pulmonary embolism incidence. They reported that pulmonary embolism incidence was correlated positively with vapor pressure and rainfall. Our study did not take rainfall, but air pressure was observed to be a factor with an impact on pulmonary embolism incidence, consistent with the above mentioned study. Various proposals have been made to explain the association between venous thromboembolism and seasonal factors. Patients in this study were observed to have formed 2 similar clusters. Temperature and air pressure were the differences between the clusters. We established that predisposition factors to pulmonary embolism did not differ between the clusters. The number of patients in our study was limited since we enrolled only the patients who possessed all predisposition factors.

In conclusion, this present study which is aimed to analyze the patients with pulmonary embolism through cluster analysis, a multivariate classification method, revealed that clinical variables did not have an impact on the formation of the clusters and seasonal factors were associated with the classification of patients with pulmonary embolism, and that these factors may have an impact on clinical variables.

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Busulfan induced myoclonus

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Busulfan is an alkylating chemotherapeutic agent. In combination with other chemotherapeutic drugs, it is an acceptable preparative (conditioning) agent before bone marrow and peripheral blood stem cell transplantation. Busulfan rapidly enters the central nervous system (CNS) and may cause seizures when used in a high dose.[1] Consequently, patient should receive prophylactic phenytoin (with therapeutic level) to begin before busulfan and continued for 24 hours after the last dose. There is a wide intra- and interindividual variation of absorption and metabolism of busulfan. In addition, pharmacokinetic differences exist between age groups.

Our patient is a 16-year-old Omani lady with precursor B cell acute lympholytic leukemia, diagnosed on September 2004. She has an unremarkable medical history. She was started on chemotherapy. As cytogenetic study showed Philadelphia chromosome positive, that put her at a high risk of relapse, it was decided to proceed to allogeneic bone marrow transplantation as early as possible in the first remission. She was admitted to bone marrow transplantation unit on April 2005, her body weight was 39 kg, height 154.6 cm and body surface area 1.3 m.[2] The preparative regimen consisted of fludarabine and busulfan.[3] Aiming at decreasing the regimen related to toxicity, a busulfan pharmacokinetic study was performed.[2] Two days before starting the regimen, a study was carried out using a busulfan test dose as a guide to the appropriate actual dose. This was followed by monitoring of the serum busulfan level throughout the regimen.[4] Phenytoin was started before the Busulfan (BU) test dose, continued through the regimen and until 24 hours after the last dose of BU. She received an oral stat dose of phenytoin 300 mg, followed by 100 mg tid oral. Serum free phenytoin level before starting BU was 3.5 umol/l (therapeutic range 3.3-9.6). The 4th day regimen consisted of a single daily intravenous (iv) dose of BU and fludarabine. On the first and second days she received daily 140 mg BU, while on the third and fourth she received daily 160 mg, guided by the prior regimen study and serum level monitoring. After receiving the last dose of BU, she developed for a few second a generalized myoclonus. Again and after the fourth hour of this episode, she

had an interrupted repeated myoclonus, each last for few seconds. This cluster of seizures spontaneously stopped and did not recur. She never had lost her consciousness. Clinical examination did not show any neurological abnormality. Since admission till the onset of the convulsions she had normal temperature, 0_2 saturation, serum electrolytes and serum glucose. As planned she received peripheral blood stem cells transplantation on twelfth and thirteenth February 2005, and she was successfully engrafted on 1st March 2005.

Phenytoin is a widely used prophylaxis to prevent BU induced seizures. The researches in this field virtually studied the conventional daily 4 doses of oral BU, while in this study we used the single daily dose of iv BU regimen.[3] Our patient had seizures in spite of a prior regimen pharmacokinetic study, monitoring of serum BU level and achieving a therapeutic serum phenytoin level. More research is expected to study this single daily dose iv BU regimen and there may be a need to investigate for other(s) effective and safe alternative seizures prophylaxis.[5]

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Chlorambucil therapy in children with steroid-resistant nephrotic syndrome

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Chlorambucil (CHL) had been used as treatment for childhood nephrotic syndrome (NS) for more than half a century.[1] It was used mainly in children with steroid sensitive nephrotic syndrome (SSNS) as steroid sparing agent in those with frequent relapsing or steroid dependent course.[2] However, it was observed to result in higher rates of severe side effects and recommended as a second line drug after cyclophosphamide (CYC), which is considered as safer alkylating agent.[2] The alkylating agents have been used for treating steroid resistant nephrotic syndrome (SRNS).[2] However, CYC was used in most of the studies,[3] while CHL was used only rarely.[4,5] Elzouki et al showed in a small study that CHL induced complete or partial remission in patients with SRNS caused by either focal segmental glomerulosclerosis (FSGS) or mesangial proliferative glomerulonephritis (MPGN).[4]

In this retrospective study, we report our results of using CHL in children with SRNS secondary to IgM nephropathy, FSGS or diffuse mesangial hypercellularity (DMH). All patients presented to our unit over 20 months period (from February 2002 until June 2004) and were diagnosed as SRNS were recruited. Steroid resistant nephrotic syndrome was defined as a failure to go into remission after 4 weeks of prednisolone therapy at a dose of 60 mg/m^2 /day, plus 3 intravenous doses of methylprednisolone (600 mg/m^2/day or 30 mg/kg/day) on alternate days. We had 7 patients with SRNS. All patients were females. The median (range) age at presentation was 4 (2-9) years. All except 2 were Arab in origin. All studied children were primary non-responders to prednisolone and 2 were also resistant to intravenous cyclophosphamide course. All the 7 children were treated with CHL (0.1-0.2 mg/kg/day) for 8-12 weeks. The mean $\pm$ SD accumulative dose was 10.1 + 3.3 (7.0-15.2) mg/kg. All patients were continued on oral prednisolone 40mg/m^2 on alternate days and received enalapril (0.5-1 mg/kg) throughout the CHL therapy. Two patients achieved complete remission after 12 weeks of CHL therapy. One patient remained in remission for 2 years following CHL therapy and one patient had a relapse once after 1.5 years of follow up, which responded to prednisolone promptly. She had been on