Clinical Pathology

**Serum cystatin C as an early predictor of acute kidney injury in preterm neonates with respiratory distress syndrome**

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**Background:** Preterm neonates are highly vulnerable to acute kidney injury (AKI). We aimed at determining whether serum cystatin C (sCysC) on day 3 of life (D3) can early predict acute kidney injury (AKI) in preterm neonates with respiratory distress syndrome (RDS).

**Methods:** This prospective study was conducted on 50 preterm neonates with RDS and 25 without RDS. On D3, sCysC, serum creatinine (sCr) and blood urea nitrogen (BUN) were measured and estimated glomerular filtration rate (eGFR) was calculated. Neonates were evaluated for development of AKI during the first week of life according to the modified pediatric RIFLE (pRIFLE) criteria.

**Results:** Thirteen neonates with RDS developed AKI (26%). There was no significant difference between RDS group & controls regarding sCysC. RDS neonates with AKI had significantly higher sCysC than those without AKI ((1.62 ± 0.12 versus 1.16 ± 0.09 mg/l; p < 0.001). Neonate with AKI grade F (failure) had significantly higher sCysC than those with R failure and I (injury) (p = 0.028); RDS grade III–IV neonates had significantly higher sCysC than RDS grade I–II. D3 sCysC was not correlated with gestational age or birth weight. D3 sCysC at a cutoff point of > 1.3 mg/l with a sensitivity of 92.30% and specificity of 96% can predict AKI in preterm neonates with RDS while D3 sCr has poor sensitivity and specificity 76.90% and 68.0% respectively.

**Conclusion:** Preterm neonates with RDS are at increased risk of AKI. D3 sCysC can predict AKI earlier than sCr and eGFR.

**Secondary cytogenetic aberrations associated with T(8; 21) acute myeloid leukemia shed a light on disease progression and patients outcome**

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**Background:** Translocation (8; 21), t(8; 21), is one of the most common cytogenetic abnormalities in adult de novo acute myeloid leukemia (AML) patients. The t(8; 21) generates a novel fusion protein, AML1-ETO which affects a wide range of cellular molecules. Most of these alterations are in favor of increased proliferation and survival and decreased differentiation, but the fusion protein also has opposite effects, and this may explain why the fusion protein alone cannot induce leukemic transformation. More challenging will be to identify the molecular mechanisms of additional cytogenetic defects, such as loss or gain of specific chromosomes associated with t(8, 21) AML, because losses and gains suggest the presence of possibly one or more tumor suppressors or amplified oncogenes. The most frequent cytogenetic aberrations are loss of sex chromosome followed by deletion of the long arm of chromosome 9 (del 9q) and trisomy 8. However, previous studies showed conflicting data regarding the role of secondary cytogenetic aberrations in addition to t(8; 21).

**Objectives:** To our knowledge, our study is the first investigating the effect of additional aberrations; loss of X chromosome and del 9q on the clinicopathological and immunophenotypic characteristics and prognostic behavior of t(8; 21) de novo AML from the Middle East.

**Methods:** Fifty-six adults with de novo AML-M2 were enrolled. Detection of loss of X-chromosome and del 9q were performed using fluorescent in situ hybridization (FISH).

**Results:** More than half of the patients (53.6%) harbored a secondary chromosomal abnormality in addition to t(8; 21). Del 9q was found in 17.9% of the patients. A significant association was found between this chromosomal aberration and age, heptomegaly, high total leucocytic count and low platelets count (P < 0.05). Most patients had poor clinical outcome, high tendency for resistance to therapy and significantly shorter survival. On the other hand, loss of X chromosome was found in 25% of the studied patients and was not related to clinicopathological features or prognostic markers except for high platelets.

Hyperlipidemia in association with pro-inflammatory cytokines among chronic spontaneous urticaria: case-control study

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Chronic spontaneous urticaria (CSU) is a disorder characterized by recurrent transient itchy wheels of 6 weeks duration or longer. The cause cannot be pinpointed in about 40% of patients. To elucidate the possible association between CSU and hyperlipidemia, 40 CSU patients and 40 group matched healthy individuals were assessed for hyperlipidemia. Data on history, urticaria activity score (UA57), physical examination and routine laboratory investigations including (serum IL6 and TNF a) was recorded. Statistically significant increase of serum Cholesterol, Triglycerides (TG), low density lipoprotein (LDL), IL6, TNFα and decrease of high density lipoprotein (HDL) was found in CSU in comparison to control group (P = <0.001, <0.001, <0.001, <0.001, <0.001, 0.004 respectively). Regarding the different disease variables, both TG and cholesterol were positively correlated with duration of illness (P < 0.001, <0.001), urticaria score (P = 0.003, <0.001) and TNF α (P = 0.024, <0.001) respectively. Serum LDL detected significant positive correlation with duration of illness (P = <0.001), urticaria score (P = <0.001), CRP (P = <0.001) and TNF α (P = <0.001) while serum HDL detected significant negative correlation with TNF α (P = 0.033). IL6 and TNFs associated systemic inflammation could be a common pathogenic mechanism of CSU and hyperlipidemia. Patients with CSU should be evaluated for hyperlipidemia.