Correspondence

Septic arthritis in a newborn

A preterm newborn infant (36 weeks, weight 2500 g) developed fever, excessive crying and decreased movements of the left lower limb on day 10 of life. The infant was admitted in a primary care hospital for suspected sepsis. However, with fever persisting and an evolving left knee swelling, the infant was referred to us while on day 5 of intravenous antibiotics. We found the newborn to be febrile, the left knee to be affected with frank signs of septic arthritis and high inflammatory markers (C-reactive protein [CRP] 58 mg/dl). A joint effusion on an ultrasound and X-ray suggested of periosteal reaction around the distal femur extending to the distal femoral physis with a partial loss of the lateral femoral condyle (Fig. 1a). A surgical debridement drained 20 ml of pus. Intraoperatively, a part of the lateral femoral condyle was found to be de-vitalized and lying separate from the rest of the femur (Fig. 1b). The infant improved after debridement of the joint, as shown[1] by decreased inflammatory markers (CRP 3.8 mg/dl within 3 days of surgery). The infant received amoxicillin–clavulanate for 4 weeks based on the sensitivity pattern of *Staphylococcus aureus* that grew on blood culture (methicillin-sensitive).

Devastating joint damage in neonatal septic arthritis continues to be a concern in low-resource settings.[1–3] It is complicated by numerous uncertainties and knowledge gaps. First, a major burden of preterm and low-birth weight newborns in low- to middle-income settings are predisposed to septic arthritis; an inherent risk in infancy.[2] Second, subtle signs of joint inflammation evoke less suspicion in the newborn nursery, especially in preterm infants, until optimal inflammatory signs appear. Screening of a joint by an ultrasound and/or X-ray is less informative as an abnormality is indicative of an evolved disease and does not rule out complications *per se*; an abnormal joint ultrasound was noted in only 60% of septic arthritis in one report.[2,3] So, a 'definitive indication to intervene' based on clinical or radiological tests is often late and suboptimal to prevent joint complications as seen in our patient. Third, recent reports suggest continued controversy regarding timing and outcome of surgery. Surgical drainage of the joint is the mainstay of treatment in neonatal septic arthritis as it decreases the bacterial load and intra-articular pressure, thereby decreasing the risk of complications. However, there is no consensus on whether all patients with septic arthritis must undergo surgery.[3] There is agreement on unfavourable joint outcomes in the presence of an abnormal X-ray or substantial joint effusion on ultrasound at admission. The complication of unilateral femoral condyle loss is postulated to cause vascular ischaemia due to the compartment syndrome in the presence of infrapatellar synovial septae.[4,5] Disturbance in the growth of long bones resulting in angular deformity or limb length discrepancy is a sequelae of septic arthritis in infancy.[5–10] Such an event does not have an obvious solution for every affected child and attempts must be made to avoid this complication. Clinicians caring for a newborn must be aware of subtle signs of neonatal septic arthritis and seek an early opinion from their orthopaedic colleagues.

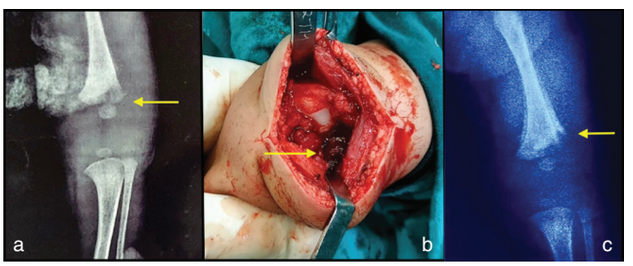

FIG 1. (a) Preoperative antero-posterior X-ray of the left knee showing a lucent zone (yellow arrow); (b) intraoperative image demonstrates an intact medial condyle and a deficient lateral femoral condyle (yellow arrow); (c) postoperative (10 days) antero-posterior X-ray showing the now absent lateral femoral condyle (yellow arrow)

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Prevalence of non-alcoholic fatty liver disease (NAFLD) among adults in urban Goa

Once considered innocuous, non-alcoholic fatty liver disease (NAFLD) has emerged as the leading cause of chronic liver disease.[1] It is not attributable to excessive alcohol consumption, drugs, toxins, infectious diseases or other identifiable causes.[2] NAFLD is now the most common liver disease in the western world.[3] It has a pooled global prevalence of 25.4%, with the highest prevalence in the Middle East and South America (around 30%) and the lowest in

Africa (~13%).[4–6] It is closely associated with metabolic syndrome and its components—hypertension, dyslipidaemia, hyperglycaemia, obesity and insulin resistance.[7] Hepatocellular carcinoma and cirrhosis are the end stages of progression of NAFLD.[5,8] Individuals with NAFLD are at increased risk of liver-related, cardiovascular and all-cause mortality.[9] NAFLD has also been associated with type 2 diabetes mellitus, atherosclerosis, cardiovascular disease, chronic kidney disease, polycystic ovarian syndrome, obstructive sleep apnoea, etc.[10]

In India, the prevalence of NAFLD has been reported to be 9% to 53%.[11–13] In the state of Goa, while the focus has been on the health effects of high level of alcohol consumption, no attention has been given to NAFLD. Hence, we decided to estimate the prevalence of NAFLD as well as associated factors in adults aged ≥30 years in an urban community in Goa.

