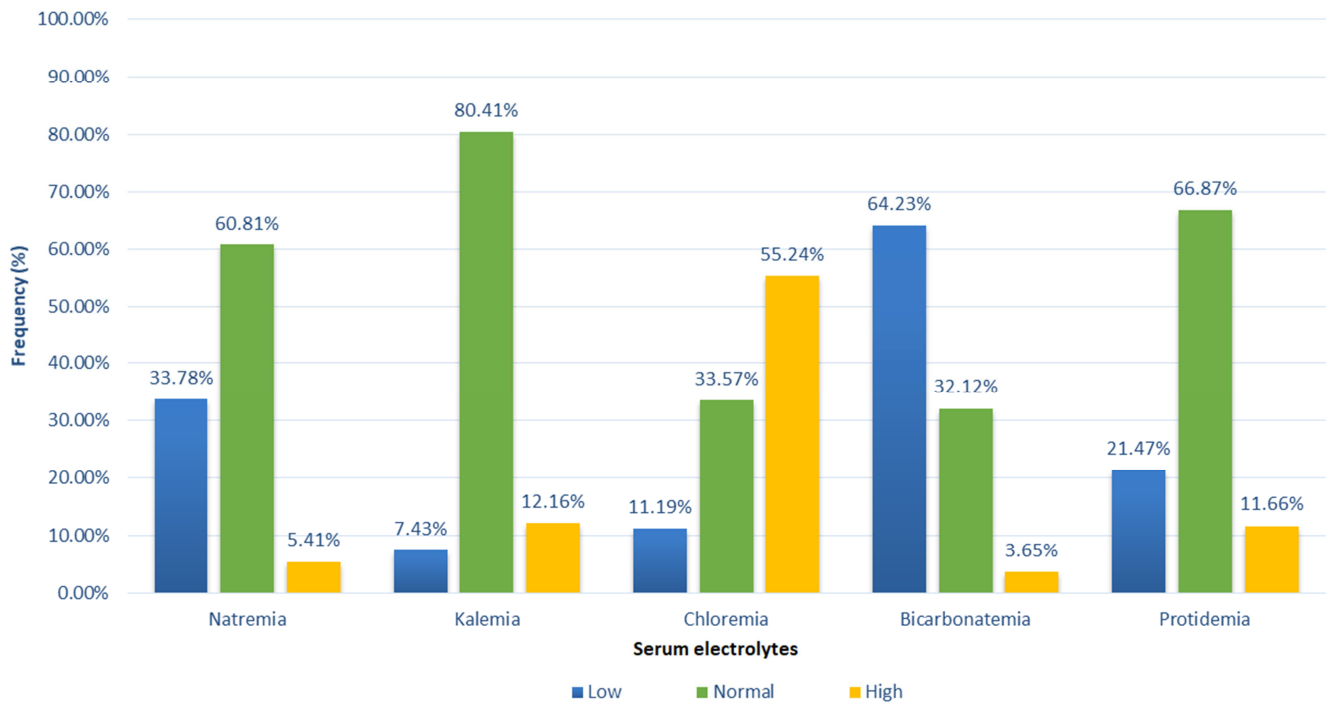


Figure 1. Distribution of patients by serum electrolytes results on admission.

Table 2. Comparison of mean serum electrolytes values at admission according to epidemiological data.

Parameters		Natremia		Kalemia		Chloremia		Bicarbonatemia		Proteinemia	
		mmol/L	p-value	mmol/L	p-value	mmol/L	p-value	mmol/L	p-value	g/L	p-value
Gender	Male	132.99	0.7924	4.98	0.0311	106.75	0.3033	18.04	0.8716	65.35	0.4518
	Female	131.99		4.53		108.62		18.18		66.73	
	Newborns	132.15		5.90		110.13		18.13		62.63	
Age	Infants	133.41	0.6111	4.62	0.000	106.98	0.5905	17.96	0.6701	66.23	0.0003
	Toddlers	122.91		4.35		106.73		17.44		78.79	
	Older children / Teenagers	134.30		3.95		106.18		20.57		56.68	

