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Electrolyte Disorders in Pulmonary Tuberculosis

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Abstract

Pulmonary tuberculosis remains a major cause of morbidity and mortality, particularly in high-endemic countries. The aim of our study was to analyze the clinical and paraclinical differences between tuberculosis patients presenting electrolyte disorders and those without such abnormalities. Identifying profiles of tuberculosis patients at risk of hydroelectrolytic disorders therefore represents an important clinical challenge. We conducted a retrospective descriptive and analytical study over three years, from June 1, 2021 to June 1, 2024, including 40 patients with pulmonary tuberculosis hospitalized in the pulmonology department of the 20 August Hospital in Casablanca. Patients were divided into two groups: Group A (with electrolyte disorders, n=20) and Group B (without disorders, n=20). Hyponatremia was the most frequent hydroelectrolytic disorder (42.5%). Hypocalcemia, hypokalemia, and hyperkalemia were each observed in 5% of cases. These abnormalities were significantly associated with low BMI, tachycardia, anemia, neutrophilia, lymphopenia, hypoproteinemia, and hypoalbuminemia ($p<0.01$, Fisher test). Moderate associations were observed for rhonchi and elevated transaminases ($p<0.05$). Other elements such as bronchial syndrome, vomiting, fever, lower limb edema, and the presence of miliary tuberculosis showed significance with the Chi-square test ($p<0.05$), but not with Fisher's test. Mean CRP, urea, GOT, and GPT levels were significantly higher in Group A according to Student's t-test. These results highlight the central role of systemic inflammation and the syndrome of inappropriate antidiuretic hormone secretion (SIADH) in the development of electrolyte imbalances, particularly hyponatremia. Adverse effects of anti-tuberculosis drugs (nephrotoxicity, hepatotoxicity, digestive disorders, and disturbances of vitamin D metabolism) may also contribute to their occurrence.

Keywords: Pulmonary tuberculosis, Electrolyte disorders, Hyponatremia, Inflammation, SIADH, Hypoalbuminemia, Risk profile

Introduction

Pulmonary tuberculosis remains the most frequent form of tuberculosis. Despite diagnostic and therapeutic advances, certain complications persist, particularly electrolyte disturbances whose etiology is complex and multifactorial. Often underdiagnosed, their investigation should particularly concern patients presenting severe forms, malnutrition, digestive symptoms, or disseminated involvement. Their early identification is essential for optimal management.

Case report:

Our study included 40 patients with pulmonary tuberculosis divided into two equal groups: Group A (n=20) presenting electrolyte disorders and Group B (n=20) without electrolyte disturbances. The mean

age in Group A was 43.5 years compared with 47.3 years in Group B. Patients aged between 15 and 40 years represented 50% of cases in Group A versus 30% in Group B (Figure 1).

Male predominance was observed in both groups, with a sex ratio of 2.3 in Group A compared with 1.5 in Group B. Active smoking was present in 13 patients in Group A and 12 patients in Group B. Alcohol consumption (8 cases), cannabis use (6 cases), and a previous confirmed history of tuberculosis (3 cases) were more frequent in Group A, whereas a history of pulmonary tuberculosis (3 cases) was more frequent in Group B. The main comorbidities observed in both groups were diabetes (3 cases) and heart failure (1 case), while hypertension was reported in two cases in Group B and one case in Group A. Clinically, dyspnea was the predominant symptom (15 cases in Group A versus 13 cases in Group B). Bronchial syndrome was more frequent in Group A (9 cases versus 3 cases in Group B). Digestive manifestations were exclusively reported in Group A, including vomiting (4 cases), diarrhea (2 cases), and abdominal pain (2 cases). General symptoms were frequent in both groups and were dominated by asthenia (16 cases in each group), associated with anorexia, weight loss, night sweats, and febrile sensations (Table 1).

