in 25%, similar observation reported by other workers,[3] those patients usually presented with fever and painful crisis without respiratory symptoms or pulmonary infiltrate and ACS usually develops 2-3 days later. The radiological findings showed the lower lobes of the lungs predominantly involved, similar finding observed by other workers.[3] Bacterial infection was identified only in 2 patients (17%), one due to *Streptococcus pneumoniae* and other due to *Staphylococcus aureus*, low incidence of bacterial infection was also reported in other studies.[1] Most of the studies relied on blood cultures and is therefore likely to underestimate the frequency of bacterial pneumonia. Bronchoscopy is more sensitive method in determining the etiology of ACS. Despite low incidence of bacteremia in our patients all received empirical intravenous antibiotics (ceftriaxone + erythromycin), vancomycin was added for those with severe ACS and prolonged fever, considering the emerging resistance of pneumococcus to the usual antibiotics. The majority of our patients (67%) received simple blood transfusion (BT), and all show dramatic clinical improvement. Transfusion of packed red blood cells has a number of potential benefits, it increases oxygen carrying capacity, decreases the fraction of Hb S and thereby potentially reduces or reverses intrapulmonary sickling.[4] Simple BT should be given for marked decrease in Hb and also for clinical deterioration, however BT should be avoided in cases of Hb above 10g/dl to prevent increase blood viscosity, which may lead to stroke or exacerbate ACS. Exchange transfusion should be reserved for rapid clinical deterioration, widespread pulmonary involvement, hypoxemia not corrected by delivery of supplemental oxygen and multiorgan failure.[4] The low recurrence and death rate also support the observation of mild ACS in western region similar to the eastern province.[1] As the etiology of ACS appears to be multifactorial, there is no effective and specific therapy, although a beneficial effect of dexamethasone in children with mild to moderately severe acute chest syndrome was demonstrated in a prospective randomized double blind placebo controlled clinical trial,[5] the study showed significant reduction in the length of hospitalization, decrease duration of oxygen supplementation, decrease duration of opioid analgesia, decrease occurrence of clinical deterioration and decrease the need for BT. The steady state hematological data (**Table 1**) did not show clear risk factors for occurrence of ACS.

In conclusion, we found that the majority of ACS in our region is due to undetermined origin presumably secondary to rib infarction, atelectasis or fat embolism and further prospective study is required to confirm this possibility, we also conclude that bronchial asthma is an important precipitating factor for occurrence of ACS in SCD. Finally, we also recommend simple BT to be part of the management of ACS in children.

Acknowledgment. We would like to thank the staff of the Pediatric Department, Miss Khadija Karani and Miss. Ghadir Al-Muallad, Staff Nurse in Hematology Unit, for their help in collecting medical data, to Miss Darna S. Alie for her secretarial work. Thanks are also due to the Hospital Director, Dr. Nabeel A. Yamany for giving us permission to undertake the study and to Dr. Ghulam Nabi Registrar, Neonatology Department Madina Medical and Children Hospital, Madina, Kingdom of Saudi Arabia for his critical review.

Received 13th June 2003. Accepted for publication in final form 10th September 2003.

From the Department of Pediatrics, Madina Maternity and Children's Hospital, Madina, Kingdom of Saudi Arabia. Address correspondence and reprint requests to: Dr. Zakaria M. Al-Hawsawi, Consultant Pediatrician, Department of Pediatrics Madina Maternity and Children's Hospital, PO Box 6205, Madina, Kingdom of Saudi Arabia. Tel. +966 (1) 8380395. Fax. +966 (1) 8368333. E-mail: zhawsawi@yahoo.com.

References

1. Al-Dabbous IA. Acute chest syndrome in sickle cell disease in Saudi arab children in the Eastern province. ***Annals of Saudi Medicine*** 2002; 22:167-171.
2. Vichinsky EP, Styles LA, Colangelo LH, Wright EC, Castro O, Nikerrson B, and The Cooperative Study of Sickle Cell Disease. Acute chest syndrome in sickle cell disease: Clinical presentation and course. ***Blood*** 1997; 89 : 1787-1792.
3. Vichinsky EP, Neumayr LD, Earles N, Williams R, Lennette ET, Dean D et al. Causes and outcomes of the acute chest syndrome in sickle cell disease. ***N Engl J Med*** 2000; 342: 1855-1865.
4. Wayne AS, Kevy SV, Nathan DG. Transfusion management of sickle cell disease. ***Blood*** 1993; 81: 1109-1123.
5. Bernini JC, Rogers ZR, Sandler ES, Reisch JS, Quinn CT, Buchanan GR. Beneficial effect of intravenous Dexamethasone in children with mild to moderately severe acute chest syndrome complicating sickle cell disease. ***Blood*** 1998; 92: 3082-3089.

