

145 (64.7%) were coins, 16 (7.1%) were fish bones, and 11 (4.9%) were meat bolus (**Table 1**). The vast majority 10 (90.9%) of the patients with meat bolus were men aged 55 years and above. The patients with safety pins 5 (2.2%) were women all below 16 years old. The highest incidence of FBs (68.6%) was found among children under 5 years old. This age group is curious and tries to interact with the surroundings. Less than a half of the patients (43%) presented at the same day of FB aspiration. The reason behind this delay could be due to unawareness about the problem, living in remote, mountainous and inaccessible areas, or could be due to seeking traditional healing solutions. In our study, 10 (4.4%) of the patients presented with dyspnea and 6 (2.7%) with cough. Therefore, esophageal FBs patients may present in respiratory distress and may be treated for bronchitis or asthma. Thus, it is important to differentiate dyspnea and cough due to respiratory pathology and esophageal FBs impaction. Asymptomatic patients with esophageal FBs were (7.1%). Some of these FBs have been long standing, these patients may have grown accustomed to the presence of the FBs, and the symptoms are related to the size, kind, and location of the FBs in the esophagus. A high percentage of coins (91%) were removed under direct laryngoscopic vision and McGill forceps without general anesthesia. In our experience, this technique is safe and effective for removing cervical esophageal coins. For other esophageal FBs, we used rigid esophagoscopy. It was successful in 93.5%, and we found that rigid esophagoscopy is safe, quick, easy, and with minimum trauma. We had only 4 (1.8%) complications due to the trial for removing sharp impacted FBs by inadequately experienced hands. Coins (145 [64.7%]) were the most common FBs in the esophagus and this finding is comparable with other studies in Jordan,⁷ while higher than the proportions reported from Saudi Arabia⁶ and Oman.⁵

In our study 5 (2.2%) FBs were safety pins and swallowed by women. In our country, females practice the wearing of the scarf, and they hold the safety pin between their teeth or lips and, can unintentionally swallow the pin.

Received 26th March 2005. Accepted for publication in final form 10th July 2005.

From the Department of Special Surgery (Al-Mahbashi) and the Department of Community Medicine (Raja'a), Faculty of Medicine and Health Sciences, Sana'a University, Republic of Yemen. Address correspondence and reprint requests to Dr. Yahia A. Raja'a, PO Box 2058, Sana'a, Republic of Yemen. Tel. +967 (7) 1687682. Fax. +967 (7) 1617759. E-mail: yahiarajaa@yahoo.com

References

1. Mahafza T, Btieha A, Subh M, Khrais T. Esophageal foreign bodies: a Jordanian experience. *Int J Pediatr Otorhinolaryngol* 2002; 64: 225-227.

2. Shankar BR, Yachha SK, Sharma BC, Singh B, Mahant TS, Kapoor VK. Retained esophageal foreign bodies in children. *Pediatr Surg Int* 1996; 11: 544-546.
3. Gilchrist BF, Valerie EP, Nguyen M, Coren C, Klotz D, Ramenofsky ML. Pearl and Perils in the management of prolonged peculiar, penetrating esophageal foreign bodies in children. *J Pediatr Surg* 1997; 32: 1429-1431.
4. Kruk-Zagajewska A, Szymeja Z, Woirowicz J, Wierzbicka M, Piatkowski K. Foreign bodies in the esophagus. *Otolaryngol Pol* 1999; 53: 283-288.
5. Murty PSN, Ingle VS, Ramakrishna S, Shah FA, Philip V. Foreign bodies in the upper digestive tract. *SQU J Sci Res: Medical Science* 2002; 3: 117-120.
6. Ashoor AA, Al-Momen A. Foreign bodies of the esophagus: A two-year prospective study. *Ann Saudi Med* 2000; 20: 173-175.
7. Al-Qudah A, Daradkeh S, Abu-Khalaf M. Esophageal foreign bodies. *Eur J Cardiothorac Surg* 1998; 13: 494-499.

Mycobacterium tuberculosis and CD4+ T-lymphopenia. A grave combination

*Shahid Aziz, MBBS, MRCP (UK),
 Awadh R. Al-Anazi, MBBS, MD,
 Mogbil A. Al-Hedaihy, MBBS, FRCP (C),
 Hani A. Al-Shobaili, MBBS,
 Abdulkarim I. Al-Aska, FACHARZT.*

Tuberculosis (TB) is still a common health problem in many parts of the world including Saudi Arabia. According to some of the estimates approximately one third of the world's population is infected with mycobacterium tuberculosis (MTB), with 6-8 million new active cases each year, and accounting for 2-3 million deaths each year. Infection with MTB has protean manifestation, with single or multiple organ system involvement. Hematological abnormalities with the disease have been enlightened by many studies. Correction of these abnormalities with initiation of treatment indicates a good response of the disease.¹ In the present study, we looked at the occurrence of CD4+ T-lymphopenia with active MTB infection.