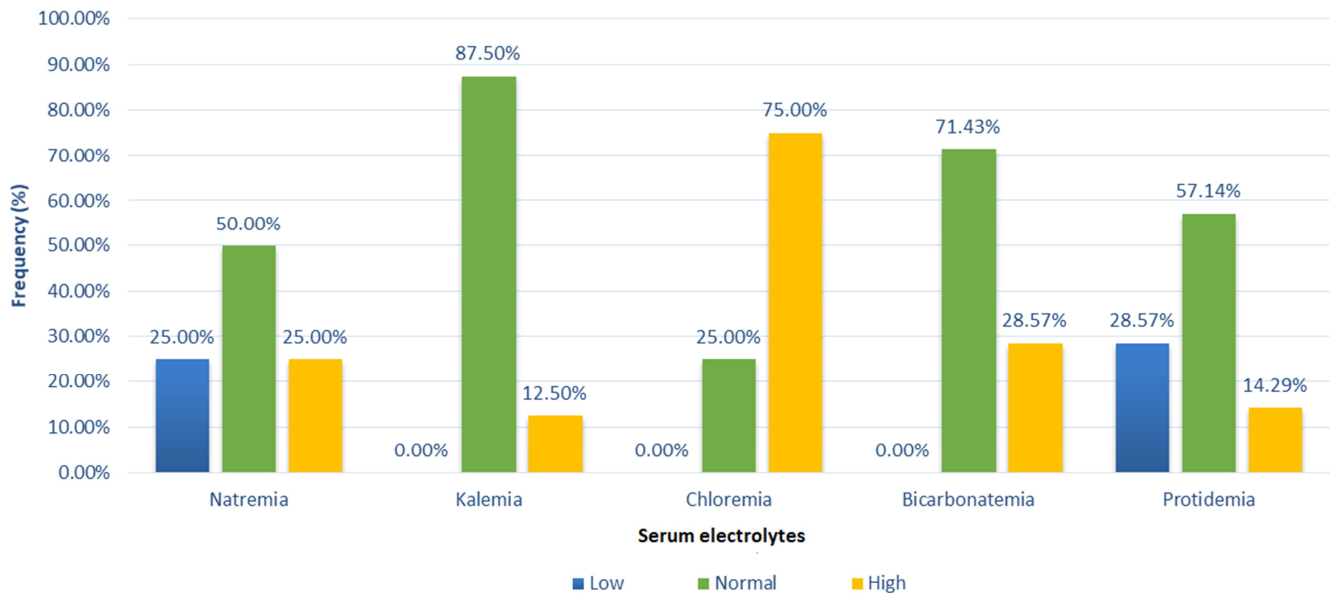
Table 3. Comparison of mean serum electrolytes values at admission according to clinical data.

Parameters		Natremia		Kalemia		Chloremia		Bicarbonatemia		Proteinemia	
		mmol/L	p-value	mmol/L	p-value	mmol/L	p-value	mmol/L	p-value	g/L	p-value
Time to from accident to consultation at CHUP-CDG	≤ 1H	132.70	0.0113	4.42	0.4414	108.62	0.0777	18.00	0.9364	69.85	0.000
	]1H-6H]	137.30		4.70		110.03		17.79		70.15	
	]6H-12H]	140.05		4.54		108.94		18.78		71.73	
	> 12H	134.52		4.97		105.14		18.11		61.12	
Intoxication route	Oral route	136.45	0.0366	4.76	0.0172	107.61	0.3961	18.01	0.3326	65.74	0.2310
	Others	128.82		6.27		103.05		20.75		72.75	
	Phytomedicines	134.84		5.24		105.41		18.17		60.69	
Types of toxicant	Caustics	138.29	0.1775	4.51	0.0048	108.99	0.2867	18.09	0.9652	68.31	0.000
	Pesticides	137.51		4.35		108.42		18.64		71.35	
	Others	136.44		4.53		108.98		17.78		69.81	
	Recovery	136.80		4.82		108.78		18.46		67.23	
Mode of discharge	Escape/Against medical advice	140.15	0.0442	4.42	0.8773	112.40	0.0007	15.50	0.2386	74.67	0.0004
	Death	132.84		4.73		99.62		16.48		58.14	

