SHORT REPORTS

Aflatoxins in breast milk, neonatal cord blood, and serum of pregnant women

Aflatoxins are fungal metabolites commonly found as toxic contaminants of food commodities in the tropics.[1] Aflatoxins have been detected in breast milk from mothers in the Sudan.[2] A study in the United States has shown that aflatoxins cross the complex porcine placenta and exert adverse biological effects on neonatal pigs.[3] Investigations conducted in Accra, Ghana, and Jos, Nigeria, therefore, sought to confirm the presence of aflatoxins in human breast milk and explored the possibility that aflatoxins cross the human placental membrane.

Subjects, methods, and results

In Accra 142 breast milk samples were collected in the mid-dry season and a further 122 samples at the onset of the wet season. Cord blood samples were obtained from 188 infants at birth. In Nigeria venous blood was collected from 77 mothers during pregnancy and cord blood samples from their infants after complete delivery of the placenta. All specimens were transported frozen to Liverpool for aflatoxin analysis by high performance liquid chromatography using fluorescence detection after extraction and partition.[4,5] Concentrations of aflatoxins B_1, B_2, G_1, G_2, M_1, and M_2 and aflatoxicol were determined.

Aflatoxins were detected in 90 (34%) of the 264 breast milk samples. The frequency of detection was higher in the wet season (50 samples; 41%) than in the dry season (40; 28%) (exact $p=0{\cdot}039$). Aflatoxin M_1 was detected most frequently (59 specimens), in concentrations ranging from 20 to 1816 ng/l. The mean concentration was higher in the wet season (445 (SD 442) ng/l) than in the dry season (293 (281) ng/l). Aflatoxin M_2 was detected in 18 milk samples (16-2075 ng/l), aflatoxin B_1 in 17 (130-8218 ng/l), and aflatoxin B_2 in two (49 and 50 ng/l) and aflatoxicol in three (64, 128, and 270 ng/l).

Aflatoxins were detected in 63 (34%) of the 188 Ghanaian cord blood specimens. Aflatoxins M_1 and M_2 were each detected in 21 specimens in concentrations ranging from 34 to 7320 ng/l and 30 to 572 ng/l respectively. Aflatoxins B_1 and B_2 were each detected in 17 specimens (concentrations 185-43 822 ng/l and 11-925 ng/l respectively). Aflatoxin G_1 was detected thrice (611, 1354, and 354 ng/l), aflatoxin G_2 once (37 ng/l), and aflatoxicol once (117 ng/l).

Blood samples from Nigeria showed aflatoxins in 16 (21%) of 77 maternal samples and 9 (12%) of 78 cord blood samples (including a set of twins). Aflatoxins were found in maternal and cord blood in seven instances (see table). One stillbirth was recorded in the study. The maternal blood contained aflatoxin B_1 553 ng/l.

Nigerian aflatoxin concentrations (ng/l) detected at delivery

Study No	Maternal blood	Cord blood	Study No	Maternal blood	Cord blood
1	M_2 175	M_2 245	50	M_1 483	B_2 10
8	B_1 553	Negative	51	B_1 5005	Negative
12	B_1 4880	Negative	56	Negative	M_2 378
26	B_1 540	Negative	101	M_1 265	M_1 8942
27	B_1 878	M_1 25	102	M_2 48	M_2 330
33	B_2 33	Negative	104	Negative	M_2 208
37	M_1 38	Negative	106	B_2 28	Negative
42	B_1 10 390	M_1 593	111	M_2 90	M_2 155
45	M_2 3480	Negative			

Comment

The frequency of detection of aflatoxin in breast milk in Ghana was similar to that reported in Sudan[2]; concentrations, however, were considerably higher in this series, and the presence of aflatoxin B_1 in 17 samples in concentrations up to 8218 ng/l was surprising. These findings confirm that newborn infants in Africa are frequently exposed to aflatoxins in breast milk and that there are seasonal fluctuations in the level of exposure.

Finding aflatoxins in 34% of Ghanaian and 12% of Nigerian cord blood samples provides firm proof that aflatoxins cross the human placental barrier. The very high concentrations in some Ghanaian specimens and the higher concentrations of aflatoxins in some cord blood than maternal blood samples collected simultaneously in Nigeria suggest that aflatoxins may accumulate in the fetus when exposed to these toxins in utero.

These studies and findings in Kenya reported separately show that a high proportion of infants in tropical Africa have prenatal and continuing postnatal exposure to aflatoxins. Given the adverse effects of such exposure on immunity, liver function, nutrition, and overall survival in controlled experiments on pigs,[3] we should be concerned about the possible consequences of aflatoxin exposure for the human fetus and infant.

This investigation was supported in Liverpool and Ghana by the Commission of the European Communities Director-General for Science Research and Development.

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(Accepted 8 December 1987)

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Plastic material from a syringe causing fatal bowel necrosis in a neonate

Plastic particles from syringes have been reported to enter the circulation through intravenous lines.[1] We report on a neonate in whom this caused ischaemia of the bowel with a fatal outcome.

Case report

A male infant weighing 860 g was born vaginally at 25 weeks' gestation. He had Apgar scores of 3 at one minute and 9 at five minutes. He was ventilated from birth because of the respiratory distress syndrome, and an umbilical artery catheter was inserted at 1 hour of age. Injections were given into this catheter with polypropylene syringes (Omniflex 2 ml, Braun Melsungen). Ventilatory requirements decreased, and by day 4 ventilator pressures were 16/3 cm water at five breaths per minute in 35% oxygen. An ultrasound scan of the central nervous system was normal. The umbilical artery catheter was removed, but within an hour his abdomen became distended and the abdominal wall turned black. Meconium peritonitis was diagnosed. Radiography of the abdomen showed distended loops of small bowel but no gas in the bowel wall.

Nasogastric feeds were stopped, and he was given intravenous nutrition, penicillin, gentamicin, and metronidazole. His condition was static until day 12, when his abdomen became more distended owing to scrotal crepitus and a mass in the right iliac fossa. An abdominal radiograph showed free gas. By day 15 he had not passed further stools and his abdominal signs were unchanged. An enema of sodium and meglumine diatrizoates (Gastrografin) showed that contrast did not pass beyond the hepatic flexure. At laparotomy an inflammatory mass consisting of caecum, gall bladder, and distal ileum was found; the ileum contained a stricture and perforation. The remainder of the small bowel looked viable, and there was no evidence of necrotising enterocolitis. The stricture was resected and an end to end anastomosis made.

For the next 19 days the infant required ventilation; his abdominal signs were unchanged. Rectal washouts and enemas of iopamidol resulted in the passage of only small amounts of meconium. Serial plasma concentrations of immunoreactive trypsin were normal. At laparotomy the small bowel was limp, showed scattered necrotic areas, and contained plugs of inspissated meconium. An ileal stoma was formed, but despite washouts with acetylcysteine no stool was passed and he eventually died at 52 days of age.

On histopathological examination the resected part of the small bowel showed signs of acute infarction. The mesenteric arteries were thrombosed. The thrombus contained irregular fragments of plastic 50-200 μm long, which were identified by laser raman spectroscopy as polypropylene. At postmortem examination there was no evidence of any residual infarction of viscera. Sections