We included in this study, all patients admitted in the Department of Medicine, under care of Infectious Diseases Unit, at King Khalid University Hospital, Riyadh, Kingdom of Saudi Arabia from July 2002 to June 2003 with culture proven MTB infection, and CD4+ T-cell count <300 x 10⁶/L. We tested the human immunodeficiency virus (HIV) status of all the patients by western blot method and excluded those with a positive result. In addition, the patients on immunosuppressive drugs for any reason were excluded from the study. A tuberculin skin test (TST) was carried out for all the patients

by injecting 5 units of purified protein derivative (PPD) intradermally and read after 48 hours, an induration size of 5 mm or more was considered as positive. All the anti-TB drugs (calculated as per body weight of the patient) were given orally (PO) once daily (OD). For the initial 2 months of the treatment, all the patients received 4 drugs; namely, isoniazid (INH), rifampicin (RIF), ethambutol (ETH), and pyrazinamide (PZA) and later on, INH and RIF to complete their treatment. The CD4+ T-cell count was tested before starting anti TB drugs and at 3 and 6 months after starting the treatment. All the patients were followed in the infectious diseases clinic for a minimum of one year. The total number of the patients admitted with active MTB infection over this time was 31, and 6 of them were found to have CD4+ T-cell count $<300 \times 10^6/L$, along with negative HIV serology, giving an incidence of 19% for this severe CD4+ T-lymphopenia with active MTB infection. The patient information and treatment outcome is given in **Table 1**. The CD4+ T-cells are considered to be the primary T-cell subset responsible for regulating the immune response to MTB. The HIV pandemic provides direct evidence that loss of CD4+ T-cell number and function resulted in progressive primary infection, reactivation of endogenous MTB, and enhanced susceptibility to re-infection. Many studies in mice also have confirmed this protective role of CD4+ T-cells against MTB. However, active MTB infection can lead to CD4+ T-lymphopenia with counts as low as $300 \times 10^6/L$, the incidence of which has been reported by Kony et al² as 14.4%, whereas, according to our results it is 19%. The mean CD4+ T-cell count in our patients at the time of presentation was $139.5 \times 10^6/L$. The patients, that expired had a CD4+ T-cell count of $<150 \times 10^6/L$, whereas, those who survived had CD4+ T-cell count

$>150 \times 10^6/L$. Our results further support the previous findings of Pilheu et al,³ showing high mortality in patients with CD4+ T-cell count $<300 \times 10^6/L$, and severe pulmonary TB. We further suggest that, those with CD4+T-cell count $<150 \times 10^6/L$ are likely to have even higher mortality. All the patients who survived in our study started to recover their CD4+ T-cell count by 3 months, which returned to normal with in 6 months. This finding shows that the CD4+ T-lymphopenia produced by active MTB infection is reversible with the effective treatment of the disease. The mean age of the patients in our study was 37.1 years, and none of them had any co-morbid systemic disease. Although military TB is usually considered to be the disease of the elderly (or very young), and immunocompromised patients, the findings of our study suggest that, severe mycobacterial disease (pulmonary or disseminated) along with the MTB induced CD4+ T-lymphopenia is more common in relatively young patients. This might be explained by the fact that young, genetically susceptible, patients probably mount more vigorous immunological response to the infection with MTB, leading in turn to more severe depletion of antigen responsive T-cells (due to the phenomenon of compartmentalization and selective increased programmed cell death of activated T-cells),^{4,5} and this leads to the spread of the disease (intra- or extra-pulmonary), due to the lack of production of interferon-gamma and interleukin-2 from CD4+ T-cells. These are the cytokines known for helping in granuloma formation and thus limiting spread of the disease. The mean time taken for presentation from the start of symptoms in our patients was 5.5 months. This long period of active infection before seeking medical advice also, might have contributed to the development of CD4+ T-lymphopenia in

Table 1 - Patient information and treatment outcome.

Patient	Age in years	Gender	Time of first presentation from the start of symptoms	Percent body weight lost before presentation	TB site	Time taken for defervescence (in days) after starting anti-TB drugs	CD4+ T-cell count $\times 10^6/L$ before and after starting anti-TB drugs			Outcome
							0 month	3 months	6 months	
1	22	F	6 months	26.6	P	-	20	-	-	died
2	33	M	8 months	24.2	P + EP	60	165	552	839	recovered
3	70	M	3 months	22.2	P + EP	-	148	-	-	died
4	26	F	5 months	16.6	P + EP	25	180	630	910	recovered
5	35	F	4 months	20	P + EP	30	151	592	876	recovered
6	37	M	7 months	28.5	EP	22	173	436	950	recovered
Mean \pm SD	37.1 \pm 17	-	5.5 \pm 1.8 months	-	-	34.2 \pm 17.4	139.5 \pm 59.8	552.5 \pm 83.9	893.7 \pm 47.4	-