We did a community-based cross-sectional study in an urban area in Goa, India. Individuals ≥30 years of age, with no alcohol use and living in the area for more than 6 months were recruited in the study. Pregnant and lactating women and individuals with any amount of alcohol intake, individuals on hepatic steatogenic medication in past one year were excluded from the study. Those testing positive for hepatitis B or C among those diagnosed with fatty liver on ultrasound were also excluded.

Considering the NAFLD prevalence to be 16.6[14] in a study in an urban area in western India and absolute precision as 5%, we calculated the sample size to be 206. Taking into consideration non-responses and refusals to undergo ultrasound, we decided to recruit 236 participants in the study. The systematic random sampling technique was used to recruit the study subjects.

Data were collected using a structured questionnaire. For clinical examination and anthropometry, digital sphygmomanometer, non-stretchable measuring tape and portable weighing scale were used. For investigations, ultrasonography (Siemens Acuson S2000 with 6C1 HD and 4V1 transducer) was used by a trained radiologist for the diagnosis of NAFLD. The Hepatic Steatosis Ultrasound Images Assessment manual[15] was used to assess the steatosis in the participants. NAFLD was graded into mild, moderate and severe NAFLD.[15] Participants found to have fatty liver by ultrasound were tested for hepatitis B surface antigen and hepatitis C core antigen. Hepatitis C was tested with a rapid test kit (SD Bioline HCV, Abbot Laboratories) with manufacturer reported sensitivity and specificity of 99.3% and 100%, respectively. Calorie intake was measured by 24-hour dietary recall. Physical activity was measured by the Global Physical Activity questionnaire. For waist:hip ratio (WHR) we used cut-off of >0.9 for men and 0.85 for women. For biochemical parameters, we used the following cut-offs: 126 mg/dl for fasting blood sugar level, 200 mg/dl for serum cholesterol, 150 mg/dl for serum triglyceride, 1.2 mg/dl for serum bilirubin, 40 i.u./L for aspartate aminotransferase (AST) and 56 i.u./L for alanine aminotransferase (ALT). We categorized patients based on body mass index (BMI) into three categories as per Indian cut-offs: lean (18.5–22.9 kg/m^2), overweight (23.0–24.9 kg/m^2) and obese (≥25 kg/m^2).

Statistical analysis was done using IBM SPSS Statistics for Windows (Version 24.0. Armonk, NY: IBM Corp). NAFLD prevalence with 95% CI was calculated. Prevalence odds ratio with 95% CI was calculated for various associated factors. Chi-square was the test of significance used and a p value <0.05 was considered statistically significant. Ethics approval for the study was obtained from the Institutional Ethics Committee and written informed consent was obtained from all the participants recruited in the study.

Of the 278 adults approached for the study, 42 participants reported either alcohol intake or intake of hepatic steatogenic drugs and were excluded. Thus, a total of 236 participants were recruited in the study and interviewed. Fifteen participants did not report for laboratory investigations and 11 did not report for ultrasonography. Eventually complete data were available for 210 study participants and were included in the final analysis. Women comprised 62.9% of the study participants. The majority of participants were Hindus (56.7%) followed by Christians (31.4%) and Muslims (11.9%). Most participants belonged to socioeconomic class II (43.8%) by BG Prasad classification, closely followed by socioeconomic class III (37.6%). Around 11.9% belonged to class IV, 2.4% to socioeconomic class V and 4.3% to socioeconomic class I.

The overall prevalence of NAFLD was 34.8% (95% CI 28.5%–41.4%). The prevalence among men (46.2%; 95% CI 35.3%–57.3%) was significantly higher (p=0.008) than among women (28%; 95% CI 20.8%–36.1%). As far as grading of NAFLD was concerned, 57.5% were mild, 38.4% moderate and 4.1% were severe.

Individuals >35 years of age had higher prevalence of NAFLD (39.4%) compared to those ≤35 years (6.7%). The observed difference in prevalence between the different age groups was statistically significant (p=0.006). The highest prevalence was seen among Hindus (38.7%) followed by Christians (36.4%) and the lowest among Muslims (12%). This difference was found to be statistically significant (p=0.037). Higher prevalence of NAFLD was observed in individuals belonging to higher socioeconomic classes I (44.4%) and II (48.9%). Prevalence was low in the lower socioeconomic classes IV (4.0%) and V (20%).

