Disseminated Intravascular Coagulation in Meningococcal Infection

Sir,

The description by Fox in the recent issue of the *Archives* (October 1971) of histological evidence of widespread intravascular coagulation in 13 patients dying of the Waterhouse-Friderichsen syndrome (with proved meningococcal septicaemia in two) and his suggestion that heparin should be used in such patients, prompts us to report the following patient:—

A male infant aged 4 months was admitted with the complaint of sudden onset of fever, drowsiness, and purpuric rash on his arms, legs, trunk, and face. He was born of an uneventful pregnancy by spontaneous vertex delivery with a birthweight of 4·13 kg. He was breast fed from birth and had had no illnesses.

On examination he was critically ill and had a temperature of 40 °C. Pulse rate was 120 beats a minute and regular. Blood pressure was 80/40 mm Hg (5 cm cuff right arm, lying). He had widespread purpuric spots on his face, trunk, and extremities, and most of them were confluent. His peripheral circulation was good. Anterior fontanelle tension was normal and there was no nuchal rigidity. Examination of major systems was normal.

Blood urea and electrolytes were normal. Gram-negative diplococci, morphologically identical with meningococci, were found in a smear made from a purpuric spot. Blood and CSF cultures were sterile. The CSF was normal.

Treatment with penicillin, sulphadimidine, and hydrocortisone was initiated. Meningococci were subsequently isolated from a sib.

On the night of admission (day 0) the platelet count was 190,000/mm³, with numerous platelets present on the blood film. Hb and WBC were normal. There were no burr cells.

The following morning (day 1) the platelet count had fallen to 35,000/mm³ and platelets were scanty on the film. Hb was 11g/100 ml and there was a leuco-cytosis (37,000/mm³) with an increase in polymorphs, lymphocytes, and Turk cells. Burr cells were still absent.

This rapid drop in platelet count coincided with a progressive clinical deterioration, with extension of the purpura which became confluent and necrotic in areas, and the occurrence of fits. Rapid coagulation screening tests showed a grossly prolonged Quick's prothrombin time of 40 sec (control 11 sec) and a slight reduction of the fibrinogen titre to 1 : 64 (control 1 : 128). The combination of these findings was thought to be strongly suggestive of disseminated intravascular coagulation and we decided to start serial coagulation studies and treatment with continuous heparin (25μ/kg per hr). The sequential changes are shown in the Table. The heparin was stopped on day 6 when the platelet count was normal. (All drugs were given intravenously while on heparin to avoid IM injections.)

The low levels of the consumable coagulation factors (*V*, *VIII*, and fibrinogen) and plasminogen, with grossly raised fibrin degradation products (FDPs), together with a return to normal after heparin were conclusive evidence of consumptive coagulopathy, this being the *in vivo* equivalent of the pathological demonstration of intravascular fibrin deposition reported by Fox. Burr cells appeared on day 2 followed by a fall in Hb level, presumably as a consequence of intravascular fibrin deposition.

The correction of this consumptive coagulopathy was followed by a return to normal of the platelet count; together with clinical improvement, no further fits and no further extension of purpura. The patient made an uneventful recovery with no residual neurological defect on subsequent follow-up.

TABLE
Meningococcal Septicaemia

Day	Platelets	FDPs	Fibrinogen	V̄	AHG	Plasminogen
1	35,000	160	227	2%	23%	0·44
		R Heparin		25 μ/kg per hr	—	—
2	23,000	80	328	10%	75%	0·44
4	71,000	80	421	90%	150%	1·33
5	123,000	40	332	80%	120%	1·11
6	163,000	40	318	110%	105%	1·33
Normal Values	>150,000/mm³	>5 μ/ml	>300 mg/100 ml	50–200%	50–200%	>3·0 c.u./ml

The potential value of heparin therapy in this disorder has similarly been shown by Abildgaard *et al.* (1967); Hitzig *et al.* (1968); Winkelstein *et al.* (1969), and Ellman (1971), but is not widely appreciated in this country. This may constitute a serious omission in management since it is the patient with evidence of intravascular coagulation in whom there is a poor prognosis (McGehee, Rapaport, and Hjort, 1967; Evans *et al.*, 1969).

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