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CASE REPORT

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Small cell lung cancer case report: acute tumor lysis syndrome after chemotherapy and management strategies for high-risk patients

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Abstract

Background In the treatment of small cell lung cancer (SCLC), acute tumor lysis syndrome (ATLS) is one of the oncologic emergencies that requires particular attention. Previous studies have generally indicated that ATLS increases mortality risk during treatm ent. Therefore, early identification of ATLS, along with proactive prevention and symptomatic management, is particularly crucial.

Methods In this report, we detail the clinical management of a patient with SCLC and multiple metastases who was identified as being at relatively high risk for ATLS due to a large tumor burden and concurrent liver and kidney dysfunction.

Results Despite rapid tumor progression, the treatment team implemented aggressive hydration and urine alkalinization as pretreatment measures and personalized dose-reduced chemotherapy based on the standard EC regimen. Nevertheless, the patient developed ATLS, which progressed rapidly, and despite intensive treatment, the condition remained irreversible.

Conclusion This case highlights that in some SCLC patients, pre-chemotherapy evaluation reveals a higher risk for tumor lysis syndrome, and adjusting treatment strategies for these patients requires further investigation. This suggests that managing such high-risk patients in clinical practice requires more cases and optimized treatment strategies to guide management. Therefore, this case is presented to offer insights into this perspective.

Clinical trial number Not applicable.

Keywords Small cell lung cancer, Tumor Lysis syndrome, Acute renal failure, Hyperuricemia

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Introduction

ATLS is a clinical emergency caused by the rapid release of intracellular contents following the death of tumor cells [1]. It is characterized by hyperuricemia, hyperphosphatemia, hyperkalemia, and hypocalcemia [2]. It most commonly occurs during the treatment of malignant tumors [3], typically develops 48–72 h after chemotherapy, marked by severe electrolyte and pH imbalances, as well as difficult-to-manage renal failure. In this article, we describe the clinical management of a patient with SCLC and multiple metastases who developed ATLS following chemotherapy. We highlight the key features of the patient's condition and review the relevant literature, aiming to provide clinical insights into the diagnosis and treatment of similar cases.

Case presentation

A 74-year-old male patient, newly diagnosed with stage IV SCLC with multiple metastases, presented with a two-month history of anorexia and weight loss. Abdominal MRI revealed hepatomegaly with innumerable hepatic masses. PET-CT further confirmed extensive liver involvement with increased FDG uptake, along with enlarged hilar and mediastinal lymph nodes, thickened gastric antrum, and localized uptake in the right iliac bone, T4 and L5 vertebrae, and sternum. Serum NSE levels were significantly elevated, suggesting a neuroendocrine tumor originating from the lung with extensive metastasis. Enhanced MRI did not indicate brain metastases.A pre-chemotherapy liver biopsy confirmed metastatic SCLC. Immunohistochemical staining showed positivity for CK, CD56, and synaptophysin, with a high Ki-67 index (90%) and TTF-1 positivity. The diagnosis was stage IVB (T1N3M1c) SCLC, with multiple liver and bone metastases, as well as hepatic and renal insufficiency.

Before chemotherapy, the patient presented with anorexia, decreased intake, and marked fatigue, with an ECOG performance status score of 1. Physical examination revealed mild scleral icterus, a firm but non-tender liver edge, and no peripheral edema. Following multidisciplinary team (MDT) discussions, it was determined that the patient's tumor was progressing rapidly and required timely therapeutic intervention. However, given the large tumor burden, rapid progression, and concomitant hepatic and renal dysfunction, the patient was considered at high risk for ATLS. It was recommended to implement aggressive ATLS prevention before proceeding with personalized dose-reduced antitumor therapy.

