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Case Report

Tumor lysis syndrome after chemotherapy for metastatic colic carcinoma: About two adult cases

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Abstract

Tumor Lysis Syndrome (TLS) is a major oncological emergency involving metabolic perturbations. It occurs when tumor cells release their contents into the bloodstream, either spontaneously or in response to therapy. TLS is characterized by hyperuricemia, hyperkalemia, hyperphosphatemia and hypocalcemia following massive lysis of malignant cells. Although this syndrome is well described, it is rarely seen or suspected in solid malignancies. The frequency and severity of TLS is partly dependent upon the biology of the disease and type of therapy administered. We report in this work two cases of tumor lysis syndrome occurring after chemotherapy for endocrine colon carcinoma with small metastatic cells.

Introduction

Tumor lysis syndrome is an oncological emergency with potentially serious consequence [1,2]. It results from massive tumor cell lysis, sometimes spontaneous but most often secondary to local or systemic treatment of cancerous lesions [2,3].

This syndrome is more frequently described in the case of malignant hemopathies (high grade lymphomas, acute leukemias) [1,2]. Solid tumors are rarely complicated by tumor lysis syndrome [1,2,3].

It most often combines hyperuricemia, hyperkalemia, hyperphosphatemia, hypocalcaemia and an increase in serum Lactate Deshydrogenase (LDH) [2,3].

Observation 1

A 46-year old female who was in a normal state of health until five months prior to this admission was diagnosed as having colonic cancer following initial work-up for upper right abdominal pain. She had no family history of cancer. She underwent left colectomy

The three-layer immunoperoxidase histological analysis on dewaxed sections confirmed an endocrine tumor with poorly differentiated small cells with positive cytoplasmic labeling for antichromogranin and for antisynaptophysin.

An abdominal CT scan found diffuse liver metastases invading approximately 70% of the hepatic parenchyma. Before chemotherapy, Serum Glutamo Pyruvate Transférase (SGPT) was 4N, Glutamo Oxalo acétate Transférase (SGOT) was 4N and alkaline phosphatase was 5N. She underwent chemotherapy: cisplatin (100mg/m^2 fist day) and etoposide (120mg/m^2 day 1 to day 3) combined with hydration with three liters of 2.5% glucose solution, 500ml of 10% mannitol and 20mg of furosemide. Multidisciplinary Concertation Meeting decided adjuvant chemotherapy. (FOLFIRI+cetuximab (Cmab) therapy).

After the first chemotherapy session (and despite hyperhydratation), the patient presented significant localized pain in the right hypochondrium, tachypnea and tachycardia but without digestive intolerance. Biological tests revealed showed severe acidosis acute renal failure, hyperuricemia, hyperphosphatemia, elevation of Lactic Deshydrogenase (LDH) (Table1).

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Table1: Metabolic disturbances occurring after the administration of the chemotherapy.

	LDH	Uricemia	kaliema	Calcemia	Phosphorus
Normal Values	**135-214 UI/l**	**142-339 µmol/l**	**3.5-4.5 mmol/l**	**2.15-2.55 mmol/l**	**0.81-1.45 mmol/l**
Before chemotherapy	541	307	4.7	2.5	1.12
3 days after chemotherapy	972	475	4.7	2.2	1.73
1 mouth after chemotherapy	365	600	4.8	2.33	0.84

Clinical findings and biological disturbances had confirmed the diagnosis of tumor lysis syndrome.

The patient was treated by hyperhydration and urine alkalization (3 liters of physiological saline and 1 liter of bicarbonate at 14 ‰ per 24 hours), with normalization of metabolic anomalies.

The third curse of chemotherapy had modified in favor of combination of carboplatin and etoposide taking into account the occurene of severe hearing loss secondary to cicplatin. Alkaline hyperhydration (3 littres of physiological saline and 1 liter baking soda at 14‰ per 24 hours) started the day before and continued during the cure could prevent a recurrence of the tumor lysis syndrome. An abdominal scanner performed after this last course had confirmed a regression of about 50% of liver damage and a quasi-normalization hepatic assessment (ASAT: N, ALAT: N, PAL: 1.9N).

