Infants of Diabetic Mothers (IDMs)

BACKGROUND AND PATHOPHYSIOLOGY: With insulin-dependent diabetes mellitus, maternal hyperglycemia, hypoglycemia and ketosis can occur during fetal organogenesis, and there is increased incidence of fetal anomalies. Careful attention to pre-conception control of diabetes decreases the risk of anomalies. With gestational diabetes, because glucose intolerance does not occur during organogenesis, the risk of anomalies is not increased.

Glucose transport across the placenta is not limited. Fetal hyperglycemia stimulates beta-cell hypertrophy, increases insulin production and fetal oxygen consumption. Insulin has mitogenic and anabolic effects on many tissues (*e.g.*, adipocytes, skeletal and cardiac muscle, hepatic and connective tissue), but not brain. Therefore, delivery of IDMs may be complicated by large shoulders and abdomen that can cause dystocia.

CLINICAL PROBLEMS IN IDMs:

Congenital anomalies: Incidence 6-9%, and these account for 50% of mortality. No single anomaly is pathognomonic, but several are *much* more frequent including:

- **Cardiovascular** (*e.g.*,VSD, transposition of great vessels)
- **Skeletal:** Especially, the caudal regression syndrome
- **CNS:** Meningomyelocele, anencephaly, holoprosencephaly
- **Other:** Renal, gastrointestinal

Unexplained fetal demise.

Polyhydramnios is associated with poor control.

Macrosomia (birthweight ≥4,000 grams) Most macrosomic infants are born to non-diabetic mothers. Risk of macrosomia is reduced by good glycemic control during 20-30 weeks of gestation. Shoulder dystocia is more likely in IDM than non-IDM macrosomic infants of similar weight. Macrosomia increases risk of **traumatic delivery** (*e.g.*, brachial plexus palsy, fracture of clavicle) and **asphyxia.**

Intrauterine growth retardation occurs usually with severe diabetes (chronic hypertension, vascular disease) and is associated with congenital malformations.

Hypoglycemia is common and occurs in LGA and SGA. **Screen all IDMs for hypoglycemia** **(**see section on Hypoglycemia, P. 153)**.** First nadir in glucose is 30-90 min post delivery and may take several days to resolve. Glucose requirements may be very high (10-15 mg/kg/min). Rebound hypoglycemia occurs in response to large, rapid boluses of glucose. IDMs are less symptomatic (than non-IDMs), even with significant hypoglycemia.

Hyperbilirubinemia (see section on Jaundice, P. 118)

Hypocalcemia occurs in 17%, usually 2-3 d after birth, and often with hypomagnesemia.

Respiratory Distress Syndrome: Risk is increased 6-fold.

Septal hypertrophy of heart occurs in infants of gestational and insulin dependent diabetics. Left ventricular compliance and cardiac output are decreased. Obtain Cardiology consult. Consider treatment with propranolol (to slow heart rate and allow increased ventricular filling). Hypertrophy gradually resolves by age 6-12 months.

Small left colon (Hypoplastic left colon syndrome) presents as lower bowel obstruction and may be confused with Hirschsprung's Disease. Cause is thought to be delayed innervation of distal bowel. Diagnosis is made by barium enema and history of maternal diabetes. Condition should clear within several days.

Polycythemia is associated with poor glycemic control or maternal vascular disease. (see section on Polycythemia, P. 112)

Persistent pulmonary hypertension (see P. 91 for management)

Low cardiac output: IDMs who have had perinatal asphyxia with metabolic acidosis, hypoglycemia and/or hypocalcemia may have cardiomagaly with ↓ contractility. This responds to combined correction of all metabolic abnormalities.

Poor feeding is common. An IDM may take several days to establish nipple feedings.

MANAGEMENT of IDMs:

•Screen all IDMs for **hypoglycemia, hypocalcemia and polycythemia**, and treat appropriately.

•Careful examination and observation looking for conditions described above.

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