F - female, M - male, TB - tuberculosis, SD - standard deviation, P -pulmonary, EP - extra-pulmonary

genetically predisposed individuals. The mean time taken for defervescence in our patients was 34.2 days after starting anti TB drugs; this increased the length of hospital stay and caused additional costs to the health care system. These findings further emphasize the need for early detection, and treatment of MTB infection. More than 80% of our patients lost 20% or more of their body weight at the time of presentation and all of them were non-reactive to TST. These findings are in agreement with the previous results of Pilheu et al.,³ who noted 20% or more of body weight reduction at the time of presentation, in a study of 17 patients with severe pulmonary TB. Our results are also in agreement with the results of Kony et al.,² reporting that patients with CD4+ T-cells count <300 x 10⁶/L are less likely to react to TST, and have higher frequency of extra-pulmonary involvement, in the form of pleural effusion, lymphadenopathy, miliary disease, and oral candidiasis.

In conclusion, the results of our study show that, active MTB infection can induce profound CD4+ T-lymphopenia, as seen in patients with HIV infection, however, the defect is reversible with the effective treatment of MTB infection. Further, studies are probably needed with a larger patient number to develop general guidelines for the management of this sub-group of patients, having this grave combination of active MTB infection, and CD4+ T-lymphopenia. It is worth reminding that, in patients found to have lymphopenia with MTB infection; one should carefully investigate for the presence of disseminated disease and check their CD4+ T-cell count. Patients with counts <300 x 10⁶/L have poor prognosis, particularly those with CD4+ T-cell count <150 x 10⁶/L have the highest mortality. Such patients should be managed in the setting of intensive care unit during the initial phase of their treatment.

Received 30th April 2005. Accepted for publication in final form 18th June 2005.

From the Department of Medicine, King Khalid University Hospital, King Saud University, Riyadh, Kingdom of Saudi Arabia. Address correspondence and reprint requests to Dr. Shahid Aziz, Senior Registrar, Department of Medicine (38), King Khalid University Hospital, PO Box 7805, Riyadh 11472, Kingdom of Saudi Arabia. Tel. +966 (1) 4672472. Fax. +966 (1) 4670999. E-mail: shahidaziz68@hotmail.com

References

1. Morris CD, Bird AR, Nell H. The hematological and biochemical changes in severe pulmonary tuberculosis. *Q J Med* 1989; 73: 1151-1159.
2. Kony SJ, Hane AA, Larouze B, Samb A, Cissoko S, Saw PS, et al. Tuberculosis associated severe CD4+ T-lymphocytopenia in HIV-seronegative patients from Dakar. *J Infect* 2000; 41: 167-171.

3. Pilheu JA, De Salvo MC, Gonzalez J, Rey D, Elias MC, Ruppel MC. CD4+ T-lymphocytopenia in severe pulmonary tuberculosis without evidence of human immunodeficiency virus infection. *Int J Tuberc Lung Dis* 1997; 1: 422-425.
4. San JME, Valdes L, Saavedra MJ, De Vega JM, Alvarez D, Vinuela J, et al. Lymphocyte populations in tuberculous pleural effusions. *Ann Clin Biochem* 1999; 36: 492-500.
5. Vanham G, Zitvogel L, Camobell R. Generalized immune activation in pulmonary tuberculosis: co-activation with HIV infection. *Clin Exp Immunol* 1996; 103: 30-34.

Management of difficult airway in a child with arthrogryposis multiplex congenita during general anesthesia

Remziye Sivaci, MD, Canan Balci, MD,
Gokhan Maralcan, MD, Ilhami Kuru, MD.

Arthrogryposis multiplex congenita (AMC) consists of complex congenital anomalies characterized with multiple contractures. The possibility of autosomal dominant inheritance with reduced penetrance is suggested for this apparently new syndrome.¹ This syndrome may include foot deformities such as pes equinovarus (PEV), and congenital convex pes valgus. There may be a variety of deformities of the knee: flexion deformity, genu recurvatum, and genu valgum. Hip deformities such as unilateral or bilateral dislocation contractures may also exist. Craniofacial abnormalities, multiple joint contractures, pulmonary hypoplasia, cryptorchidism, and unusual ophthalmological findings are the other characteristics of this syndrome. In some severely affected persons, the central nervous system may also be affected.² Arthrogryposis multiplex congenita, which is diagnosed at birth presents with multiple joint contractures. Anesthetic management of these patients requires special care. As this disease often progresses until dysfunction of multiple organ systems occur, it may have an impact on the anesthetic management. Difficult tracheal intubation may be encountered due to limited neck extension, inadequate mouth opening, and short epiglottis.³

A 3-year-old and 20 kg weight boy who was diagnosed as PEV was prepared for surgical correction of the deformity. During preoperative examination, there was no history of any congenital anomaly or marriage of close relationship. The other 2 brothers were healthy. His mother had history of non-specific febrile infection and had no treatment