

confirmed and he was started on hydrocortisone and fludrocortisone. He had a corrective surgery and his stretched penile length post-operatively was 3.5cm.

Lutfallah et al,² recently revised the hormonal criteria of HSD3B2 deficiency, they showed that the baseline and the ACTH-stimulated $\Delta 5$ -17P levels, and the baseline and the ACTH-stimulated $\Delta 5$ -17P/F ratios have more accurate prediction of HSD3B2 deficiency. In their series, genotype proven children with ambiguous genitalia had baseline and ACTH-stimulated $\Delta 5$ -17P levels of >26.4 nmol/L (>12SD) and >165 nmol/L (>35SD) and baseline and ACTH-stimulated $\Delta 5$ -17P/F ratios of >94 (>15SD) and >216 (>23SD). For genotype proven adult males with ambiguous genitalia, the baseline and ACTH-stimulated $\Delta 5$ -17P levels were >159 nmol/L (>74SD) and >289 nmol/L (>21SD) and the baseline and ACTH-stimulated $\Delta 5$ -17P/F ratios were >1943 (>193SD) and >4010 (>221SD). For genotype normal females with hirsutism and menstrual disorder, baseline and ACTH-stimulated $\Delta 5$ -17P levels were <45 nmol/L (<16SD) and <150nmol/L (<12SD) and baseline and ACTH-stimulated $\Delta 5$ -17P/F ratios were <43 (<5SD) and <151 (<10SD). Our patients showed very high $\Delta 5$ -17P levels and $\Delta 5$ -17P/F ratios on ACTH stimulation test, which may represent a severe form of HSD3B2 deficiency, although they were not severely affected clinically.

The patients described in this report had neonatal ambiguous genitalia and salt losing secondary to HSD3B2 deficiency. However at puberty, they had normal development of the secondary sex characters with normal testosterone and estrogen levels. We believe that HSD3B2 deficient patients have normal *HSD3B1* activity, which is responsible for the extra-adrenal and extra-gonadal conversion of $\Delta 5$ -hydroxysteroid precursors into the corresponding $\Delta 4$ -ketosteroids. This peripheral *HSD3B* activity could explain why some patients with HSD3B2 deficiency will have normal puberty.³ Alos et al,⁴ reported a 46xy patient with neonatal ambiguous genitalia, who had normal masculinization at puberty, but the patient was azoospermic. However, other reports described normal paternity in affected males with proven HSD3B2 deficiency.⁵

Male children with HSD3B2 deficiency may present with micropenis secondary to androgen deficiency. We believe that patients should be raised according to their genetic sex. Male patients who presumed to have a testosterone responsive micropenis may need one or more courses of intramuscular testosterone injection to achieve an adult penile length within the functioning range.

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Risk factors for recurrent miscarriage in Sudanese women

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Recurrent miscarriage is defined as 3 or more consecutive miscarriages, its effects can be distressing and devastating. While the condition affects 1% of all women,¹ sporadic miscarriage is common, complicating 10-15% of all clinically diagnosed pregnancies.² This concludes that the statistical probability of having 3 consecutive miscarriages is 0.34%. Therefore, recurrent miscarriage is more complicated than a simple chance occurrence. Recurrent miscarriage is a multi-factorial condition, causes and associations include (chromosomal) genetic (thrombophilia), anatomical, endocrine and idiopathic factors.

While some causes of recurrent miscarriage remain difficult to treat such as, polycystic ovary syndrome and chromosomal abnormalities; the use of aspirin and heparin have proven to improve the outcome in cases of thrombophilia; namely, antiphospholipid (APL) syndrome.

