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PROTHROMBOTIC STUDY IN FULL TERM NEONATES WITH ARTERIAL STROKE

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Sir,

Neonatal cerebral infarction is being reported with increasing frequency, with an incidence of 1 in 4000 term infants.[1] Until recently this entity has been explained mainly by severe perinatal distress. Nowadays, risk factors (maternal and fetal) seem to be important in the aetiology of infarction: placental emboli (ageing placenta and maternal thrombophilic factors), thrombotic events (polycythaemia, slow blood flow, disseminated intravascular coagulation, metabolic disorders and thrombophilic factors) and cerebral hipoperfusion (asphyxia).[1-3]

We present the results of the protrombotic study made in 14 term children (7 boys and 7 girls) with neonatal arterial stroke diagnosis (10 in the left and 4 in the right middle cerebral artery territory) and 12 of their mother's.

By the time of sample collection the children's average age was 4 years and 6 months. The protrombotic study included prothrombin, activated partial thromboplastin, thrombin and reptilase times, lipoprotein (a) and fibrinogen levels, activities of antitrombin III, protein S and C, plasminogen, normalized protein C sensibility ratio (FV Leiden when < 0,85) and antiphospholipid antibodies (lupus anticoagulant [LA], anticardiolipin [ACA] and anti beta2-glycoprotein I antibodies).

We haven't found congenital protrombotic risk factors either in children or in mother's population. However, found a high frequency (58%) of antiphospholipid antibodies (APLA) in the mother's population (4 with LA and ACA IgG, 2 with ACA IgG and 1 with LA alone), in contrast with the children population where no APLA was found. Other risk factors for cerebral artery infarct in newborns such as asphyxia, trauma, congenital heart and metabolic diseases were excluded.

The close association between cerebrovascular disease and APLA has been observed in well-controlled prospective studies in young patients.[5] In the newborn period there are few case reports that associate newborn infarction and maternal APLA.[6-9] Theoretically APLA syndrome may occur primarily in newborn arterial territory following placental transmission of IgG APLA antibodies from an affected mother. Alternatively, recent evidence shows that fetal and newborn neurological damage and also cerebral palsy may be related to thrombophilia and resultant thrombosis in placenta, with posterior embolization, possibly involving fetal cerebral vessels. Recent cohort and case control studies did not demonstrate increase in neonatal complications in infants born to APLA positive mothers, with the exception of increased risk of prematurity.[10,11] However, neonatal stroke is a quite rare condition and the number of newborns of APLA positive mothers included in these studies might not be sufficient to draw definite conclusions.

It is our belief that it would be important to perform a prospective multicenter case-control study to assess the pathogenic role of maternal APLA in neonatal stroke.

References

1. Cowan F, Mercuri E.Cerebral infarction in the newborn infant: Review of the literature and personal