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Malignant Hypercalcaemia Requiring Acute Haemodialysis: A Case Series of 36 Patients

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ABSTRACT

Introduction: Hypercalcaemia is a common metabolic disorder. Malignant hypercalcaemia is defined as a serum calcium level exceeding 3.5 mmol/L, associated with life-threatening cardiovascular or neurological consequences. Haemodialysis may be indicated when malignant hypercalcaemia fails to respond to first-line treatment. The aim of this study was to characterise the epidemiological, aetiological, therapeutic, and clinical outcomes of patients with malignant hypercalcaemia requiring haemodialysis.

Material and methods: This retrospective study was conducted over a 24-month period (January 2021–January 2023) and included patients who underwent emergency haemodialysis in the Nephrology Department of Ibn Rochd University Hospital, Casablanca, with malignant hypercalcaemia as the primary indication. Epidemiological, clinical, biochemical, and follow-up data were collected and analysed using SPSS Statistics version 20.

Results: Thirty-six patients were enrolled (mean age 55.08 ± 14.9 years; male-to-female ratio 2:1). Neoplastic disease was present in 80.6% of patients (solid tumours in 50%; haematological malignancies in 30.5%). All patients presented with altered general condition; dyspnoea was reported in 41.6% and neurological disorders in 27.8%. Physical examination revealed extracellular dehydration (91.7%), tachycardia (100%), and oligo-anuria (16.7%). Renal failure was documented in 72.2% and anaemia in 80.6% of patients. Mean corrected serum calcium was 169.16 ± 17.2 mg/L. Electrocardiographic signs of hypercalcaemia were present in all patients. All patients received one or more sessions of intermittent haemodialysis (mean 1.79 sessions per patient). Symptomatic management included intravenous fluid resuscitation, bisphosphonates (90%), loop diuretics after volume restoration (36.1%), and corticosteroids (22.2%). Serum calcium normalised in 26 patients (72.2%). Mortality at the end of the study period was 22.2%.

Conclusion: Malignant hypercalcaemia remains a potentially life-threatening condition owing to its cardiovascular, neurological, and renal complications. Malignant disease is the predominant aetiology. Haemodialysis constitutes a rapidly effective and potentially life-saving intervention in cases of hypercalcaemia with cardiac compromise.

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KEYWORDS:

malignant hypercalcaemia, haemodialysis, metabolic emergency, treatment.

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1. INTRODUCTION

Hypercalcaemia is a metabolic complication frequently encountered in nephrology, oncology, haematology, endocrinology, and critical care medicine. It is defined as a total serum calcium level exceeding 2.6 mmol/L (104 mg/L), or an ionised calcium level exceeding 1.3 mmol/L (52 mg/L). Clinical manifestations include systemic, neurological,

cardiovascular, and renal features; the severity of hypercalcaemia correlates primarily with the rapidity of its onset [1]. Malignant hypercalcaemia — defined as a serum calcium level exceeding 3.5 mmol/L (140 mg/L) with cardiovascular or neurological impairment — constitutes a metabolic emergency requiring urgent and appropriate management.

The aetiology of hypercalcaemia is wide-ranging; however, primary hyperparathyroidism, bone metastases from solid tumours, and haematological malignancies collectively account for approximately 90% of all cases [2].

The management of hypercalcaemia encompasses both aetiological and symptomatic approaches, the latter including intravenous hydration, loop diuretics, and bisphosphonates. Extracorporeal renal replacement therapy may be indicated in cases of malignant hypercalcaemia refractory to first-line treatment.

The objective of this study was to describe the epidemiological, aetiological, therapeutic, and outcome profiles of patients with malignant hypercalcaemia requiring haemodialysis.

2. MATERIAL AND METHODS

This retrospective cohort study was conducted over a 24-month period (January 2021 to January 2023) in the Department of Nephrology, Dialysis and Renal Transplantation at Ibn Rochd University Hospital, Casablanca. It included all patients who underwent emergency haemodialysis with malignant hypercalcaemia as the primary indication.