### 3.3.3. Distribution of Patients According to Serum Electrolytes Results at Monitoring

The serum electrolytes were checked in only eight (08) patients in the study. The disturbances found were hyperchloremia (75%, n=8), hyponatremia 25% (n=8),

hypoproteinemia in 28.57% (n=7), hyperkalemia (12.50, n=8) and decrease in bicarbonate ions in 28.57% of patients (n=7). Figure 2 shows the distribution of patients according to serum electrolytes at first check.



**Figure 2.** Distribution of patients according to serum electrolytes results at first check.

## 4. Discussion

Because of the retrospective nature of the study, we were confronted with the incompleteness of the clinical records, making them unusable. In addition, despite the presence of many ionic disorders on admission, monitoring of this test was performed in only eight patients, i.e., only 4.14% of patients. Thus, the evolution of the recorded ionic disorders could not be evaluated in the study. In addition, the literature did not find enough studies on the biological profile of AAP.

The reported hospital incidence in the study was 2.43% and the mortality rate, 16.06%. Several ionic disorders were present on admission of patients to the CHUP-CDG. Thus, hypobicarbonatemia was found in 64.23% of them and hyperchloremia in 55.24%. In addition, hyponatremia, hyperkalemia and hypoproteinemia were present in 33.78%, 12.16% and 21.47% respectively (Figure 1).

Bicarbonate ion has significant role in pH changes and the decrease in its concentration leads to the development of metabolic acidosis. In the study, the main mechanism incriminated in hypobicarbonatemia is represented by bicarbonate digestive losses, compensated by chloride ions (justifying the hyperchloremia). Indeed, the main clinical signs found in the study were vomiting and diarrhea in children, explaining the digestive losses in bicarbonates.

Another mechanism that could explain the decrease in bicarbonate ions would be the occurrence of lactic acidosis, caused by the ingestion of toxic substances in the body, as

highlighted by many studies [16–19]. Indeed, the ingestion of some toxic substances (especially drugs, acids) is accompanied by the accumulation of toxic acid metabolites. In addition, acute renal failure in patients could also explain these ionic disorders.

The blood volume study in the patients would have provided orientation to the mechanism of certain disorders, in this case hyponatremia. Given the retrospective context, clinical and biological data essential to volemic status assessment were not included. In general, hyponatremia can result from either sodium deficiency, water excess, or potassium deficiency. For this study, hyponatremia would probably be depletion (deficit), resulting from a negative sodium balance, which is more deficient than water balance. More precisely, it would be due to sodium elimination excess, through extrarenal losses (vomiting, profuse diarrhea) or renal losses (interstitial nephropathy due to toxins) [17]. Another mechanism could also explain hyponatremia: the syndrome of inappropriate antidiuretic hormone secretion (SIADH). Indeed, SIADH leads to increased synthesis of antidiuretic hormone, compromising water excretion and urinary concentration mechanisms. It manifests as hyponatremia and its symptoms, including neurological ones. There are several possible causes of SIADH, including drugs and other toxicants [20].

As for potassium, this is essentially regulated by cellular transfer under the dependence of acid-base metabolism, catecholamines, insulin and secondarily by the kidney. The majority of hyperkalemiases are due to renal insufficiency and to medication.

Indeed, in acute renal failure (which occurs in AAP),

hyperkalemia occurs due to decreased distal tubular flow or distal tubular necrosis that reduce potassium secretion. With regard to drug intake, potassium movement from the intracellular to extracellular medium (cell shifts) can be modulated by some drugs; which interfere with potassium uptake and entry into the cells by blocking  $\text{Na}^+$ ,  $\text{K}^+$ -ATPase. In addition, in the case of hyperchloremic metabolic acidosis as described above, the cell being impermeable to chlorine, potassium leaves the cell to maintain electroneutrality, resulting in hyperkalemia. Finally, concerning the proteinemia, its decrease could be explained by the digestive losses.