Individuals consuming a non-vegetarian diet were twice as likely to have NAFLD compared to vegetarians (OR 2.83; 95% CI 1.28–6.24) and this difference in NAFLD prevalence between vegetarians and non-vegetarians was statistically significant (p=0.008). The prevalence of NAFLD was significantly higher (p=0.0001) among people with diabetes (52.1%) compared to those without diabetes (25%). We found a similar increased risk for NAFLD among hypertensive individuals (Table I).

Using high fasting blood sugar (≥126 mg/dl) as a marker for poor glycaemic control, we found a higher prevalence of NAFLD (59.4%) among those with high fasting blood sugar compared to those with normal blood sugar (40.6%). We also found significant association of high cholesterol level (≥200 mg/dl) and high triglyceride levels (≥150 mg/dl) with NAFLD with individuals with high cholesterol 3.34 times (OR 3.34; 95% CI 1.84–6.09) as likely and individuals with high triglyceride 2.22 times (OR 2.22; 95% CI 1.17–4.24) as likely to have NAFLD compared to those with normal levels of these two biochemical markers (Table I).

We found no association between physical activity as measured by the Global Physical Activity Questionnaire and NAFLD (p=0.35) but BMI was significantly associated with NAFLD (p=0.001). WHR was also significantly associated with NAFLD (p=0.003). The mean values of BMI, fasting blood sugar level, total cholesterol, serum triglyceride, serum bilirubin, AST and ALT were significantly higher in subjects with NAFLD compared to those without NAFLD.

Binary logistic regression analysis by the forward stepwise Wald method identified only WHR, fasting blood sugar level and total cholesterol as significantly associated with NAFLD. The model correctly classified 75.2% of the cases. The logistic regression model was statistically significant (p<0.001) and Nagelkerke R^2 was 0.353 indicating the model explained 35.3% of the variability (Table II).

The prevalence of NAFLD was found to be 34.8% (95% CI 28.5%–41.4%). A meta-analysis of 237 studies from Asia[16] for the period 1999 to 2019 reported overall prevalence regardless of diagnostic method as 29.62% (95% CI 28.13–31.15). The pooled prevalence for India[16] was reported to be 32.74% (95% CI 13.89–59.49). Mohan *et al*.[17] reported an NAFLD prevalence of 32% in their study in Chennai, India. While Amarapurkar *et al*.[14] reported a prevalence of 16.6% in an urban population in Mumbai, India.

The prevalence of NAFLD was significantly higher among men than women (46.2% *v*. 28.0%). Mohan *et al*.[17] reported a higher prevalence among men (35.1%) compared to women (29.1%).

TABLE I. Association of non-alcoholic fatty liver disease (NAFLD) with key risk variables in urban Goa

Variable	*n*	% prevalence of NAFLD (95% CI)	Prevalence odds ratio (95% CI)	Prevalence ratio (95% CI)	p value
Type of diet					
Non-vegetarian	162	39.5 (32.2–47.2)	2.83 (1.28–6.24)	2.12 (1.14–3.91)	0.008
Vegetarian	48	18.8 (9.6–31.6)	1 (reference)	1 (reference)	
Diabetes mellitus					
Present	73	52.1 (40.6–63.3)	3.16 (1.74–5.76)	2.04 (1.42–2.92)	0.0001
Absent	137	25.5 (18.8–33.4)	1 (reference)	1 (reference)	
Hypertension					
Present	54	55.6 (42.2–68.4)	3.28 (1.73–6.24)	2.02 (1.42–2.85)	0.0002
Absent	156	27.6 (20.9–34.9)	1 (reference)	1 (reference)	
Body mass index (kg/m^2)					
Lean (18.5–22.9)	43	14.0 (5.9–26.8)	1 (reference)	1 (reference)	0.001
Overweight (23–24.9)	51	31.4 (19.8–45.0)	2.82 (0.99–8.02)	2.25 (0.96–5.24)	
Obese (≥25)	116	44.0 (35.1–53.1)	4.84 (1.90–12.35)	3.15 (1.46–6.81)	
Waist:hip ratio					
High	130	48.5 (39.9–57.0)	6.58 (3.12–13.89)	3.88 (2.11–7.11)	0.000
Normal	80	12.5 (6.5–21.2)	1 (reference)	1 (reference)	
Fasting blood sugar (mg/dl)					
≥126	69	59.4 (47.6–70.5)	4.99 (2.68–9.29)	2.62 (1.82–3.76)	0.000
<126	141	22.7 (16.4–30.2)	1 (reference)	1 (reference)	
Total cholesterol (mg/dl)					
≥200	74	52.7 (41.3–63.9)	3.34 (1.84–6.09)	2.11 (1.47–3.03)	0.000
<200	136	25.0 (18.3–32.8)	1 (reference)	1 (reference)	
Serum triglycerides (mg/dl)					
≥150	51	49.0 (35.6–62.6)	2.22 (1.17–4.24)	1.62 (1.13–2.34)	0.014
<150	159	30.2 (34.3–52.6)	1 (reference)	1 (reference)	

