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REVIEW

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Acute tumor lysis syndrome in solid tumors – a case report and review of the literature

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Abstract *Purpose*: Tumor lysis syndrome (TLS) is a potential complication in cancer therapy. It may occur in highly sensitive tumors, especially in childhood cancers and acute leukemias, whereas it is rare in the treatment of adult solid tumors. TLS is characterized by hyperuricemia, hyperkalemia, hyperphosphatemia and hypocalcemia following massive lysis of malignant cells. Complications include acute renal failure and metabolic acidosis. We report the first case of TLS during chemotherapy in a patient with metastatic medulloblastoma, together with a review of the literature regarding the occurrence of TLS in patients with solid tumors. *Methods*: Data regarding clinical and biochemical parameters were extracted from the actual patients' files. Reports of TLS in the English language literature up to 2002 were identified by searching Medline. *Results*: A 23-year old male with metastatic medulloblastoma received chemotherapy with cisplatin and etoposide due to massive extracerebral manifestations including metastases to the liver, mediastinal lymph nodes and bone marrow metastases. The patient developed classical signs of TLS on the second day of chemotherapy, including acute renal failure. A 17-fold increase in plasma LDH up to 87608 U/l was observed together with a 4-fold increase in plasma creatinine. The patient was treated with aggressive hydration, allopurinol and repeated hemodialysis. During the following days the patient improved and the biochemical markers all returned to normal. *Review*: Reviewing the literature, a total of 45 patients with solid tumors who developed TLS have been reported. Most of the patients presented with metastatic, therapy-sensitive disease. Although preventable in practically 100% of patients, TLS is a potentially fatal complication, and in this material the mortality rate was one in three. Risk factors included increased LDH, hyperuricemia and pretreatment azotemia. *Conclusions*: TLS is only rarely associated with treatment of solid tumors. Precautions should be taken to avoid this potentially fatal complication in (chemo)therapy of solid tumors, especially in therapy-sensitive tumors presenting with bulky, metastatic disease and preexisting risk factors, including azotemia, elevated LDH and hyperuricemia. Prophylactic treatment to avoid TLS includes allopurinol, hydration prior to treatment and alkalization of the urine. Urate oxidase (rasburicase) is now beginning to replace allopurinol as a more effective way of reducing hyperuricemia and thereby the risk of TLS.

Keywords Tumor lysis syndrome · Medulloblastoma

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Introduction

Tumor lysis syndrome (TLS) is characterized by the release of intracellular components into the bloodstream due to massive lysis of malignant cells, spontaneously or following antineoplastic therapy [1, 2, 3]. The syndrome is characterized by hyperuricemia, hyperkalemia, hyperphosphatemia and hypocalcemia. Hyperuricemia is caused by purine degradation and may lead to precipitation of uric acid crystals in the collecting tubules in the kidney, resulting in obstructive nephropathy. Hyperkalemia, following potassium effusion from the cytoplasm, may lead to cardiac arrhythmias and cardiac arrest. Hyperphosphatemia, following nucleoprotein degradation, may cause precipitation of calcium phosphate in the renal tubules aggravating the acute renal failure. Hypocalcemia follows the precipitation of calcium phosphate in the tissues and may cause neuromuscular irritability including tetany, convulsions and changes in mental behavior. Development of acidosis may also be associated with the syndrome. Potassium, uric acid and phosphate are primarily excreted by renal clearance, and development of renal failure will further aggravate the electrolyte derangement.

Case story

A 23-year-old male presented with initial symptoms comprising dizziness and headache. A computed tomography (CT) scan of the cerebrum showed tumor in the fourth ventricle. An incomplete resection of the tumor was done, and histological examination showed medulloblastoma WHO grade IV. A magnetic resonance (MR) scan of the cerebrum and the spinal cord following the operation showed involvement of the spinal cord with meningeal lesions, pointing to carcinomatosis. Radiotherapy was given following the operation against the cerebrum, posterior fossa and spinal cord. Subsequently, MR scan revealed complete remission with no tumor manifestations from the central nervous system. Six months later the patient presented with malaise, diffuse abdominal pain, nausea and anorexia. A relapse was demonstrated with massive extracerebral manifestations of the medulloblastoma including metastases to the liver, mediastinal lymph nodes and bone marrow carcinosis. Subsequently, the patient received a chemotherapy regimen including cisplatin 20 mg/m^2 per day and etoposide 50 mg/m^2 per day, both administered daily for 5 days every 3 weeks.

