SHORT REPORTS

Maternal autoimmune thrombocytopenia and the newborn

That a humoral factor was the causative agent in idiopathic thrombocytopenic purpura was suggested nearly 30 years ago after the observation that mothers with the disease often give birth to children who develop transient thrombocytopenia.[1] Maternal platelet autoantibodies were presumed to pass into the fetal circulation. With present techniques[2,3] platelet autoantibodies are detectable in most patients with idiopathic thrombocytopenic purpura. We have therefore tried to confirm the passage of autoantibodies through the placental barrier and determine their immunochemical characteristics. We used specific antiglobulin reagents labelled with fluorescein isothiocyanate.

Patients and methods

We studied four pregnant women who fulfilled the criteria for idiopathic thrombocytopenic purpura.[3] Three were primigravidae whose thrombocytopenia was first diagnosed during pregnancy. Two had platelet counts below 40×10^9/l (40 000/mm^3) on the day of diagnosis and were treated with prednisone two and three weeks, respectively, before delivery. The fourth woman was in her second pregnancy. During her first pregnancy she had also suffered thrombocytopenia (70×10^9/l) which did not need treatment. The infant did not have purpura, although the platelet count in the neonatal period was not determined. All four women were delivered vaginally without excessive blood loss. The infants showed no evidence of haemorrhagic tendency or severe thrombocytopenia in the neonatal period. The table lists the results of the serological investigations.

Ether eluates prepared from platelets from maternal and cord blood in one case were tested against a panel of normal donor platelets typed for the platelet-specific alloantigens Zw, Ko, and Bak^a and with the platelets of a patient with Glanzmann's thrombasthenia. Both eluates reacted equally well with all normal donor platelets, but not with thrombasthenic platelets, a reaction observed in about two-thirds of patients with autoimmune idiopathic thrombocytopenic purpura (unpublished observations).

Comment

In all four cases we confirmed the suspected transfer of IgG autoantibodies through the placenta. We also showed in two cases that additional IgM antibodies did not pass the placental barrier. The gradual disappearance of antibodies from the infants' platelets supports the passively acquired nature of these antibodies. This is further supported by the finding that the IgG subclass and blood group specificity pattern of the autoantibodies in two cases were identical in the mother and newborn.

None of the newborns developed severe thrombocytopenia, and no relation was found between the degree of platelet sensitisation in mother and child and the platelet count in the neonatal periods. Mothers, in remission of idiopathic thrombocytopenic purpura after splenectomy,[1] or with autoantibodies and moderate thrombocytopenia ($80\text{-}180 \times 10^9$ platelets/l), however, may give birth to a severely thrombocytopenic infants. Alloantibodies against platelets may also be formed by the woman during pregnancy, usually resulting in a more severe transient neonatal thrombocytopenia but with completely different therapeutic consequences.[4] The introduction of reliable tests for detecting antibodies against platelets makes it possible to determine the presence of these antibodies and to differentiate between their autoimmune and alloimmune character.[4,5] This enables optimal measures and precautions to be taken in the present or future pregnancy, delivery, and neonatal period.

We thank the clinicians who participated in this study.

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Acquired megacolon associated with acute lead poisoning

The cause of acquired megacolon in adults is often obscure. Although lead poisoning is mentioned in some standard texts[1] as a cause, we have not been able to find any documented report linking the two conditions. We describe a patient with acute plumbism who presented with reversible megacolon.

Case report

A 39-year-old man had been employed as a painter and decorator for three years. Two weeks before admission he developed episodes of muscular weakness and colicky abdominal pain, together with a sensation of abdominal bloating and distension. One week after the onset of these symptoms he developed pains in his ankles, knees, hips, and wrists. He lost his appetite and also noted a diminution in his sense of taste, though during this period he observed an unusual craving to drink large amounts of milk. Towards the end of the second week of his illness he developed complete constipation, worsening of the abdominal pain and increase in his girth, insomnia, increased nervous irritability, and paraesthesia, principally in the arms and legs. During the three weeks before the onset of symptoms he had been cleaning the paint off the outside of an old building, down to the original surface, using a motorised wire brush. The original surface paint was applied in the 1920s. He had not worn any protective mask while doing this

Results of serological investigations in four mothers with autoimmune thrombocytopenia and their infants

Case No	Maternal blood			Infants' blood			3rd-day platelet count ($\times 10^9$/l)	Direct platelet immuno-fluorescence test	4th-week platelet count ($\times 10^9$/l)	Direct platelet immuno-fluorescence test
	Platelet count ($\times 10^9$/l)	Direct platelet immuno-fluorescence test	Ig class	Cord blood platelet count ($\times 10^9$/l)	Direct platelet immuno-fluorescence test	Ig class				
1	70	+	G	135	(+)	G	220	(+)	238	−
2	104*	+ +	G+M	145	+ +	G	196	(+)	256	−
3	140*	+ +	G_1+M	199	(+)	G_1	240	−	>200	Not tested
4	70	+ +	G_1	202	+ +	G_1	101	+ +/+	238	(+)

*After treatment with steroids.