Serum calcium was measured using the Arsenazo III colorimetric method. Total calcium was corrected using the following formula: measured calcium (mg/L) + [40 − serum albumin (g/L)]. Parathyroid hormone (PTHi) and vitamin D levels were measured by chemiluminescence on an Abbott Alinity analyser.

Epidemiological and clinical data were collected from all patients. Follow-up data were obtained from medical records in the respective inpatient units, and biochemical data were retrieved from the Kalisil laboratory information system of the Ibn Rochd University Hospital Biochemistry Laboratory. Statistical analysis was performed using SPSS Statistics version 20. Continuous variables are expressed as means with standard deviations; categorical variables are expressed as frequencies and percentages.

3. RESULTS

During the study period, 36 patients were enrolled. The mean age was 55.08 ± 14.9 years (range: 18–75 years). There was a predominance of male patients, with a male-to-female sex ratio of 2:1.

Regarding past medical history, 38.9% of patients were smokers, 11.1% had diabetes mellitus, and 80.6% had a known oncological diagnosis. Of these, 18 patients (50%) had solid tumours and 11 (30.5%) had haematological malignancies. The two most common solid tumour sites were lung (19.4%) and breast (11.1%). Bone metastases were present in 44.4% of patients. Multiple myeloma was the predominant haematological malignancy identified in this series. Seven patients had relapsed disease, while four had a new diagnosis of multiple myeloma, with hypercalcaemia detected at the time of initial diagnosis (Table I).

With respect to admitting department, the emergency resuscitation unit (ICU) was the most frequent setting (36.1%), followed by oncology (30.5%) and haematology (19.4%) (Table II).

The main presenting symptoms included altered general condition in all patients, dyspnoea in 41.6%, and neurological disorders in 27.8%. Initial physical examination revealed extracellular dehydration in 91.7% of patients, tachycardia in all patients, and oligo-anuria in 16.7% (Figure 1).

Laboratory investigations demonstrated associated renal failure in 72.2% of cases. Mean corrected serum calcium was 169.16 ± 17.2 mg/L. Anaemia was present in 80.6% of patients. PTHi and vitamin D levels were available in only seven patients; elevated PTHi was found in four, consistent with primary hyperparathyroidism (Table III).

All patients exhibited electrocardiographic signs of hypercalcaemia, including sinus tachycardia (100%; mean heart rate 120 bpm), shortened corrected QT interval (100%), ST-segment changes (cupola pattern) (30.6%), J-point elevation (11%), and cardiac arrhythmias (16.8%).

All patients underwent one or more sessions of intermittent haemodialysis, with a mean of 1.79 sessions per patient. Vascular access was achieved via a femoral catheter in 52.8% of cases and a jugular catheter in 47.2%. All sessions were completed without significant adverse events, with no episodes of severe hypotension, cardiac arrhythmia, or neurological deterioration. Haemodialysis was accompanied by intravenous fluid resuscitation with 1,000–1,500 mL of isotonic saline (0.9%).

Symptomatic treatment, in addition to extracorporeal renal replacement therapy, included intravenous rehydration (all patients), bisphosphonates (90%), loop diuretics following volume restoration (36.1%), and corticosteroids (22.2%).

Aetiological treatment was also initiated according to the underlying aetiology of hypercalcaemia (chemotherapy, surgery, hormonal therapy, or parathyroidectomy). Palliative care was instituted in 19.4% of patients.

Outcomes: Serum calcium normalised in 26 patients (72%). Mortality during the study period was 22.2%.

4. DISCUSSION

Calcium plays a pivotal role in numerous intracellular and extracellular biological processes [3]. Calcium homeostasis is regulated by three principal hormones — parathyroid hormone (PTH), 1,25-dihydroxyvitamin D, and calcitonin — acting at the level of the kidney, bone, and gastrointestinal tract [4]. Hypercalcaemia may arise from increased bone resorption, enhanced intestinal calcium absorption, or

decreased renal calcium excretion [2].