On admission, levels of hemoglobin, plasma sodium, plasma potassium and plasma creatinine were all within normal limits. There were elevated levels of plasma bilirubin, plasma alkaline phosphatase and plasma aspartic aminotransferase (p-AST). Chemotherapy was administered as planned for the first 2 days without immediate complications. On the 2nd day the patient complained of fatigue and difficulty in breathing, and a decrease in urinary output was noted. The patient was treated with infusions of normal saline and high doses of diuretics without effect on the urine volume, while the biochemical markers showed signs of hemodilution with hemoglobin levels declining to 5.1 mmol/l (normal range 8.0–11.0 mmol/l) and plasma sodium to 106 mmol/l (136–146 mmol/l). Simultaneously, the body weight increased from 62 kg to 74 kg, demonstrating a fluid overload. The patient developed hyperkalemia and rising azotemia parameters with plasma creatinine 0.330 mmol/l (normal range 0.060–0.130 mmol/l), plasma carbamide 22.6 mmol/l (2.5–7.5 mmol/l) and plasma potassium 6.5 mmol/l (3.2–4.7 mmol/l), and oliguria with a 24-h urinary output less than 200 ml. TLS was further indicated by the following laboratory results: plasma ionized calcium 0.69 mmol/l (normal range 1.15–1.35 mmol/l), plasma phosphate 3.53 mmol/l (0.80–1.50 mmol/l) and plasma urate 2.03 mmol/l (0.20–0.45 mmol/l; see Fig. 1).

Together with cardiac monitoring the patient was treated with intravenous glucose, insulin and oral polystyrensulfonate (Resonium) but with an unsatisfactory effect on the increasing potassium level. On day 3 ultrasonic examination of the abdomen showed no sign of hydronephrosis. Hemodialysis was initiated on day 3 due to progressing renal failure and hyperkalemia and was repeated on day 4. The patient received intravenous calcium and also allopurinol 300 mg orally once daily from day 5. During the following days the patient improved and the biochemical markers returned to normal levels. The plasma level of lactate dehydrogenase (LDH) was 4792 U/l before chemotherapy, rising to 87608 U/l on day 5, declining to 7869 U/l on day 15 and further to 542 U/l on day 46 (normal range 150–450 U/l), indicating at first heavy cell lysis and subsequently decreased tumor burden.

Following the patient's clinical and biochemical recovery, three additional series of chemotherapy were given, during which no sign of TLS was observed. A CT scan after these four treatment courses showed partial remission with regression of the tumor manifestations in the liver and lungs and normalization of the biochemical parameters of the liver.