Antiphospholipid syndrome is defined as the combination of adverse pregnancy outcome or tendency towards thrombosis with the presence of anticardiolipin (ACL) antibodies or lupus anticoagulant or both. There is a wide geographical and racial variation in the prevalence of APL antibodies and thrombophilia in general. Most of the studies that have been carried out in Europe and the United States of America, found the prevalence of APL antibodies to be around 15% among women with recurrent miscarriage, compared to 2% in the general population.³ The population in Sudan is multi-ethnic (including pure African and variable degrees of African-Arabic mix). There are significant associations between recurrent miscarriages and chromosomal abnormalities, uterine anatomical abnormalities, polycystic ovarian syndrome and thrombophilia. In one-third of the patients, there may not be any identifiable cause for the problem; however, most of these women will successfully carry pregnancies to term. Therefore, we are still far from fully understanding the causes and associations of this condition. To our knowledge, no study has been carried out to determine the association of recurrent miscarriage with APL antibodies in Sudanese women.

This is a case control study, carried out in Soba University Hospital and Khartoum Teaching Hospital (both hospitals are located in the capital of Sudan, Khartoum) between October 1999 and August 2001. Ethical approval was granted by the ethical committee, Faculty of Medicine, University of Khartoum. The inclusion criterion for the case group was the presence of at least 3 consecutive miscarriages at any age (52 patients); while the inclusion criteria for the control group was the presence of at least one or more successful pregnancy, with no history of recurrent miscarriage or any abnormal pregnancy outcome, such as intra-uterine fetal loss (50 participants). All participants gave informed consent before joining the study. We controlled for cofounders such as socio-economic class and occupation. The median age in the studied group was 27.7 years compared with 27.1 years in control group. The data were collected using a designed questionnaire. A 5 ml venous blood sample was taken from each patient, with consent, for enzyme-linked immunosorbent assay to check for ACL antibodies. Those who tested positive were asked to give another blood sample after 6 weeks to repeat the test (criteria for diagnosis). Only those who tested positive twice were considered as having APL antibodies and hence APL syndrome. All patients positive for APL antibodies and who have had recurrent miscarriages were offered treatment and follow up at the maternal fetal medicine unit in Soba University Hospital.

In this study, we investigated the association of recurrent miscarriage with different risk factors,

Table 1 - Risk factors for recurrent miscarriage.

Risk factors	Cases n=52	Control n=50	OR	CI
Positive ACL antibodies	10	1	1.9696	2.23-1.69
Age < 35 years	35	31	0.8905	0.95-0.83
Age ≥ 35 years	17	19	1.2299	1.32-1.13
Race-Afro-Arabic	40	43	0.7631	0.84-0.68
Race-pure-African	12	7	1.3105	1.44-1.18
History of contraceptive use	2	38	0.0625	0.11-0.01
BP > 140/90	2	1	1.3188	1.44-1.19
Blood group A	14	11	1.347	1.48-1.21
Blood group B	10	14	0.7783	0.86-0.69
Blood group O	25	22	1.0835	1.14-1.02
Blood group AB	3	3	0.9795	1.00-0.95
Rh-positive	47	46	0.9096	0.96-0.85
Rh-negative	5	4	1.0995	1.16-1.03

OR - odds ratio, CI - confidence interval, ACL - anticardiolipin, BP - blood pressure, Rh - rhesus.

which include ACL antibodies as the main treatable risk factor, with other hypothetical risk factors, age, ethnic group, blood pressure, blood group and Rhesus factor status (**Table 1**). To our knowledge, there are no data concerning this issue in the Sudanese literature. Seven hypothetical risk factors for recurrent miscarriage were tested in both cases, these included the presence of ACL antibodies (as a diagnostic marker for APL syndrome), age, ethnic origin, history of contraceptive used, blood pressure, blood group and Rhesus factor status. The prevalence of ACL antibodies was 19.3% in women with recurrent miscarriage compared to 2% in the control group, this reproduces the published findings of other studies.⁴ The presence of ACL antibodies clearly increases the risk of recurrent miscarriage (odds ratio (OR) 2, confidence interval 2.23-1.69). Women who are positive for ACL antibodies are twice more likely to experience recurrent miscarriage than those who are not. This justifies routine testing for ACL antibodies in all women with recurrent miscarriage. As this is in line with the literature from other parts of the world,⁵ one may conclude that Sudanese women (with their diverse ethnic origin) are no different to other homogeneous ethnic groups.