TABLE II. Binary logistic regression analysis for factors associated with non-alcoholic fatty liver disease (Forward Stepwise Wald method)

Risk factor	Beta coefficient	SE	Wald	p value	Adjusted OR (95% CI)
Waist:hip ratio					
High	1.869	0.418	20.034	<0.001	6.48 (2.86–14.70)
Low					1 (ref)
Fasting blood sugar (mg/dl)					
≥126	1.271	0.346	13.461	<0.001	3.57 (1.81–7.03)
<126					1 (ref)
Total cholesterol (mg/dl)					
≥200	1.265	0.355	12.695	<0.001	3.54 (1.77–7.11)
<200					1 (ref)

Amarapurkar *et al*.[14] also reported a higher prevalence among men compared to women (24.6% *v*. 13.6%). Li *et al*.[16] in their meta-analysis of Asian studies also reported a higher prevalence among men compared to women (37.11% [95% CI 35.04–39.24] *v*. 22.67 [95% CI 20.61–24.88]). However, the relationship between gender and NAFLD is believed to be diametrically divided between either genders in several studies, some studies showing a preponderance of women while others show a preponderance of men in prevalence.[18]

Individuals ≥35 years of age had a significantly higher prevalence of NAFLD (39.4%) compared to those <35 years old (6.7%) and the prevalence increased with increasing age. NAFLD and ageing are believed to be strongly correlated and increasing age is considered robust epidemiological factor for NAFLD, non-alcoholic steato-hepatitis and fibrosis.[19] The common age of presentation of NAFLD in India has been reported to be between 30 and 50 years.[14,20,21] A higher prevalence of NAFLD was observed in individuals belonging to higher and middle socioeconomic classes. Singh *et al*.[22] in a hospital-based study reported that a majority of patients with NAFLD belonged to the middle-income group.

The prevalence of NAFLD was 52.1% among people with diabetes compared to 25% among those who did not have diabetes. Insulin resistance of varying degree is suggested to be the cellular level abnormality underlying NAFLD.[11] A strong association between type 2 diabetes mellitus and NAFLD has been documented.[17,23–26] A meta-analysis of 24 studies has reported the pooled prevalence of NAFLD to be 59.67% (95% CI 54.31–64.92) among people with diabetes.[7]

We found a significant increased risk for NAFLD among people who had hypertension and a similar association between hypertension and NAFLD was reported by Majumdar *et al*.[27] in their population-based study in Haryana (OR 2.7; 95% CI 1.4–5.3; p=0.003). We found a higher prevalence of NAFLD in individuals with fasting blood sugar ≥126 mg/dl (prevalence 59.4% *v*. 22.7%), total cholesterol ≥200 mg/dl (prevalence 52% *v*. 25%) and serum triglyceride ≥150 mg/dl (prevalence 49% *v*. 30.2%). Speliotes *et al*.[28] in their study observed that high fasting blood sugar was associated with NAFLD (OR 2.95; 95% CI 2.32–3.75; p=0.001). Significant association between high triglyceride level and NAFLD (OR 3.7; 95% CI 1.2–13.3) has been reported by Leite *et al*.[29] A similar association between high fasting blood sugar (OR 4.0; 95% CI 0.9–17.6, p=0.03), high cholesterol (OR 2.5; 95% CI 1.1–5.6; p=0.03) and high triglyceride level (OR 2.3; 95% CI 0.99–5.2; p=0.05) with NAFLD has been reported by Majumdar *et al*.[27]

BMI and WHR were found to be significantly associated with NAFLD. Several studies[17,25,26] have also reported significant association between higher BMI and NAFLD. There is believed to be a close pathogenic connection between obesity and NAFLD as patients with

NAFLD are often obese and obese people have a higher prevalence of NAFLD.[11]

Limitations: Our study participants were aged ≥30 years, so there is a possibility of overestimation of the prevalence. We did not exclude other aetiologies such as Wilson disease or autoimmune hepatitis. Due to issues with high-density lipoprotein (HDL) testing at the institutional laboratory, we were unable to study the association between decreased HDL level and NAFLD as well as metabolic syndrome and NAFLD.

Considering the high prevalence of NAFLD in urban Goa, there is a need to focus on this important but seemingly invisible chronic disease before it reaches epidemic proportions given the changing patterns in lifestyle, diet and increasing burden of other non-communicable diseases such as diabetes, obesity, hypertension and dyslipidaemia.

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