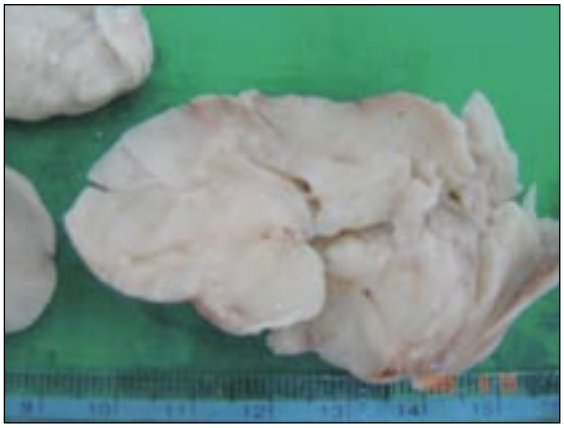

Figure 1. Whitish yellow, greasy, lobulated appearance of the infantile lipoma.

Figure 2. Lobules of lipoblasts, showing a plexiform vascular pattern.

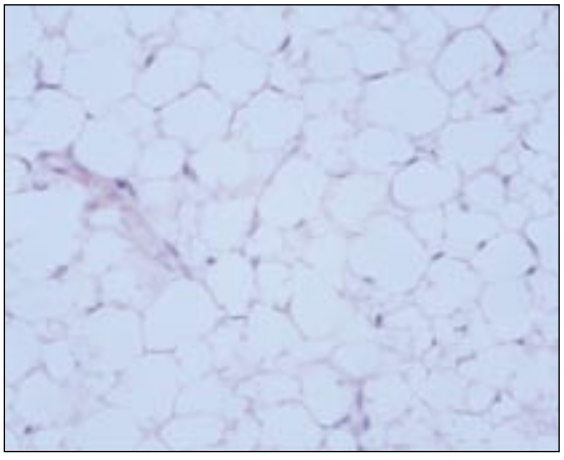

Figure 3. A high power view of the lipoblasts.

Lipoblastoma and infantile lipoma

To The Editor: A 1-year-old male Saudi child was admitted in the pediatric ward with a soft, mobile right axillary mass measuring 3×4 centimeters. No signs of inflammation were seen over the swelling, and no similar swelling was noticed on the child. His blood examination showed lymphocytosis, and the tuberculin test was negative. An excisional biopsy of the axillary mass was performed to rule out lymph node disorder. The specimen, sent to the histopathology lab in 10% buffered formalin, consisted of a fairly circumscribed, whitish yellow, lobulated fatty mass measuring 6×3×1 centimeters. The cut surface of the mass was whitish yellow, homogenous with lobulations (Figure 1). Touch imprint of the mass showed adipocytes and no lymphoid cells. Microscopic examination showed lobules of lipoblasts interspersed between spindle and stellate mesenchymal cells, and suspended in a myxoid strauma with a plexiform vascular pattern (Figure 2).

Lipoblastoma are benign mesenchymal tumor of embryonic white fat, with a postnatal spectrum of differentiation, ranging from prelipoblasts and mature adipocytes. The term infantile lipoma was suggested, as it reflects many of the tumor characteristics, such as early occurrence, an ability to mature into a simple lipoma, and a benign course, as the word blastoma is associated with a more malignant nature.[5] Moreover, the microscopic arborising vascular pattern, myxoid strauma, and lipoblasts, in a case of lipoblastoma, resembles myxoid liposarcoma—a malignant tumor. However, liposarcomas rarely if ever occur in infants and children, and lack the lobular organization of lipoblasts seen in lipoblastoma.[6] Lipoblastoma also lack giant cells and pleomorphic nuclei.[7] Ultrastructurally, cells varying in appearance from primitive mesenchymal cells to typical multivaculated lipoblasts are seen in lipoblastoma,[8,9,10] and these cells are very similar to cells in myxoid liposarcoma.[11]

Cytogenetic studies show that infantile lipoma is associated with rearrangements of 8q and trisomy of chromosome 8.[12] In contrast, the cytogenetics of myxoid liposarcoma are characterized by reciprocal translocation of t(12 :16) (q 13:9 11), which results in the rearrangement of the transcription factor gene CHOP, involved in the adipocyte differtiation.[13,14,15,17]

A pre-operative diagnostic test for lipoblastoma would include fine needle aspiration cytology.[15] An intra-operative touch imprint and frozen section can rule out other differential diagnosis. Excision biopsy is the treatment of choice and a recurrence rate of 14% to 25 % are reported from incomplete removal.[16]

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Patterns of mortality in adult sickle cell disease in Al-Hasa region of Saudi Arabia

To the Editor: Sickle cell disease (SCD) is an autosomal recessive disorder characterized by production of abnormal hemoglobin S and is associated with higher morbidity and mortality because of anemia, susceptibility to infections and multiple organ dysfunctions. Lately, due to better understanding of SCD pathophysiology and better management of the complications, the life expectancy has significantly improved.[1-3] The nature of SCD in the Eastern province of Saudi Arabia is benign because of high Hb F, concurrent inheritance of alpha-thalassemia (which may modify the severity of clinical course) and with preserved splenic functions in the majority of the patients until adulthood. This reduces the risk of severe pnemococcal infection related mortality in this region, unlike most African or American patients.[4] In the Eastern province SCD is responsible for 15.7% to 21.1% of hospital admissions to pediatric and medicine wards.[5]

The present retrospective study was undertaken at the 502-bed King Fahad Hospital and Tertiary Care Center, Al-Hofuf, Eastern province of Saudi Arabia. Records of all the patients having SCD, admitted to the Medicine Department during the period of January 1997 to December 2005 were reviewed. All these patients were confirmed as having SCD on the basis of hemoglobin electrophoresis results, other supportive hematological parameters and past clinical history. The cause of death was established on the basis of presentation of the patient to the emergency department, the course of illness during hospitalization, supportive laboratory evidence and clinical management of these patients.

Of 10 461 admissions of SCD adult patients to the medical department during this period, 77 (0.73%) expired. Of those, 43 (55.8%) were male and 34 (44.2%) female, with a male female ratio of 1.3:1. The mean age of the male patients was 30±14 years (range, 16-67 years) and of female patients 27±13 years (range, 14-67 years). The overall mortality of 0.73% in the present study was much lower than that reported from other parts of the world.[6] The majority of the deaths (51.9%) occurred among patients in the 20 to 30 years of age group and 20.7% were younger than 20 years of age. The majority of the patients had high Hb F (20% to 24%), a steady status leukocytes below 15 000/μL and no evidence

Table 1. Causes of mortality among sickle-cell disease patients hospitalized during 1997-2005.

Causes of mortality	Male	Female	Total	Percentage
Acute chest syndrome	13	9	22	28.5
Salmonella septicemia	7	2	9	11.6
Other Sepsis	4	4	8	10.3
Multiorgan failure	5	1	6	7.7
Hepatic crisis	2	4	6	7.7
Central nervous system	2	3	5	6.4
Delay hemolytic reaction	0	4	4	5.1
Pulmonary hypertension	2	1	3	3.8
Malignancy	1	2	3	3.8
Chronic renal failure	2	0	2	2.5
Other medical problems	1	1	2	2.5
Unknown cause	4	3	7	9.0
Total	43	34	77	100%