On clinical examination, patients in Group A presented more frequently tachycardia (11 cases), low BMI below 18.5 kg/m² (10 cases), fever (8 cases), and oxygen saturation below 90% (5 cases). Some signs were observed only in this group, including hypotension (2 cases) and lower limb edema (4 cases). Pleuropulmonary findings included rhonchi (7 cases), parenchymal condensation syndrome (3 cases), and air effusion syndrome (5 cases), which were more frequent in Group A. In contrast, pleural effusion predominated in Group B (6 cases). Extrathoracic abnormalities such as neurological involvement, lymphadenopathy, ascites, purpura, or Cushing syndrome were almost exclusively observed in Group A. Imaging in Group A showed a predominance of miliary forms (8 cases), alveolo-interstitial forms (4 cases), and cavitary forms sometimes complicated by pneumothorax (3 cases), reflecting severe and disseminated involvement. In Group B, predominant abnormalities were interstitial and nodular forms (7 cases), pseudotumoral forms (3 cases), suggesting more localized disease. The most frequent extrapulmonary localizations were pleural and mediastinal in both groups, whereas meningeal, digestive, lymph node, or vertebral forms mainly concerned Group A. Biologically, hyponatremia was the most frequent electrolyte disorder, observed in 17 patients in Group A (85% of cases), most often moderate (130–135 mmol/L), with a mean sodium level of 130.5 mmol/L. Other electrolyte disorders were observed, including two cases of hypokalemia, two cases of hyperkalemia, one case of hypercalcemia, and two cases of hypocalcemia. Four patients presented combined electrolyte abnormalities (Table 2).

All patients received anti-tuberculosis treatment according to the National Tuberculosis Control Program [1]. Correction of hydroelectrolytic disorders was individually adapted. Three patients required intensive care due to severe hyponatremia and the associated risk of neurological complications. In addition, two patients developed acute renal failure during treatment. Analytical study based on the Chi-square test and confirmed by Fisher's test did not

show any significant association with age, sex, or past medical history. However, the occurrence of electrolyte disorders was significantly associated with malnutrition and systemic involvement, illustrated by low BMI ($p = 0.0034$), tachycardia ($p = 0.008$), hypoalbuminemia ($p = 0.0001$), and hypoproteinemia ($p = 0.0003$), as well as hematological abnormalities including anemia, neutrophilia, and lymphopenia ($p < 0.05$). Variables with intermediate significance included rhonchi and elevated GOT levels. Other manifestations such as bronchial syndrome, vomiting, fever, lower limb edema, and miliary form were significant only with the Chi-square test without confirmation by Fisher's test. These findings allowed the identification of a profile of tuberculosis patients particularly exposed to electrolyte disorders,

characterized by malnutrition (low BMI, hypoalbuminemia, hypoproteinemia), systemic involvement manifested by tachycardia, and hematological abnormalities, to which certain respiratory signs such as rhonchi or elements of clinical or radiological severity may be added (Table 3).