Spectrum of childhood poisoning in a tertiary center in the Eastern Saudi Arabia

Afrah Kashmar, *KFUF (Ped),*
Aftab S. Chishti, *MD, FAAP.*

Poisoning is a major and persistent cause of accidental morbidity in children worldwide despite being preventable. Fortunately, in most of the cases, toxic agent ingested has minimal significant effect. Although a lot of developments have been made in the prevention, diagnosis and treatment of pediatrics poisoning in the last 5 decades, yet accidental poisoning continues to burden the health care system worldwide and remains in one of the top 10 leading causes of death. There have been sparse data from the Kingdom of Saudi Arabia (KSA); therefore, we report a retrospective analysis of accidental poisoning in

Table 1 - Demographics of accidental poisoning.

Age (year)	Male	Female	Total
0-1	4	6	**10**
1-2	41	32	**73**
2-3	15	20	**35**
3-4	12	7	**19**
4-5	2	1	**3**
>5	5	6	**11**
Total	**79**	**72**	**151**

Table 2 - Medicines taken in poisoning episodes.

Poison agent	n of cases	(%)
Antihistamine	21	(14)
Paracetamol	16	(10.5)
Bronchodilators	11	(7.25)
Anticonvulsants	11	(7.25)
Non-steroidal anti-inflammatory drug	9	(6)
Antibiotics	5	(3.3)
Antihypertensives	5	(3.3)
Multivitamins	5	(3.3)
Cardiac drugs	3	(2)
Iron	3	(2)
Anticoagulants	3	(2)
Diuretics	2	(1.3)
Antithyroids	2	(1.3)
Antiemetics	2	(1.3)
Others	11	(7.25)
Total	**109**	**(72)**

Table 3 - Household chemicals taken in poisoning episodes.

Poison agent	n of cases	(%)
Chlorox (Sodium hypochlorite)	11	(7.25)
Drain opener	8	(5.25)
Rat poison	5	(3.3)
Hydrocarbons	3	(2)
Organophosphates	2	(1.3)
Detergents	1	(0.7)
Lamp oil	1	(0.7)
Miscellaneous	8	(5.25)
Total	**39**	**(26)**

childhood from a large tertiary care center in the Eastern Province of Saudi-Arabia. We performed a retrospective chart review of all the poisoning cases of children <13 years admitted to the Pediatric Ward of King Fahd Hospital of the University (KFHU) in Al-Khobar, KSA from January 1998 to December 2002.

Over the 5 years study period, 155 cases of accidental poisoning were admitted to the pediatric ward of KFHU from the accident and emergency department, with a yearly frequency of approximately 31 cases per year (ranging 16-47), accounting for 2.3% of total pediatric medical admissions. Unfortunately, the data from 4 of the 155 charts were excluded from the analysis due to its incompleteness. Most of the cases were admitted during spring season in the months of March to May (35%). Most of the patients were Saudis (84%). There was no difference based on gender as 79 (52%) were males and 72 (48%) were females. **Table 1** shows age and gender distribution of the total ingestions, 83 of them (53%) were in <2 years, 54 (35%) were in 2-4 years and 14 (9%) were in >4 years. The mean hospital stay was 1.4 days with a range of 1-7days, and 29 (19%) patients needed pediatric intensive care admission. In these patients, the offending agents were mostly anti-epileptics, anti-psychotics or caustic chemicals. There was no specific seasonal predilection noted although maximum cases were noted in spring months. The most common medications involved in accidental poisoning were antihistamines (14%) followed closely by acetaminophen (10%) and the most common household product involved was Clorox or sodium hypochlorite (7%) (**Table 2 & 3**). In the present study it is shown that the poisoning is becoming less frequent reason for hospitalization in pediatric age group (2.25%) than reported in previous studies from the KSA and outside.[1] There was no difference in incidence on the basis of gender but the age group 1-4 years was the commonly involved in accidental poisoning.[1,2] This is the age where children show great curiosity, impulsiveness and, are at their oral stage of psychological development, as they want to explore everything by ingesting it. Medications remain to be the more common agent (72%) of poisoning than the household agents (26%). Unfortunately, the general public in our part of the world still lacks the knowledge on the proper and safe storage of medications. In addition, even the prescription medications are still available over the counter. We reported that Chlorox was the most common among the household chemicals. We had only one solitary case of kerosene ingestion unlike the previous reports from the KSA[1] probably as long as it is not used as

source of household fuel anymore. The similar trend or changing pattern was also noted in the study by Singh et al[3] in the Northern India. There is still strong need for improving the overall drug and medication dispensing services and to enhance the awareness of the lay person's ability to identify the potential hazards of improper medication storage and use. The data are clear that proper storage in terms of location and the child resistant containers definitely reduce the accidental ingestion of medication.[4,5] We are still lagging behind on these measures from the Western societies. Definitely, the availability of poison control centers is a step in the positive direction but their presence need to be publicized more effectively; therefore, they can be utilized more efficiently. It is shown that they can reduce the load on the hospitals by proper and timely advice to the parents and prevent undue panic and expenses.[6]