Before chemotherapy, tumor markers NSE and CYFRA 21 – 1 were elevated, along with liver function impairment (ALT 280 U/L, AST 207 U/L, total bilirubin 43.4 μmol/L). Renal function was also compromised (creatinine 161 μmol/L, eGFR 36 mL/min/1.73 m^2)(Table 1). After aggressive liver protection and supportive treatment to improve liver function, the patient's hepatic and renal functions showed some improvement. Consequently, the patient began personalized dose-reduced EC chemotherapy. Approximately 1500 mL of hydration and urine alkalinization were administered the day before chemotherapy. On the second day, etoposide 0.1 g (days 1–3) and carboplatin 150 mg (day 1) were administered. During the first 24 h post-chemotherapy, the patient experienced no significant discomfort, but at approximately 48 h after chemotherapy, the patient developed pronounced abdominal distension and fatigue, accompanied by abdominal pain (NRS score 4). Liver function worsened (ALT 240 U/L, AST 269 U/L, total bilirubin 180.5 μmol/L), and renal dysfunction persisted (creatinine 161 μmol/L, eGFR 36 mL/min/1.73 m^2).

Considering the patient's high risk for ATLS, aggressive hydration and close monitoring of electrolytes were performed, revealing hyperkalemia (potassium increased by 25% from baseline or ≥ 6.0 mmol/L) and hyperuricemia (uric acid increased ≥ 8.0 mg/dL from baseline). The patient's renal colic symptoms progressively worsened, despite intensive monitoring, diuretics,

Table 1 Laboratory values on admission

Description	Results	Units	Reference range
ALT	280	IU/L	0 - 45
AST	207	IU/L	0 - 41
Total bilirubin	43.4	umol/L	3.4-20.5
Creatinine	161	mmol/L	57-111
eGFR	36	mL/min/1.73 m^2	90-120
LDH	185	IU/L	60 - 200
Potassium	4	mmol/L	3.5 - 5

Table 2 SCLC in TLS

Year	Author	Age/Gender	Tumor staging	Site of metastasis	Treatment	Onset of TLS	Out come of TLS
1983	Nicholas J	57 years/female	Extensive-stage	Liver, Bone, adrenal, and lymph nodes	Chemotherapy	36h	Died
1990	Atif M	57years/male	Extensive-stage	Multiple sites of involvement *	Cyclophosphamide, doxorubicin, and vincristine	4 days	Resolved
1999	S. L.Vanhees	62 years/male	Limited-Stage	Non	Vindesine, ifosfamide and cisplatin	24 h	Resolved
1997	Gregory P	74 years/female	Extensive-stage	Bone	Cisplatin plus etoposide	72 h	Resolved
2000	S. L.Vanhees	62years/male	Limited-Stage	Non	Vindesine, ifosfamide and cisplatin	24 h	Resolved
2002	HASSANAL I H	55years/male	Extensive-stage	Liver	Etoposide, carboplatin, and a matrix metalloproteinase inhibitor (BAY 12-9566)	24 h	Died
2011	Bassel Jallad	75 years/female	Extensive-stage	Liver	Chemotherapy-naïve patients	/	Died
2013	Sosipatros A	74 years/female	Limited-Stage	Non	Cisplatin plus etoposide	7 days	Resolved
2015	Chanudi Weerasinghe	65years/male	Extensive-stage	Liver, bone, and lymph nodes	Chemotherapy-naïve patients	/	Resolved
2017	Venkatkiran Kanchustam bham	53years/male	Limited-Stage	Non	Chemotherapy-naïve patients	/	Resolved
2017	Boonphipho p Boonpheng	55years/female	Extensive-stage	Liver and Lymph nodes	Chemotherapy-naïve patients	/	Dialysis dependent
2017	Fasihul Khan	64years/male	Extensive-stage	Liver , left adrenal and Lymph nodes	Steroid therapy	3 days	Died
2018	Prajwal Dhakal	70years/male	Extensive-stage	Liver	Chemotherapy-naïve patients	/	Died
2019	Phyo Thazin Myint	66years/female	Extensive-stage	Liver	Chemotherapy-naïve patients	/	Died
2020	Aydan Mutis Alan	59years/male	Extensive-stage	Liver	Chemotherapy-naïve patients	/	Died
2021	Sarah Maryon Hayes	86years/female	Extensive-stage	Liver, bone, and lymph nodes	Nivolumab	7 days	Died
2023	Simran Koura	67years/female	Extensive-stage	Liver	Chemotherapy-naïve patients	/	Died

* Subcutaneous tissue, the adrenals, and left retrobulbar area, and diffuse cervical.thoracic, abdominal, and pelvic adenopathy.

and uric acid-lowering agents. The patient rapidly developed lethargy, deep breathing, and signs of renal failure. Laboratory tests indicated severe infection (PCT 41.54 mg/mL, CRP 65.17 mg/L). At approximately 72 h post-chemotherapy, the patient passed away due to electrolyte imbalances caused by acute and rapid tumor lysis, despite intensive treatment.