Normal serum calcium ranges from 2.2 to 2.6 mmol/L. Hypercalcaemia is classified as mild (2.63–3.0 mmol/L), moderate (3.0–3.5 mmol/L), or severe (>3.5 mmol/L), the latter carrying a risk of life-threatening complications [2]. Approximately 45% of circulating calcium is protein-bound (primarily to albumin), 10% is complexed with anions, and 45% circulates as free ionised calcium (reference range: 1.17–1.33 mmol/L) [1]. Ionised calcium measurement is considered more sensitive and specific for diagnosing hypercalcaemia than total calcium [5]; however, this assay was not available in our cohort.

Primary hyperparathyroidism and malignancy together account for approximately 80% of all cases of hypercalcaemia [2]. Granulomatous diseases and drug-induced hypercalcaemia account for a further 15% [5,6]. The main causative drugs include thiazide diuretics, lithium, vitamin D, and vitamin A [5]; up to 8% of patients receiving thiazide diuretics develop hypercalcaemia [7]. Thyroid dysfunction and prolonged immobilisation may also contribute [8].

The prevalence of primary hyperparathyroidism — the leading single aetiology — is estimated at 1–7 cases per 1,000 individuals, with a two- to three-fold female predominance, predominantly affecting those older than 65 years [9]. It typically presents with mild, chronic hypercalcaemia, though severe forms are recognised [10]. In our series, primary hyperparathyroidism was identified as the cause of hypercalcaemia in 11.1% of patients.

Hypercalcaemia of malignancy is common among cancer patients, with an incidence reaching up to 44.1%, primarily in advanced-stage disease, and is associated with poor overall survival [11]. The underlying pathophysiological mechanisms are varied: PTH-related protein (PTHrP) secretion in a paraneoplastic setting — observed in breast cancer, ovarian cancer, urological malignancies, and non-Hodgkin lymphoma — promotes bone resorption and tubular calcium reabsorption [12]. Osteolytic bone metastases, particularly in breast cancer and multiple myeloma, liberate skeletal calcium stores [11]. Ectopic 1-alpha-hydroxylase activity occurs in certain lymphomas and ovarian tumours [12], while ectopic PTH secretion has been described in small-cell lung carcinoma and adenocarcinomas [11].

Several studies have demonstrated that patients with underlying malignancy present with higher serum calcium levels and are therefore more frequently symptomatic; serum calcium levels above 3.25 mmol/L are typically observed in this context [11,13,14]. The occurrence of severe, symptomatic hypercalcaemia should prompt thorough investigation for an underlying malignancy [1]. In our series, 80% of patients had a malignant aetiology, consistent with published literature [13].

The overall prevalence of hypercalcaemia is likely underestimated due to the non-specificity of its clinical presentation [15,16]. A prevalence of approximately 1% has been reported in the general population, rising to around 3% in hospitalised patients [5]. A Swiss study evaluating 77,847 emergency department admissions identified a hypercalcaemia prevalence of 0.7%, of which only 26% were symptomatic [13].

The severity of the clinical presentation correlates with the degree and rapidity of onset of hypercalcaemia. Mild-to-moderate hypercalcaemia is frequently asymptomatic [1]. Severe hypercalcaemia manifests with systemic features (anorexia, asthenia, fever, deterioration of general condition), neurological complications (confusion, obtundation, or coma), cardiovascular abnormalities (hypertension, cardiovascular collapse, QT interval shortening, arrhythmias), and renal complications (acute kidney injury, polyuria, hypokalaemia) [5,6,13]. In the most severe cases, vital prognosis may be compromised within hours or even minutes owing to the risk of cardiac arrest [6]. All patients in our series exhibited electrocardiographic evidence of hypercalcaemia; sinus tachycardia and QT shortening were universal, and arrhythmias were present in 16.8%.