Discussion

Electrolyte disorders in pulmonary tuberculosis represent a relatively common and multifactorial complication. Hyponatremia is the most predominant disturbance, observed in 42.5% of cases in our series, a frequency comparable to that reported in several international studies [2-5]. In most cases, this hyponatremia is moderate and often reflects the severity of the inflammatory response associated with active tuberculosis. The underlying mechanism is frequently related to systemic inflammation leading to inappropriate secretion of antidiuretic hormone (SIADH), a phenomenon widely described in the literature. This mechanism promotes water retention disproportionate to sodium levels, resulting in dilutional hyponatremia [2-4]. In addition, inflammatory cytokines produced during active infection may further stimulate ADH secretion, thereby contributing to the persistence of hydroelectrolytic disturbances. Other factors may also contribute to hydroelectrolytic imbalance in patients with pulmonary tuberculosis. Digestive symptoms such as vomiting and diarrhea, excessive sweating related to febrile states, and poor nutritional status may all lead to significant electrolyte losses [5,6]. Malnutrition, which is frequently observed in tuberculosis, plays a particularly important role as it may alter both electrolyte homeostasis and the patient's overall metabolic balance. Potassium and calcium disturbances remain less frequent in comparison with sodium abnormalities. Hypokalemia may result from malnutrition, digestive losses, or adverse effects related to anti-tuberculosis therapy, whereas hyperkalemia is mainly observed in the context of acute renal injury or drug toxicity, particularly with rifampicin [7,8]. Hypercalcemia, although rare, is a well-described metabolic complication of granulomatous diseases and may occur through increased extrarenal production of calcitriol by activated macrophages within granulomas [3,9]. From a clinical perspective, several markers were significantly associated with the occurrence of electrolyte disorders in our series, including low BMI, fever, tachycardia, lower limb edema, and vomiting. These manifestations likely reflect a state of systemic inflammation combined with nutritional deficiency and hydro-sodium depletion [5,6,10]. Such findings suggest that electrolyte disturbances tend to occur in patients presenting more severe systemic involvement. Radiologically, the miliary form of tuberculosis was more frequently observed among patients with electrolyte disturbances ($p = 0.0285$). This finding may reflect hematogenous dissemination of the infection associated with a high inflammatory burden. Such systemic inflammation may favor the development of hyponatremia through SIADH or through involvement of other organs such as the meninges or adrenal glands, as reported in previous studies [11-13]. This observation supports the hypothesis that electrolyte disturbances may represent a marker of disease severity in disseminated tuberculosis. Other extrapulmonary localizations did not show statistically significant associations in our series. However, electrolyte disturbances have been described in several severe forms of extrapulmonary tuberculosis, particularly meningeal, lymph node, or pleural tuberculosis. In these situations, systemic inflammatory mechanisms or SIADH may also play an important role in the development of hydroelectrolytic imbalance [14-16]. Management of these disturbances relies primarily on careful correction of hyponatremia, generally using isotonic saline infusion, together with appropriate potassium supplementation in cases of hypokalemia. In cases of hypercalcemia, adequate hydration combined with bisphosphonate therapy may be required. These corrective measures must always be associated with appropriate etiological treatment of tuberculosis.

Finally, the potential toxicity of anti-tuberculosis drugs should also be considered in the occurrence or aggravation of electrolyte disturbances. Certain agents, particularly rifampicin, may induce renal or hepatic dysfunction, which may in turn worsen hydroelectrolytic imbalance [8,10,17]. Careful clinical and biological monitoring therefore remains essential during treatment.

Conclusion

Electrolyte disorders represent a significant complication of pulmonary tuberculosis, with hyponatremia being the most frequent manifestation. Their occurrence is often associated with marked inflammatory syndrome and severe clinical signs reflecting systemic involvement and sometimes hematogenous dissemination of infection. Some anti-tuberculosis drugs may also contribute to these imbalances. Identifying clinical, biological, and radiological risk profiles may allow earlier detection and more targeted management of tuberculosis patients presenting these abnormalities.

