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Electrocardiographic Case – Electrocardiographic Clinical Diagnosis in a Patient with Post-Chemotherapy Emesis and Oliguria

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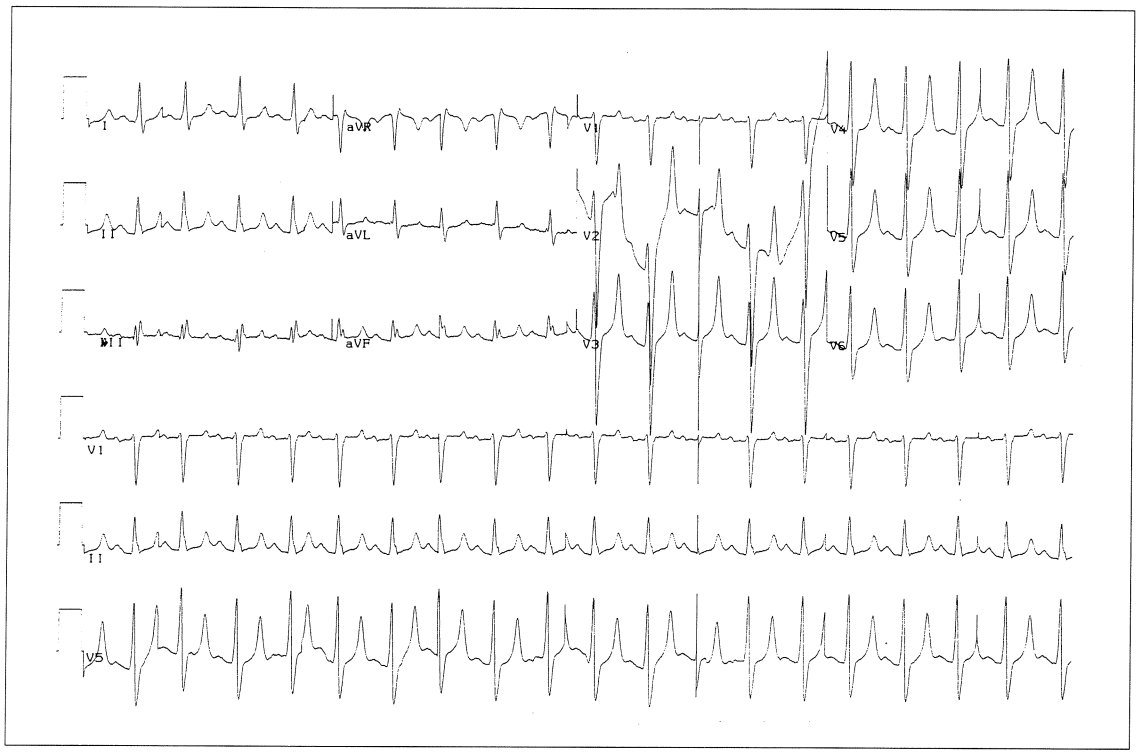

Fig 1 – 12-lead electrocardiogram (ECG) which shows typical hyperkalaemic changes with hypocalcaemic feature.

A 16-year-old girl presented with a one-month history of lethargy and extreme fatique. On examination, she was pale and her vital signs were normal. There was generalised lymphadenopathy, moderate hepatosplenomegaly associated with bilateral papilloedema and retinal haemorrhages. Chest radiograph showed anterior mediastinal mass. The white blood cell (WBC) count was 407 x 10^9/L with 90% blast cells, haemoglobin 7.3 g/dL and platelets 30 x 10^9/L. Bone marrow examination was consistent with T-type acute lymphoblastic leukemia (T-ALL). Induction chemotherapy was commenced after 3 days of hyperhydration, urine alkalinisation and allopurinol. Twelve hours later, she developed severe vomiting and oliguria.

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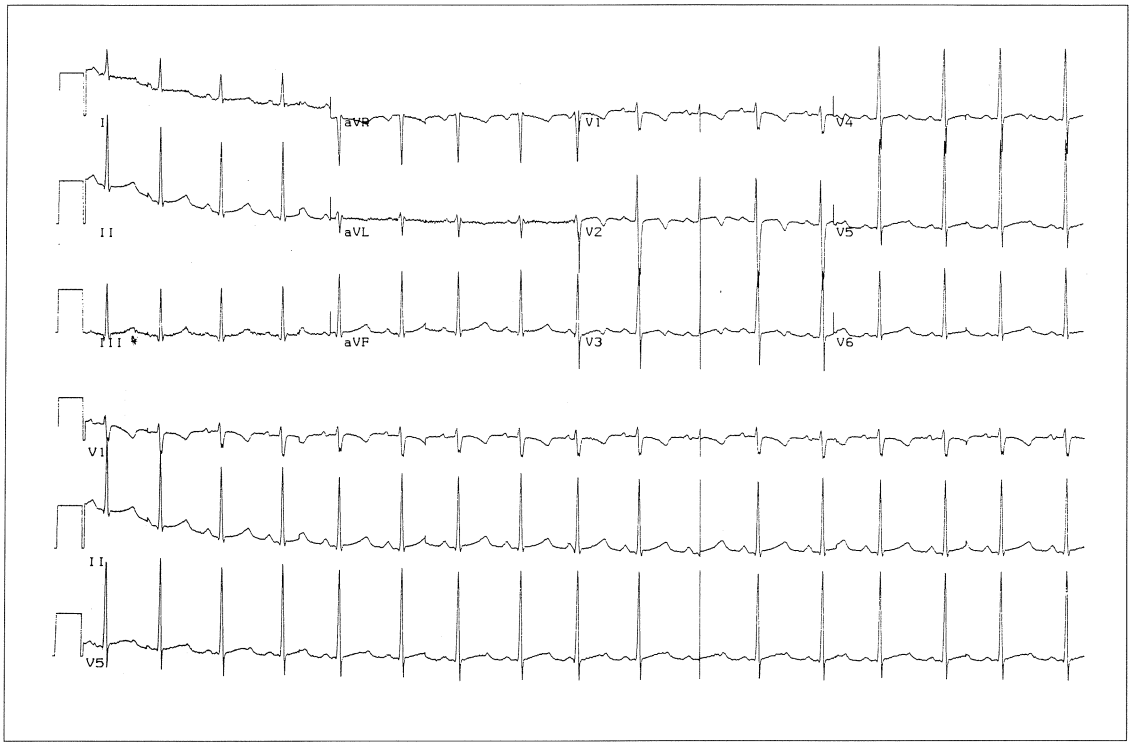

