Mean bilirubin levels ($\mu\text{mol/l}$) on day 4 in all patients and related to method of delivery and infant feeding

	Received oxytocin		Did not receive oxytocin		P
	No	mean	No	mean	
All patients ..	109	80.049	88	83.043	0.8
Normal delivery ..	77	82.679	61	89.843	0.6
Operative delivery ..	30	78.147	18	64.125	0.4
Caesarean section ..	3	36.479	8	73.744	0.5
Bottle-fed ..	75	81.304	62	74.975	0.6
Breast-fed ..	35	77.779	25	103.010	0.2

Conversion: Traditional to SI units—Bilirubin: $1 \mu\text{mol/l} \approx 0.06 \text{ mg/100 ml}$.

serum bilirubin exceeded $170 \mu\text{mol/l}$ (10 mg/100 ml) in 17 cases; eight had received oxytocin and nine had not.

Discussion

This study shows that infusion of oxytocin during labour does not affect mean bilirubin levels or increase the incidence of hyperbilirubinaemia. Hence oxytocin does not have a deleterious effect on bilirubin metabolism in the newborn. Any increase in the incidence of neonatal jaundice associated with oxytocin is likely to be due to the lower mean gestational age which is an inevitable consequence of induction of labour.

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Platelet and fibrinogen survival in acute promyelocytic leukaemia

Most authors attribute the haemorrhagic diathesis in acute promyelocytic leukaemia (APML) to accelerated consumption of clotting factors and platelets.¹ Promyelocytes in APML contain high levels of "tissue factor,"² which when liberated may initiate disseminated intravascular coagulation (DIC). As an expected corollary of DIC would be a reduced plasma half life of fibrinogen and platelets; we report the results of measuring fibrinogen and platelet survival in a case of APML. Although there was reduced life span of circulating fibrinogen, platelet survival fell within the normal range.

Case report

A 25-year-old woman presented with spontaneous bruising of one week's duration. She had no further complaints and on clinical examination, apart from generalised ecchymoses, there was no abnormality. The peripheral blood showed haemoglobin 10.6 g/dl ; WBC $2.5 \times 10^9/\text{l}$ ($2500/\text{mm}^3$) (neutrophils 43%; lymphocytes 30%; monocytes 2%; eosinophils 2%; basophils 2%; blast cells 1%; promyelocytes 30%; myelocytes 8%); platelet count $23 \times 10^9/\text{l}$ ($23000/\text{mm}^3$). The marrow was infiltrated with abnormal promyelocytes, many containing Auer-like rods. The coagulation findings were (normal values in parentheses): fibrinogen 0.39 g/l ($1.5\text{--}4.0$); euglobulin lysis time (ELT) 20 min (> 120); partial thromboplastin time (kaolin) 48 s (36); fibrin degradation products (FDPs) $80\text{--}160 \text{ mg/l}$ (< 10); thrombin time 16 s

(11); one-stage prothrombin time $1.4/1$. Prothrombin consumption index and plasminogen were normal. No abnormal fibrinogens were detected, and the ethanol gelation test was negative. Before treatment the patient was given eight donor units of platelets, and remission was induced with daunorubicin and cytosine arabinoside. After induction, no further treatment was given for five months. Relapse was heralded by recurrence of bruising and a low plasma fibrinogen level (0.5 g/l), and there was a temporary but partial response to further daunorubicin and cytosine arabinoside. Despite changing chemotherapy the patient steadily deteriorated, dying ten months after presentation with a presumptive diagnosis of cerebral haemorrhage. Necropsy was not performed.

A month before death, before the change of chemotherapy, we measured fibrinogen and platelet survivals. At this stage the platelet count was $165 \times 10^9/\text{l}$ ($165000/\text{mm}^3$) and the plasma fibrinogen 1.25 g/l . The platelet count remained stable for the next three weeks but the plasma fibrinogen concentration fell to 0.29 g/l within four weeks. In vivo ^{125}I -labelled fibrinogen survival³ was significantly reduced with a mean life span of 23 h (half life in eight control subjects 80–120 h). ^{51}Cr labelled autologous platelet survival⁴ was normal, the mean life span being 7.3 days (normal 5.5–10.3) (see figure).

Platelet survival in patient with acute promyelocytic leukaemia. Mean life span of the platelets was 7.3 days (normal 5.5–10.3).

Discussion

Although accelerated fibrinogen disappearance in untreated or relapsed APML is well documented,¹ platelet survival has not been reported. The normal platelet survival in this patient conflicts with the stated view that APML is constantly associated with DIC and platelet consumption.¹ Our result could be explained by primary fibrinogenolysis, and the role of the latter in some cases of APML is suggested by the improvement which has followed treatment with epsilon-aminocaproic acid.⁵ Such a diagnosis would be supported in this patient by the short ELT, raised FDPs, and negative ethanol gelation test at presentation.

We propose that primary fibrinogenolysis has a role in this disorder. Further investigation is required to determine its magnitude, whether it takes place only in a distinct subpopulation of patients with APML, or whether it forms a prelude to the onset of overwhelming DIC.

We are indebted to Dr S H Davies for allowing us to describe this patient who was under his care.

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