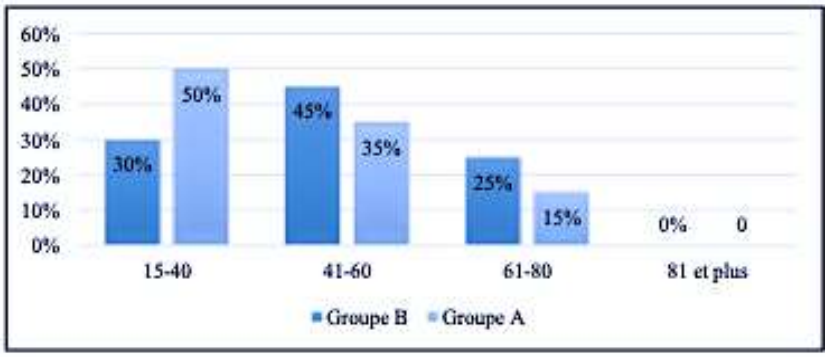

Figure 1: Distribution of patients according to age group

Signes fonctionnels		Groupe A	Groupe B
Signes fonctionnels respiratoires	Toux	9 (45%)	11 (55%)
	Dyspnée	15 (75%)	13 (65%)
	Syndrome bronchique	9 (45%)	3 (15%)
	Douleurs thoraciques	9 (45%)	12 (60%)
	Hémoptysie	3 (15%)	2 (10%)
Signes fonctionnels digestifs	Vomissements	4 (20%)	0 (0%)
	Diarrhées	2 (10%)	0 (0%)
	Douleurs abdominales	2 (10%)	0 (0%)
	Dégoût de l'eau	0 (0%)	0 (0%)
	Soif intense	0 (0%)	0 (0%)
Signes fonctionnels neurologiques	Céphalées	1 (5%)	0 (0%)
	Convulsions	1 (5%)	0 (0%)
	Fasciculations / crampes	0 (0%)	0 (0%)
	Paresthésies	0 (0%)	0 (0%)
	Tétanie	0 (0%)	0 (0%)
	Troubles de conscience	0 (0%)	0 (0%)
Signes généraux	Sensations fébriles	10 (50%)	7 (35%)
	Sueurs nocturnes	8 (40%)	5 (25%)
	Asthénie	16 (85%)	16 (80%)
	Anorexie	11 (55%)	13 (65%)
	Amaigrissement	11 (55%)	11 (55%)

Table 1: Distribution of functional symptoms in the two groups

Type de trouble HE		Nombre	% par rapport au Groupe A (n=20)	% par rapport à l'ensemble (Groupes A et B : n = 40)
Trouble de sodium	Hypernatrémie	0	0 %	0%
	Hyponatrémie	17	85%	42.5%
Trouble de potassium	Hyperkaliémie	2	10%	5%
	Hypokaliémie	2	10%	5%
Trouble de calcium	Hypercalcémie	1	5%	2.5%
	Hypocalcémie	2	10 %	5 %

Table 2: Distribution of electrolyte disorders according to type

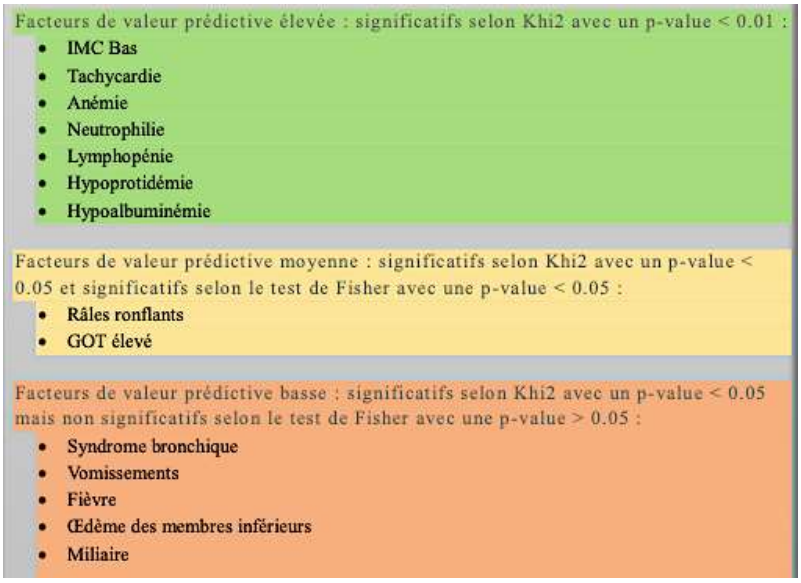

Table 3: Profile of tuberculosis patients at risk of developing hydroelectrolytic disorders combining clinical, biological, and radiological features according to the Chi-square test

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