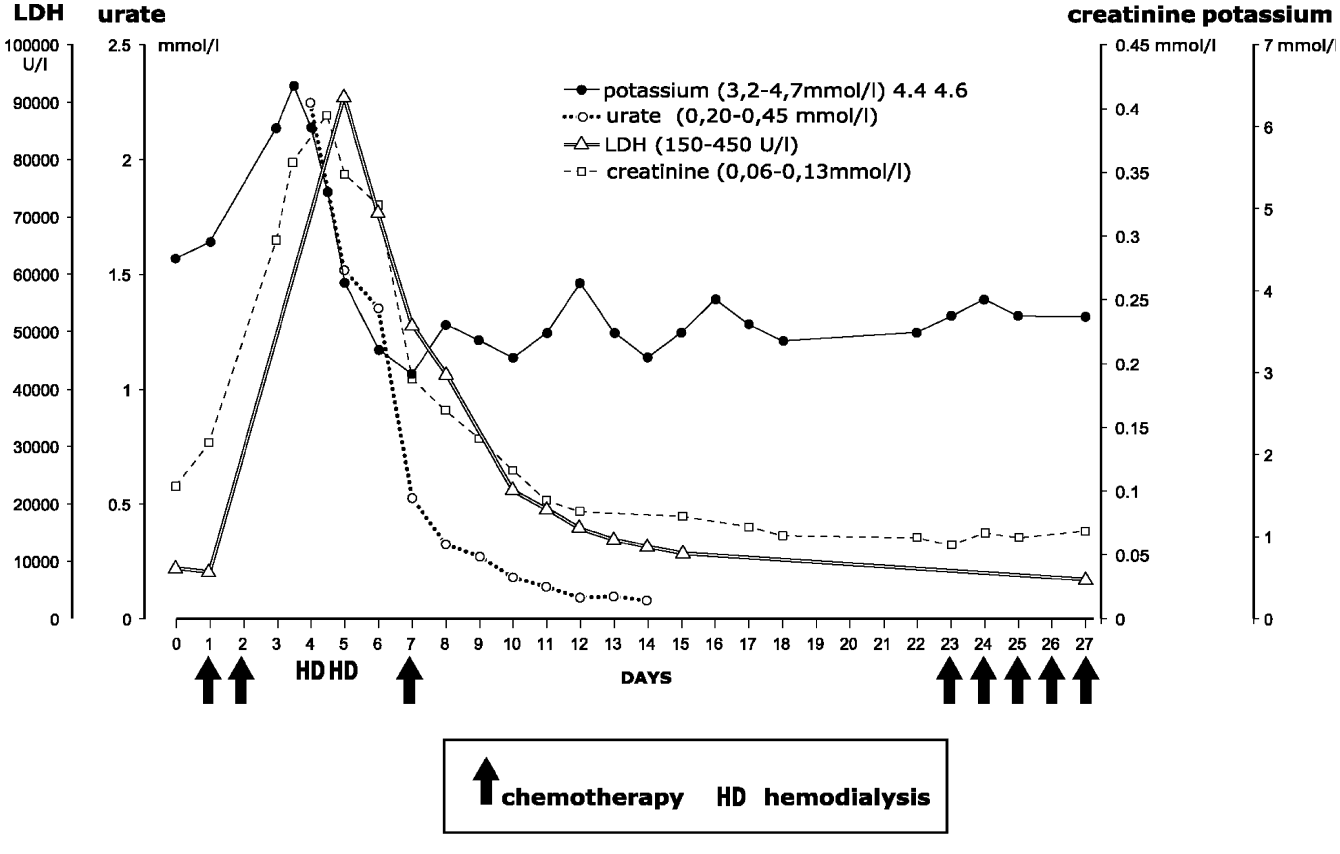

Fig. 1 Acute TLS following chemotherapy for metastatic medulloblastoma with acutely increased levels of potassium, urate, creatinine and LDH

Literature review

Previously, only one case of TLS following therapy for medulloblastoma has been reported. The particular case was a 34-year-old woman with metastatic medulloblastoma, who received palliative radiotherapy for a rapidly expanding abdominopelvic mass. Following radiotherapy the patient developed TLS, which resolved following standard treatment [4].

Reviewing the literature, a total of 45 cases were found from the first report in 1977 to 2002. Among these cases, 37 occurred in tumors highly or moderately sensitive to therapy (Table 1). However, eight of the cases occurred in tumor types (melanoma, sarcoma and hepatocellular carcinoma) traditionally regarded as relatively insensitive to therapy. In six of these latter cases the patients presented with bulky, metastatic disease.

TLS has been reported in nine patients in connection with small-cell carcinoma, in seven patients localized to the lung, in one to the skin and in one to the colon. TLS has been reported in seven patients with breast cancer. Casuistic reports include four cases of neuroblastoma, four of melanoma and four of germ cell tumors. Gastrointestinal cancer has been reported in four patients. TLS has been reported in only two patients with

Table 1 Tumor types in TLS (*ALT* autolymphocyte therapy, *Bleo* bleomycin, *Ca* carboplatin, *CCNU* lomustine, *Ci* cisplatin, *CPT* irinotecan, *CS* corticosteroids, *C* cyclophosphamide, *Da* dacarbazine, *D* doxorubicin, *Epi* epirubicin, *E* etoposide, *F*5-FU, *I* iphosphamide, *IF* interferon-alpha, *IL* Interleukin-1, *M* methotrexate, *Mi* mitoxantrone, *mAb* anti-GD3 monoclonal antibody, *Pac* paclitaxel, *RT* radiotherapy, *Tn* teniposide, *TCE* transcatheter chemoembolization, *TNF* tumor necrosis factor-alpha, *V* vincristine, *Vbl* vinblastine)