Fig 2 – The repeat 12-lead ECG after the initial treatment with intravenous sodium bicarbonate, calcium gluconate and dextrose/insulin.

Diagnosis

Severe hyperkalaemia and hypocalcaemia from acute tumour lysis syndrome

DISCUSSION

A 16-year-old girl with T-ALL presented with acute tumour lysis syndrome following induction chemotherapy despite prophylaxis with allopurinol and hyperhydration therapy. The pretreatment serum chemistry evaluation showed a potassium level of 5.8 mmol/L, sodium 136 mmol/L, urea 1.9 mmol/L, creatinine 75 μmol/L and uric acid 841 μmol/L. The baseline ECG was normal. Twelve hours following chemotherapy with oral prednisolone and intravenous vincristine and daunorubicin, the patient developed severe vomiting associated with reduction in urine output. Intravenous granisetron failed to control the vomiting episodes. A contrasted CT brain scan showed normal results. The ECG (Fig 1) shows very peaked, symmetrical and tall T waves with a narrow base QRS which are best seen in the precordial leads especially V2 – V5, typical of acute hyperkalaemia. There is prolongation of QT interval (QTc = 510 ms), consistent with hypocalcaemia. An urgent serum chemistry test showed potassium of 9.0 mmol/L, sodium 136 mmol/L, urea 17.7 mmol/L, creatinine 81 μmol/L, calcium 1.78 mmol/L, phosphate 3.6 mmol/L and uric acid 1120 μmol/L. Treatment for tumour lysis syndrome with intravenous sodium bicarbonate, calcium gluconate and dextrose/insulin, aggressive hydration, loop diuretic and high doses of allopurinol was initiated. Twenty-four hours later, the vomiting stopped and the urine output improved. The repeat ECG was normal (Fig 2). There was also regression of lymphadenopathy, hepatosplenomegaly, funduscopic changes and the anterior mediastinal mass. The WBC count was 6.7 x 10^9/L without any blast cell.

Acute tumour lysis syndrome (ATLS) is characterised by hyperkalaemia, hyperuricaemia, hyperphosphataemia, hypocalcaemia, lactic acidosis and azotaemia[1]. It usually occurs in the course of aggressive cytoreductive therapy in rapidly growing lymphoproliferative malignancies with large tumour burden due to massive tumour cell lysis[2]. Very rarely does spontaneous necrosis of malignancies causes tumour lysis syndrome[3]. The likelihood of developing ATLS is related to the sensitivity of the tumour to the particular treatment modality, the disease bulk or clinical stage, and the patient's renal function. Important pretreatment biochemical predictors include hyperuricaemia and high serum levels of lactate dehydrogenase, both of which correlate with total tumour burden and renal failure. In our patient, the presence of bulky disease, high blast cell counts, hyperuricaemia and hyperkalaemia at presentation indicates that the risk of developing ATLS is high. This is further supported by the highly chemosensitive nature of the neoplastic cells as evidenced by regression of the tumour mass and normalisation of the white cell count within 24 to 48 hours following induction chemotherapy.

The nature and severity of the metabolic derangements in ATLS vary and may be influenced by the intensity of the chemotherapy, the magnitude of cell lysis, and the general condition of the patient with respect to hydration and glomerular filtration rate. If left untreated, it may result in potentially fatal renal, cardiac and

neurologic complications. On the other hand, it may be prevented with allopurinol therapy combined with aggressive hydration aimed at establishing an ongoing alkaline diuresis to ensure optimal renal function. Despite apparently ideal preventive management, renal failure still occurred in 25% of cases in a study of Burkitt's lymphoma[4].

Suggested therapy for ATLS includes intensive hydration to maintain urine output of 3 – 4 L/day, 600 mg/day allopurinol (lower doses for paediatric patients and patients with renal insufficiency), alkalinisation of the urine and haemodialysis for frank renal failure[5]. Attempts at tumour reduction by leucapheresis in haematological malignancies with high peripheral white cell counts have not been proven successful because of the large bulk of tumour that remains in the marrow. Leocapheresis is therefore probably not indicated unless there is coexisting leucostasis[6].

CONCLUSION

ATLS and acute renal failure remain significant causes of morbidity and mortality in acute leukemia with large tumour burden despite conventional management with aggressive hydration, allopurinol and the slow introduction of chemotherapy. Recognition of risk factors and prevention are the most important steps in the management of ATLS.

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