Concerning serum electrolytes variations according to patients' characteristics at admission, kalemia was significantly higher in male gender ( $p=0.0311$ ). However, mean values of kalemia in both girls and boys remained within normal values. In addition, blood potassium levels were significantly higher in newborns ( $p=0.000$ ); this is easily explained, as normal values are higher at birth. Indeed, as the renal function is still immature, hyperkalemia will appear due to the decrease in distal tubular flow mainly, thus reducing potassium secretion. This could also be explained by pseudo-hyperkalemia, common in the smallest children, due to in vitro hemolysis caused by technical difficulties in obtaining an adequate io sample [21].

Protein levels were significantly lower in older children and teenagers ( $p=0.0003$ ) (Table 2) than in neonates, infants and toddlers. In addition, the mean protein value in older children and teenagers was below the minimum value for age, reflecting hypoproteinemia. The causes of hypoproteinemia are multiple and the mechanisms underlying it are a synthesis defect, renal or digestive leakage, intake deficiency or increased protein catabolism [22].

The digestive and/or renal losses caused by the ingestion of the toxic substances in the study alone cannot explain the hypoproteinemia observed in the population of older children and teenagers. Additional investigations, particularly of nutrition, liver and kidney, would be desirable for a better understanding.

According to the time from accident to consultation at CHUP-CDG (Table 3), the natremia was significantly lower in children who received treatment within the first hour after intoxication ( $p=0.0113$ ) and the proteinemia was lower in those who received treatment after the 12th hour ( $p=0.000$ ). This could be explained by the mode of installation of these disorders: rapid for sodium and slower for proteins. Concerning administration route of the toxicant (Table 3), the routes other than oral were associated with hyponatremia ( $p=0.0366$ ) and hyperkalemia ( $p=0.0172$ ). Depending on the type of toxicant, kalemia was significantly higher ( $p=0.0048$ ) and proteinemia lower ( $p=0.0048$ ) with phytomedicines (Table 3).

Deceased patients had significantly lower natremia ( $p=0.0442$ ), chloremia ( $p=0.0007$ ) and proteinemia ( $p=0.0004$ ) than other patients (Table 3). Hyponatremia is the most common disorder of fluid and electrolyte imbalance encountered in clinical practice. It can cause a wide range of

clinical symptoms, from mild to severe and even life-threatening in the intoxicated individual. Hyponatremia increases the risk of mortality in these cases [23]. Potassium disorders are also common abnormalities. Hypokalemia is the result of an abrupt shift of potassium from the extracellular to the intracellular compartment or more commonly the result of potassium depletion through abnormal losses (digestive or renal). Hypokalemia is generally well tolerated in healthy individuals. However, it can be life-threatening when severe. Mild to moderate hypokalemia increases the risk of mortality and morbidity in patients with cardiovascular disease. Drugs (diuretics) are the most common cause of hypokalemia [24]. It is often the cause of sudden death [25].

In spite of the presence of these ionic disturbances noted at admission in the patients, serum electrolytes control was carried out in only eight (08) patients of the study. The disturbances found were hyperchloremia (75%,  $n=8$ ), hyponatremia 25% ( $n=8$ ), hypoproteinemia in 28.57% ( $n=7$ ), hyperkalemia (12.50,  $n=8$ ) and a decrease in bicarbonate ions in 28.57% of patients ( $n=7$ ). The profile was therefore similar to that found at entry. However, real conclusions could not be drawn due to the lack of data. It would therefore be recommended in practice that the various biological controls be carried out, in order to ensure optimal management of the patients.

## 5. Conclusion

Accidental acute poisoning in children is a public health problem worldwide and particularly in Burkina Faso. At the end of the study, the frequency (2.43%) and mainly the mortality rate (16.06%) must be underlined. In addition, numerous biological complications, particularly ionic ones, which could compromise the vital prognosis of patients were found. Appropriate management of these disorders is essential for patient survival. Prospective studies on this issue would be of great help to draw better conclusions. On the other hand, the control of intoxication must be based on prevention.

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