Sensitivity	Tumor type	Age	Treatment	Onset of TLS	Outcome of TLS	Reference	Year
High	Small-cell carcinoma	74 years	Ci, E	2 days	Resolved	7	1997
		57 years	D, Ci, E, V	36 h	Died	31	1983
		78 years	D, C, V	7 days	Resolved	32	1983
		67 years	CCNU, C, M	24 h	Died	33	1988
		57 years	D, C, V	4 days	Resolved	34	1990
		64 years	Ci, E	7 days	Resolved	33	1988
		65 years	D, I	3 days	Resolved	38	1991
		61 years	Ci, E	4 days	Died	14	2001
		52 years	Ci, E	2 days	Resolved	17	1999
	Germ cell tumors	52 years	None	4 weeks	Resolved	37	2001
		24 years	None	Weeks?	Resolved	37	2001
		52 years	Ci, E, Bleo	45 h	Resolved	35	2000
		58 years	Vbi, Bleo	3 days	Resolved	24	1989
	Neuroblastoma	2 weeks	V, Tn, RT	Not reported	Resolved	27	1994
		3.5 months	RT	Not reported	Resolved	27	1994
		2 days	V, Tn, RT	Not reported	Resolved	27	1994
		4 months	C, Tn	1 week	Resolved	27	1994
	Medulloblastoma	34 years	RT	4 days	Resolved	4	1984
		23 years	Ci, E	24 h	Resolved	–	2002
	Hepatoblastoma	7 months	Surgery	Acute	Died	21	1990
Moderate	Breast carcinoma	57 years	C, M, F	2 days	Resolved	24	1989
		31 years	Mi	4 days	Resolved	13	1994
		56 years	Pac	24 h	Died	39	1997
		94 years	Tamoxifen	7 days	Resolved	28	1986
		62 years	None	15 days	Died	30	1995
		53 years	F, D, C	18 h	Died	29	1987
		73 years	RT	48 h	Died	8	2000
	NSCLC	72 years	None	Spontaneous	Died	15	2000
		38 years	CPT, Ci	13 days	Resolved	18	1998
	Vulvar carcinoma	74 years	Ci, F	9 days	Died	16	1996
		66 years	Ci, F	36 h	Resolved	42	1993
	Thymoma	33 years	Ci, D, CS	24 h	Resolved	11	1997
	Ovarian carcinoma	47 years	Ca, C	4 days	Resolved	22	1993
	Gl tract	50 years	None	Spontaneous	Died	23	1977
	Colorectal carcinoma	42 years	CPT	8 days	Died	20	1996
		38 years	CPT	6 days	Died	40	2000
	Gastric carcinoma	36 years	None	Spontaneous	Resolved	10	2001
Insensitive	Melanoma	41 years	Ci, Da, IF	2 days	Died	36	2001
		76 years	TNF, mAb	8 h	Died	25	1994
		61 years	IL, IF, Ci, Vbl, Da	24 h	Resolved	12	1999
		56 years	CS	7 h	Resolved	41	2002
	Hepatocellular carcinoma	44 years	TCE	8 h	Died	19	1998
		46 years	TCE	12 h	Resolved	19	1998
	Sarcoma	66 years	C, ALT	16 h	Resolved	9	1993
		9 years	Ca, Epi, V	48 h	Resolved	26	1993

non-small-cell lung cancer. Also, two cases of TLS in sarcoma, two in vulvar carcinoma and two in hepatocellular carcinoma have been reported. Only one case of TLS has been reported in each of the following tumor types: hepatoblastoma, ovarian cancer, thymoma and medulloblastoma.

With respect to treatment modalities, TLS was seen following single-agent or combination chemotherapy in 31 of 45 reported patients. TLS has also been reported following radiotherapy alone or in combination with chemotherapy, following surgery, following endocrine therapy with corticosteroids and tamoxifen and following treatment with biological response modifiers plus interferon, and spontaneously. The only reported case of TLS related to surgery was seen in a patient with hepatoblastoma during primary surgical resection, believed to be due to ischemic necrosis of the tumor.

Of the 45 patients with TLS, 39 presented with metastatic disease. TLS occurred in only six patients with localized or locally advanced disease. The age of the patients showed a wide range from a 2-day-old infant to 94 years of age, with a median age of 56 years.

Increased LDH prior to treatment, as a marker of tumor burden, is a risk factor for TLS and was here documented in 82% of the evaluable patients (see Table 2). Other risk factors included pretreatment hyperuricemia and renal impairment, found in 47% and 33% of the evaluable patients, respectively. The pretreatment risk factors were not found to be particularly prevalent in those patients with insensitive tumor types. The onset of TLS varied widely, from a few hours to several weeks. In 87% of the evaluable patients, the onset was within 7 days of treatment.

Table 3 shows the characteristic biochemical features presented by the patients with TLS. Some of the reports do not describe all relevant parameters. Hyperuricemia and increased LDH were the most consistent findings, presented by 98% and 96%, respectively. Acute renal insufficiency was found in 93% of the patients. Hyperphosphatemia and hypocalcemia each occurred in more than 89% of the evaluable patients. Hyperkalemia was documented in 73% of the patients. Also metabolic acidosis was a common feature, reported in 83% of the evaluable patients.

