Tumour lysis syndrome

The tumour lysis syndrome occurs after lysis of large numbers of circulating tumour cells, typically leukemic blasts. It an oncological emergencies. This massive breakdown is most often caused by cytostatics or antibody therapy of the malignancy. The cytoplasm of the cells leaks into the circulation. Most of the time, it is leukocytes that release biologically active substances - interleukins, which cause metabolic disruption. To remove these metabolites, the filtration capacity of the kidney is exceeded, creatinine and urea rise. The cells release potassium and hyperkalemia develops.

Etiology

- often begins after the initiation of cytotoxic therapy in patients with hemato-oncological disease (ALL, AML or NHL)

Diagnosis

- **LAB:** ↑ Creatinine (AKI), ↑ K^{+}, ↓ Ca^{2+}
- **BC:** ↑ LEU (50-100x10^9/l), can be ↓Hb and ↓Tro
- **Urine:** urate crystals
- **ECG:** arrhythmia
- **Markers of cell lysis:** ↑ LDH, ↑ uric acid, ↑ PO_4^{3-}

Clinical picture

- renal failure - AKI: oedema, oliguria, lethargy
- hyperkalemia: cardiac arrhythmias, nausea, diarrhea
- hypocalcemia: spasms

Therapy

- **fluid management**
  - Low risk: p.o. / i.v. **hydration**, fluid balance monitoring
  - High risk: aggressive IV therapy, **hyperhydration** F1/1,
  - **monitor urine output** → possibly initiate **forced diuresis** (furosemide);
- **prevention and treatment of urate nephropathy**
  - Alopurinol/Milurite, Rasburicase
- **correction of electrolyte balance**
  - hyperkalemia: ECG motorization, glucose with insulin
  - hyperphosphatemia: Sevelamer (phosphate binders)
  - hypocalcemia: 10% Calcium gluconicum

Links

Related articles

- Basic diagnostic tests in hematooncology

Literature used

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- KLENER, Pavel. *Vnitřní lékařství.* 3. vydání. Praha : Galén, 2006. ISBN 80-7262-430-X.

Kategorie:Vnitřní lékařství Kategorie:Hematologie Kategorie:Onkologie