In conclusion, it cannot be emphasized enough that the reduction in the incidence of the childhood poisoning cannot be achieved unless the parents and the caregivers are prepared for the behavioral change in storing household products and medications safely in their appropriate containers. The key lies with the overall improvement in the education of the community as a whole.

Received 31st May 2003. Accepted for publication in final form 2nd September 2003.

From the Department of Pediatrics, King Fahd Hospital of the University, Dammam, Kingdom of Saudi-Arabia. Address correspondence and reprint requests to Dr. Aftab S. Chishti, Assistant Professor, Department of Pediatrics, King Faisal University, PO Box 40241, Dammam 31952, Kingdom of Saudi Arabia. Tel. +966 (3) 8823518. Fax. +966 (3) 8873700. E-mail: achishti@hospital.kfu.edu.sa

References

1. Al-Sekait MA. Epidemiology of accidental poisoning of children in Riyadh, Saudi-Arabia. ***Annals of Saudi Medicine*** 1990; 10: 276-278.
2. Ho L, Heng JT, Lou J. Accidental ingestions in childhood. ***Singapore Med J*** 1998; 39: 5-8.
3. Singh S, Singhi S, Sood NK, Kumar L, Walia BN. Changing pattern of childhood poisoning (1970-1989): experience of a large north Indian hospital. ***Indian Pediatr*** 1995; 32: 331-336.
4. Shannon, M. Ingestion of toxic substances by Children. ***N Engl J Med*** 2000; 342: 186-191.
5. Rogers, GB. The Safety effects of child resistant packaging for oral prescription drugs. Two decades of experience. ***JAMA*** 1996; 275: 1661-1665.
6. Lovejoy FH, Robertson WO, Woolf AD. Poison Centers, poison prevention, and the Pediatrician. ***Pediatrics*** 1994; 94: 220-224.

C-Reactive protein in diabetes mellitus and hyperlipidemia

Samir M. Khoja, *M.Med.Sci, D.Phil,*
Fahd A. Alabbassi, *BSc, MSc,*
Abuelgassim O. Abuelgassim, *BSc, PhD,*
Omar A. Al-Bar, *BSc, PhD.*

The aim of the present investigation is to measure serum C-reactive protein (CRP) in patients with diabetes mellitus and hyperlipidemia in order to find out an association between CRP and these diseases. This study was carried out on type 2 diabetic and hyperlipidemic patients attending Diabetic Care Center in Jeddah, Kingdom of Saudi Arabia (KSA) from October 2001 through to October 2002. Concentrations of serum CRP and lipids were determined in 143 male and female diabetic patients, 119 male and female hyperlipidemic patients and 95 male and female normal controls. Different parameters were measured according to standardized enzymatic assay methods. Serum CRP concentrations of diabetic and hyperlipidemic male patients were significantly increased ($p<0.001$), so did the concentrations of total cholesterol and triacylglycerol. The concentrations of serum CRP were significantly increased ($p<0.001$) in diabetic and hyperlipidemic female patients. This increase coincides with a significant increase ($p<0.001$) of total cholesterol, triacylglycerol and low density lipoprotein (LDL)-cholesterol. Positive associations were obtained between CRP concentrations and triacylglycerol, and LDL-cholesterol concentration. The present results confirm the view that the higher CRP concentration in serum could reflect the inflammatory component of the atherosclerotic process that is so prevalent among patients with diabetes and hyperlipidemia.

C-reactive protein (CRP) is a non-glycosylated polymeric protein consisting of 5 identical subunits. In recent years there has been a marked increase in interest in the relationship between CRP and the risk of cardiovascular disease. There are now numerous prospective studies which show that elevated baseline concentrations of CRP are correlated with a higher risk of future cardiovascular events.[1] C-reactive protein levels have been considered to reflect the extent of inflammatory reactions in the atherosclerotic vessels.[2] Thus, by virtue of its acute phase behavior, CRP is a marker for severity and progression of atherosclerotic processes in the vessels.[2] Although inflammation is hypothesized to play a role in the development of type 2 diabetes, clinical data addressing this issue are limited. Pradhan et al[3] indicated that a possible role for