With respect to outcome, one-third of the reported patients (16/45) died as a result of TLS. The causes were either hyperkalemia with cardiac arrhythmias or acute uremia.

Table 2 Pretreatment risk factors for TLS in solid tumors. Denominators indicate evaluable cases in which specific results were reported

Azotemia	13/40 (33%)
Elevated LDH	24/29 (82%)
Hyperuricemia	14/30 (47%)

Table 3 Biochemical characteristics of TLS in solid tumors. Denominators indicate evaluable cases in which specific results were reported

Azotemia	41/44 (93%)
Increased LDH	25/26 (96%)
Hyperuricemia	42/43 (98%)
Hyperkalemia	31/42 (73%)
Hyperphosphatemia	38/42 (89%)
Hypocalcemia	34/39 (89%)
Metabolic acidosis	15/18 (83%)

Discussion and conclusion

Within the first 2 days after initiation of chemotherapy our patient with metastatic medulloblastoma developed TLS, which showed all the classical features and was complicated by renal insufficiency. Our patient had normal renal function prior to treatment, but significantly increased levels of LDH. He had bulky disease with massive extracerebral manifestations of the medulloblastoma including metastases to the liver, mediastinal lymph nodes and bone marrow metastases. Additionally, medulloblastoma, being a typical childhood cancer, is known to be particularly sensitive to chemotherapy. All these factors seem to place the patient in a high-risk group for developing TLS. Our patient recovered completely, demonstrating that immediate hemodialysis may reverse the toxicity.

TLS is a rare complication of cancer therapy for solid tumors, although the reported cases likely underestimate the true incidence. We found that TLS is most frequently seen in patients with metastatic disease and with tumors highly sensitive to antineoplastic therapy. However, surprisingly, TLS was also reported in patients with relatively insensitive tumor types. It is possible that improvement in the systemic treatment of various solid tumors with increasing responses seen in recent years may increase the potential incidence of TLS, if action to prevent the complication is not taken. Increased LDH, hyperuricemia and pretreatment renal impairment are here documented to be risk factors for the development of TLS.

TLS is most often seen during chemotherapy in hematological malignancies, such as acute lymphoblastic leukemia and Burkitt's lymphoma [3, 5, 6]. Hande et al. found the incidence of TLS among patients with high-grade non-Hodgkin's lymphoma to be 42%, although only clinically significant in a minority of these patients [5]. We found the mortality rate among patients with TLS in solid tumors to be higher than among those with hematological malignancies [5, 6], where prophylactic measures more often are implemented and where awareness of the syndrome is higher.

The primary aim of treatment is the prevention of hyperuricemia and achievement of a high urine flow, as this will reduce the likelihood of uric acid and/or calcium phosphate precipitation in the renal tissue, and will also increase the elimination of potassium. The mainstay of treatment has been intravenous hydration and allopurinol (oral and intravenous preparations available).

Where possible hydration and allopurinol should commence at least 24 h prior to cytotoxic chemotherapy to ensure adequate renal function. Vigorous diuresis may also prevent crystallization of xanthine and other purine metabolites, whose excretion is increased with the use of allopurinol. The use of alkalization is controversial. Although in theory the production of alkaline urine will improve the excretion of uric acid, vigorous urinary alkalization may increase the likelihood of calcium phosphate precipitation in the renal tubules, so bicarbonate administration should be used with caution or avoided altogether. In addition, metabolic alkalosis may worsen the neurological manifestations of hypocalcemia. An alternative (and more effective) drug to allopurinol is urate oxidase, a uricolytic enzyme that converts uric acid to allantoin. In several studies the use of either the nonrecombinant urate oxidase or rasburicase (a new recombinant form of urate oxidase) has demonstrated both more rapid control and lower levels of plasma uric acid and better renal function outcome compared to the use of allopurinol [43, 44, 45, 46, 47, 48].

Although rare, TLS following therapy for solid tumors is a potentially fatal complication and preventable in practically 100% of patients, which makes it important to avoid. The mortality rate of TLS in solid tumors was here found to be one in three. High awareness is required especially in patients with sensitive tumor types, metastatic disease and known pretreatment risk factors, including renal impairment, hyperuricemia and increased LDH. Prophylactic treatments to avoid TLS include allopurinol, hydration prior to treatment and alkalization of the urine. Urate oxidase (rasburicase) seems to be a more effective way of reducing hyperuricemia and thereby the risk of TLS.

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