Blood pressure $\geq 140/90$ (OR = 1.3), age ≥ 35 years (OR = 1.2), pure African race (OR = 1.3) and group A blood (OR = 1.3), have all appeared to slightly increase the risk of recurrent miscarriage. As the study was very small, a larger study is needed to either confirm or refute any possible causal relationship.

Age < 35 years (OR = 0.89), Arabic race (OR = 0.7), history of contraceptive use (OR = 0.06), blood group other than group A (OR ranging from 0.78 to 1.08), and Rhesus factor positivity or negativity (OR = 0.90 and 1.09) are not associated with increased risk of recurrent miscarriage. Contrary to the belief of many Sudanese women, contraceptive use does not increase the risk of recurrent miscarriage.

The presence of ACL antibodies is a risk factor for recurrent miscarriage among Sudanese women (OR = 2). It is therefore, important to offer women with recurrent miscarriage investigation for APL syndrome. Ethnic origin seems to play little or no role in the prevalence of APL syndrome. Most of the other risk factors did not seem to increase the risk of recurrent miscarriage significantly, however, more studies are needed to evaluate some risk factors that were shown to increase the risk slightly such as age, blood pressure and blood group A.

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The relationship between intrapartum amniotic fluid index, fetal distress and fetal acidemia

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Intrapartum assessment of amniotic fluid index (IAFI) has been observed to be as efficacious as fetal heart rate (FHR) tracings in determining women who are at risk for meconium-stained amniotic fluid or cesarean delivery for fetal distress.^{1,2} Rutherford et al,³ in a high-risk patient group observed an increase in the incidence of cesarean section for fetal distress in women with an amniotic fluid index ≤ 5 cm. They observed an incidence of cesarean section for fetal distress in their 27 patients with an amniotic fluid index ≤ 5 cm to be 11%. However, Baron et al,⁴ in a mixed group of patients observed a 4.1% incidence of cesarean section for fetal distress in their 170 patients with an amniotic fluid index ≤ 5 cm. Robson et al,¹ observed a cesarean section incidence for fetal distress in 8 of 14 patients (57%) with an amniotic fluid index < 6.2 cm. We reported a 17% (7 of 46 patients) cesarean section incidence for fetal distress. Their data confirmed that a low amniotic fluid index determined intrapartum is associated with an increased risk for a cesarean section for fetal distress. Meconium-stained amniotic fluid has been reported by many investigators to be increased in patients with an amniotic fluid index of ≤ 5 cm.¹⁻³ An increased incidence of FHR tracings exhibiting late variable decelerations have been observed in the presence of low amniotic fluid volumes.^{1-3,5}

In this study, we sought to determine the relationship between IAFI, fetal distress and fetal acidemia in a group of women undergoing fetal blood sampling as a result of repeated deceleration. Women with gestational age of ≥ 37 weeks, cephalic presentation, in active labor and ruptured membranes at the time of IAFI determination and had undergone fetal blood sampling for persistent decelerations, composed the study group. Patients excluded were those with multifetal gestations, with an imminent delivery, with polyhydramnios, in whom no amniotic fluid index was performed due to staff unavailability. Amniotic fluid volume was determined with a linear 3.5 MHz transducer, using 4-quadrant amniotic fluid index technique. All IAFI determinations were performed by the same person. The study group was divided into 2 groups and designated as having oligohydramnios with an amniotic fluid index < 6.2 cm, and normal with an amniotic fluid index > 6.2 cm. We used this threshold on the basis of earlier studies by Robson et al,¹ of amniotic fluid index distributions after either spontaneous ruptured membranes or an amniotomy. All subjects had continuous electronic FHR monitoring and tocodynamometry throughout labor. The FHR tracings were reviewed and coded by Robson,¹ who was unaware of the IAFI