Acute kidney injury (AKI) is a frequent complication of severe hypercalcaemia, most commonly of a pre-renal (functional) nature, secondary to the hypovolaemia induced by hypercalcaemia-related polyuria and hypercalciuria. Intrinsic renal injury, including acute tubular necrosis due to nephrotoxic agents, sepsis, or direct tubular toxicity (as in myeloma cast nephropathy), may also occur [17].

Management of hypercalcaemia encompasses both symptomatic and aetiological strategies, calibrated to the severity of the clinical presentation. Intravenous rehydration with isotonic saline (200–300 mL/hour) is the cornerstone of symptomatic treatment, correcting the frequent dehydration resulting from vomiting, polyuria, and impaired fluid intake. Careful monitoring for volume overload is essential, particularly in patients with cardiac failure or advanced renal insufficiency [6].

Loop diuretics enhance renal calcium excretion but may exacerbate pre-existing hypovolaemia and should therefore be reserved for use after adequate volume replacement. A systematic review conducted in 2009 found insufficient evidence to support the routine use of furosemide in hypercalcaemia management; its use should be limited to the treatment of volume overload [18,19].

Calcitonin (4–8 IU/kg every 6–12 hours for 48 hours) inhibits bone resorption and renal calcium reabsorption, with a rapid but transient effect [11]. It was not administered in our series owing to non-availability.

Bisphosphonates are the mainstay of treatment for malignant hypercalcaemia. Zoledronic acid and pamidronate, the second-generation bisphosphonates, are the most efficacious agents in this indication; their onset of action is 2–4 days post-administration. They act by inhibiting osteoclast-mediated bone resorption; zoledronic acid has demonstrated superior efficacy and duration of action compared to pamidronate in clinical trials [1]. Pamidronate is preferred in patients with

renal impairment. In our series, bisphosphonates were administered to 90% of patients.

Denosumab, a fully human monoclonal antibody targeting the RANK ligand (RANKL), may be indicated in bisphosphonate-refractory hypercalcaemia [20]. By binding RANKL, denosumab inhibits osteoclast formation, function, and survival.

Corticosteroids are indicated in hypercalcaemia associated with granulomatous diseases, certain lymphomas, and vitamin D toxicity [8], acting by reducing intestinal calcium absorption through inhibition of 1-alpha-hydroxylase [16].

Surgical parathyroidectomy is the definitive treatment for primary hyperparathyroidism complicated by hypercalcaemia. Cinacalcet, a calcimimetic agent that reduces PTH secretion, may be used as a bridge or in cases where surgery is contraindicated.

Haemodialysis may be indicated for malignant hypercalcaemia refractory to conventional therapy, particularly in patients with severe renal impairment or advanced cardiac dysfunction precluding aggressive intravenous hydration, or in the context of developing fluid overload [11]. There is no absolute serum calcium threshold mandating extracorporeal therapy; however, the indication becomes more urgent when hypercalcaemia develops rapidly, when cardiac or neurological compromise is severe and life-threatening, or when short-term renal prognosis is poor [6]. The use of a low-calcium dialysate is recommended. Haemodialysis enables rapid correction of serum calcium and concurrent management of associated fluid and electrolyte disturbances.

A Tunisian multicentre study evaluating 188 patients undergoing emergency haemodialysis found that malignant hypercalcaemia accounted for 2% of all emergency dialysis indications [21]. A Moroccan study by Bentata et al. from Oujda University Hospital reported a marked increase in the incidence of severe hypercalcaemia requiring haemodialysis during the COVID-19 pandemic (62.2% vs. 37.8% in the pre-pandemic period), attributed to delayed diagnosis and management of oncological patients. Malignant aetiologies were identified in 80% of cases. Haemodialysis was rapidly effective in lowering serum calcium, and mortality was approximately 14%, though deaths were not directly attributable to hypercalcaemia but occurred after calcium normalisation [17]. A French multicentre study by Mousseaux et al. evaluating 131 cases of severe hypercalcaemia (defined as calcium >120 mg/L) documented haematological malignancies in 44.3% and solid tumours in 22.1% of cases. Associated AKI was present in 82.4%, of whom 19% underwent haemodialysis. Overall mortality was 21% and was significantly associated with underlying malignancy [22].