Observation 2

A 52-year-old women, with no significant medical history, was diagnosed with a well differentiated adenocarcinoma of the left colic angle, metastasized to the liver and spleen. Multidisciplinary Concertation Meeting decided surgical ablation of the tumor and chemotherapy for metastasis She underwent a left colectomy followed by 3 monthly FOLFOX-based chemotherapy courses. One week after the third session, biological tests showed a serum elevation of uric acid to 500 µmol/l and of LDH to 600 IU/l. Despite alkaline hyperhydration, the evolution was marked by the progressive deterioration of the general state.

Discussion

Tumor lysis syndrome results from the massive destruction of neoplastic cells, most often following a chemotherapy or radiation therapy [1,4,5]. More rarely, it can occur spontaneously or following hormone therapy, immunotherapy or corticosteroid therapy [3-5]. This syndrome most often associates hyperuricemia hyperkalemia, hyperphosphatemia, hypocalcemia and renal failure [1]. It occurs more frequently after aggressive treatment of rapidly developing tumors such as high grade malignant lymphomas or acute leukemias [5-7]. In recent years, there have been a few case reports about TLS developing in patients with colic carcinoma.

Tumor lysis syndrome is the consequence of massive destruction of tumor cells, leading to release into the circulation of intracellular compounds. So are released the phosphates and nucleic acids, potassium and calcium. The purinosynthesis of novo results in the transformation of purine bases into uric acid and it is the main mechanism of hyperuricemia. Mitochondrial dysfunction and kidney failure acute can cause satellite acidodosis syndrome lysis. [1-3].

Kidney failure can occur mainly due to precipitation of phosphocalcic crystals or acid uric. The released calcium will bind to the phosphates, leading on the one hand to the precipitation of crystals phosphocalcic and on the other hand to paradoxal hypocalcemia. If calcium phosphate precipitates in the cardiac conducting system, serious dysrytmias can occur [6].

The low prevalence of this syndrome in the case of solid tumors is mainly explained by their low proliferative index and their relatively slow response to specific treatments [6-8]. Most cases of solid tumors complicated by lysis syndrome are of endocrine origin with small cells, especially of bronchial localization [3,4,9]. Other histological types have also been described, such as choriocarcinoma or leimyosarcoma [3]. The first lysis syndrome described in the literature for endocrine small cell colonic tumors was reported in 1988 [8].

We find similarities between our observations and those in the literature, especially massive liver metastases and a high level of LDH before chemotherapy. Such findings were considered by several authors as predisposing factors for lysis syndrome in this type of tumors. Clinical and biological anomalies associated with lysis syndrome are generally reversible when they are early diagnosed and correctly treated. However, they can be complicated by severe hyperkalemia, cardiac arrhythmias and even sudden death [2,5,6]. In a series of 25 cases of solid tumors complicated by tumor lysis syndrome, nine deaths have been reported [4]. Due to rarity of lysis syndrome in solid tumors, systematic preventive measures (in particular alkaline hyperhydration and administration of allopurinol) are not justified. However, due to the morbidity and mortality associated with it, it is recommended to carry out these measures as well as rigorous clinical and biological monitoring, especially in case of large mass tumors and high LDH levels, before and during all the specific treatments [7]. Quick identification of TLS is critical as treatment involves early aggressive hydration to increase renal perfusion and urine output and therefore decrease the risk of crystal precipitation, and correction of electrolyte abnormalities1 [10].

Conclusion

The development of TLS in solid tumors is associated with increased mortality and therefore, a high index of suspicion is essential for early diagnosis and treatment initiation. TLS is only rarely associated with treatment of solid tumors. Precautions should be taken to avoid this potentially fatal complication.

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