Discussion

SCLC patients are generally considered to be at low risk for ATLS among the overall population, but within solid tumors, SCLC still represents a high-risk group for developing ATLS. The diagnosis of ATLS depends on clinical manifestations and laboratory test results [3]. In this case, the patient was diagnosed with widely metastatic malignancy at initial presentation. Given the high risk of ATLS, a personalized EC chemotherapy regimen was initiated one week after diagnosis. However, despite prophylactic measures, ATLS developed within 48 h of treatment initiation. Despite intensive supportive care, the patient ultimately succumbed to disease progression. This case underscores the critical need for heightened clinical vigilance and highlights the urgency of developing effective preventive strategies for rapid-onset ATLS in SCLC.

To better characterize this rare oncologic emergency, we conducted a systematic review of TLS in SCLC over the past five decades, identifying 17 reported cases (9 males, 8 females). TLS onset ranged from 24 h to 7 days, with patient ages between 53 and 86 years. Notably, 8 cases were spontaneous TLS, underscoring its potential risk in treatment-naïve patients. One rare case was triggered by corticosteroid administration, warranting further attention. Despite aggressive treatment, 9 of 17 patients succumbed shortly after TLS onset, highlighting its rapid progression and high mortality (Table 2) [4–19].

Identifying high-risk populations remains a clinical priority, as extensive hepatic and bone metastases have been linked to increased TLS risk. However, its occurrence in limited-stage disease suggests additional contributing factors, emphasizing the need for further investigation to refine risk stratification and early intervention strategies. Advanced age is another critical risk factor for this oncologic emergency. In our case, the 74-year-old patient belonged to a high-risk group. Based on previous reports, elderly patients frequently exhibit significant renal dysfunction and metabolic derangements, which may contribute to disease progression and poorer clinical outcomes [20–22].

Currently, the main goals of ATLS prevention are to ensure adequate urine output and to reduce the blood concentrations of uric acid, potassium, and phosphate [23–25]. The primary preventive strategies include intravenous hydration and uric acid-lowering agents, such as allopurinol, rasburicase, and febuxostat [26, 27]. Theoretically, after such interventions, aggressive intravenous hydration can improve outcomes for high-risk patients by enhancing renal perfusion, glomerular filtration, and urine output [28]. Renal replacement therapy may improve treatment outcomes. However, despite these aggressive preventive and symptomatic interventions, the patient still experienced rapid and irreversible ATLS. This indicates the need for more personalized preventive strategies, taking into account the patient's overall health status, tumor type, and treatment plan. Furthermore, educating patients and healthcare professionals to recognize ATLS risk is essential for effective prevention. Some literature also suggests that dialysis may improve outcomes in severe ATLS cases, although the effectiveness of this intervention requires further evaluation in this context.

Conclusion

In summary, patients with SCLC who, despite undergoing pre-treatment and precise risk evaluation, still develop ATLS represent a group that requires further attention and optimization of treatment strategies in clinical practice. This case highlights the complexity and challenges in managing ATLS in SCLC patients with high tumor burden and high proliferation following first-line chemotherapy. It serves as a reminder for clinicians about the importance of early recognition, aggressive intervention, and personalized preventive strategies tailored for high-risk patients, though such measures may not necessarily improve clinical outcomes in cases of ATLS.

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None.

Author contributions
Ying Han, Zuguo Yuan and Peng Yue wrote the main manuscript text. All authors reviewed the manuscript.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethical approval
Approval was obtained from the ethics committee of the People's Hospital Affiliated to Ningbo University. The procedures used in this study adhere to the tenets of the Declaration of Helsinki.

Consent to publish
Not applicable.

Consent to participate
Not applicable.

Competing interests
The authors declare no competing interests.

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