The importance of palliative care and psychosocial support for oncological patients presenting with hypercalcaemia and their families warrants particular emphasis [11].

The principal limitations of the present study are its retrospective design and the incomplete availability of PTHi and vitamin D data for the majority of patients. Nevertheless, our findings demonstrate a high prevalence of malignant disease among patients with severe hypercalcaemia requiring haemodialysis and confirm the efficacy of haemodialysis in rapidly normalising serum calcium. Multidisciplinary collaboration between nephrologists, oncologists, haematologists, and intensivists is essential for optimal management.

5. CONCLUSION

Malignant hypercalcaemia remains a potentially life-threatening condition due to its neurological, cardiovascular, and renal complications. Malignant disease constitutes the predominant aetiology. In our cohort, an increase in the incidence of malignant hypercalcaemia was observed during the COVID-19 pandemic, likely reflecting delayed diagnosis and management of oncological patients. Haemodialysis represents a life-saving intervention in hypercalcaemia with cardiac compromise, by virtue of its ability to rapidly lower serum calcium. Both aetiological and symptomatic treatment must be instituted concurrently.

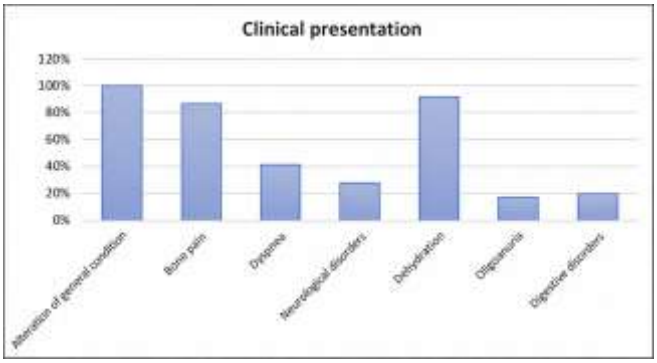

Figure 1. Distribution of main clinical signs observed in the study cohort.

Table I. Main past medical history of patients in the study cohort.

Medical History	n	Percentage
Smoking	14	38.9%
Diabetes mellitus	4	11.1%
Hypertension	5	13.9%

Medical History	n	Percentage
Neoplasia (total)	29	80.6%
Solid tumours (total)	18	50.0%
Lung	7	19.4%
Breast	4	11.1%
Endometrium	2	5.5%
Cervix	1	2.7%
Nasopharynx	1	2.7%
Penis	1	2.7%
Bladder	1	2.7%
Ovary	1	2.7%
Haematological malignancies (total)	11	30.5%
Multiple myeloma (relapsed)	7	19.4%
Multiple myeloma (new diagnosis)	4	11.1%

Table II. Distribution of patients by admitting department.

Admitting Department	n	Percentage
Emergency Resuscitation Unit (ICU)	13	36.1%
Oncology	11	30.5%
Haematology	7	19.4%
Traumatology	2	5.5%
Pulmonology	1	2.7%

Table III. Summary of main biochemical parameters at admission.

Biochemical Parameter	Mean ± SD
Corrected serum calcium (mg/L)	169.16 ± 17.2
Serum albumin (g/L)	29.21 ± 6.24
Total serum protein (g/L)	70.67 ± 22.86
Serum creatinine (mg/L)	29.94 ± 19.8
Serum sodium (mmol/L)	138.6 ± 5.5
Serum potassium (mmol/L)	3.8 ± 0.6
C-reactive protein (mg/L)	119.67 ± 81.63
Serum phosphate (mg/L)	45.38 ± 22.6
Haemoglobin (g/dL)	9.03 ± 2.5
Intact PTH (pg/mL)	293.67 ± 694.88

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