HEMATOLOGICAL ASPECT IN DENGUE-HEMORRHAGIC-FEVER PATIENT

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Bleeding diathesis, a well-known phenomenon associated with dengue hemorrhagic fever has been an active subject of recent investigation. Its spectrum manifests widely from a mild form of only purpuric spots on the skin to the most extreme one with internal hemorrhage of many organs, with which shock consistently accompanies and death eventually supervenes. Three possible factors categorized to be responsible for such mechanism of bleeding are as follows.

I. Vasculopathy

Evidence of vasculopathy manifests clinically as spontaneous purpura or by application of Tourniquet, and bylaboratory mean as spontaneous rising of hematocrit,hypovolemia suggesting an increase vascular permeability. Its defect of still an unknown nature may plausibly allow extravasation of blood elements out of the vascular channels. Speculated mechanisms enabling to be postulated for

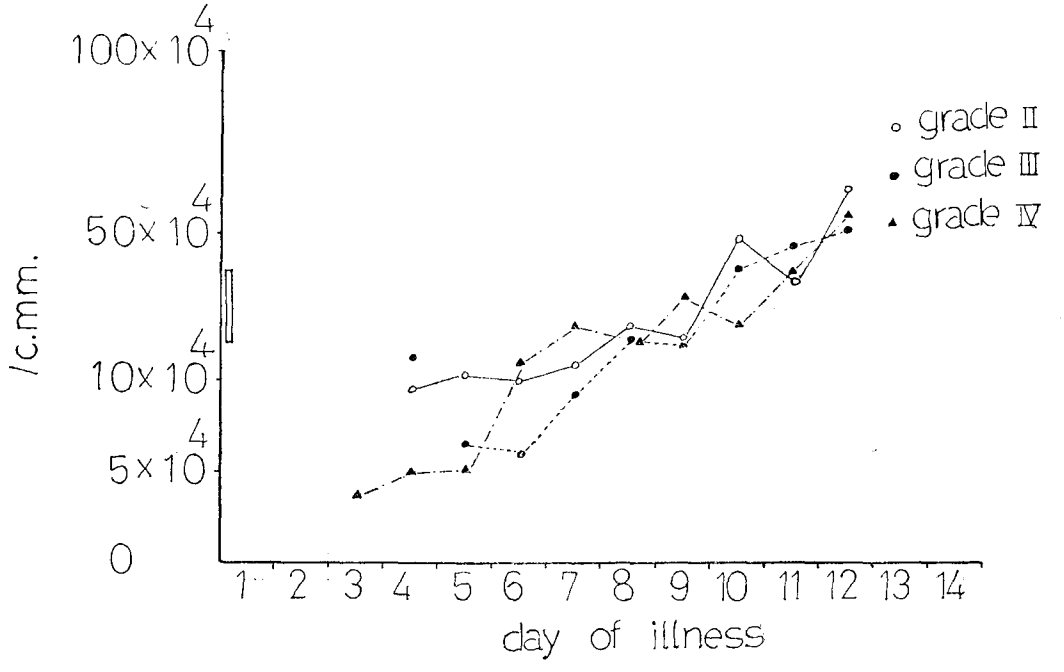

Fig I. Serial platelets count in DHF patients with various graded severity.

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inducing such increased vascular permeability would be the intermediary products of activating the complements, platelets and kallikrein – kininogen.

II. Thrombocytopenia

This is a unique finding associated with dengue hemorrhagic fever. Earlier study of the bone marrow in these patients within the first few days of their illness demonstrated hypoactivity and decreased number of megakaryocytes suggesting decreased production.[1] Clinical attempts to interrupt the bleeding problem manifested in this group of patients by administering intravenous platelet transfusion had never been revealing[2].

This earlier speculation suggested that mechanism of platelet depletion was more than the simply inadequate platelet production. Studying the number of circulating platelet revealed that it tends to deplete in proportion to the clinical severity (Fig. I). The platelet – survival study using platelet labelling with radioactive ^{51}Cr performed in a few patients discovered an unusual shortage of its half – life (Fig. II) and an increased uptake of the radioactive chromium in the liver implicating enhanced destruction of the circulating platelets

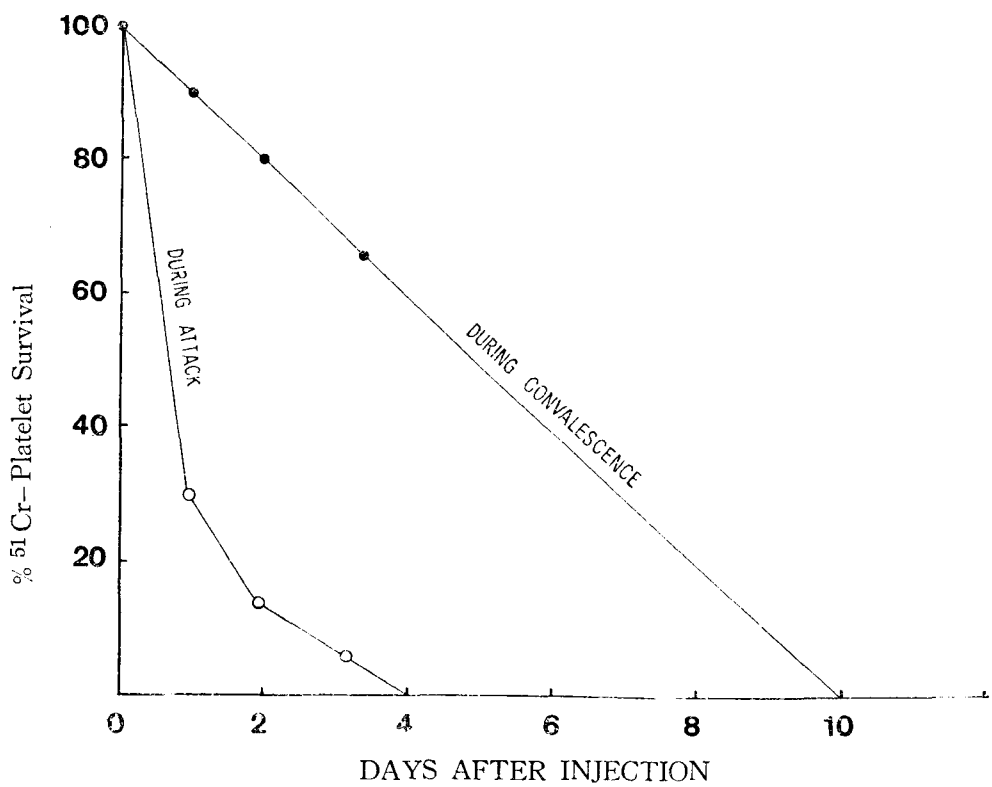

Fig. II. Platelet survival study in a patient.

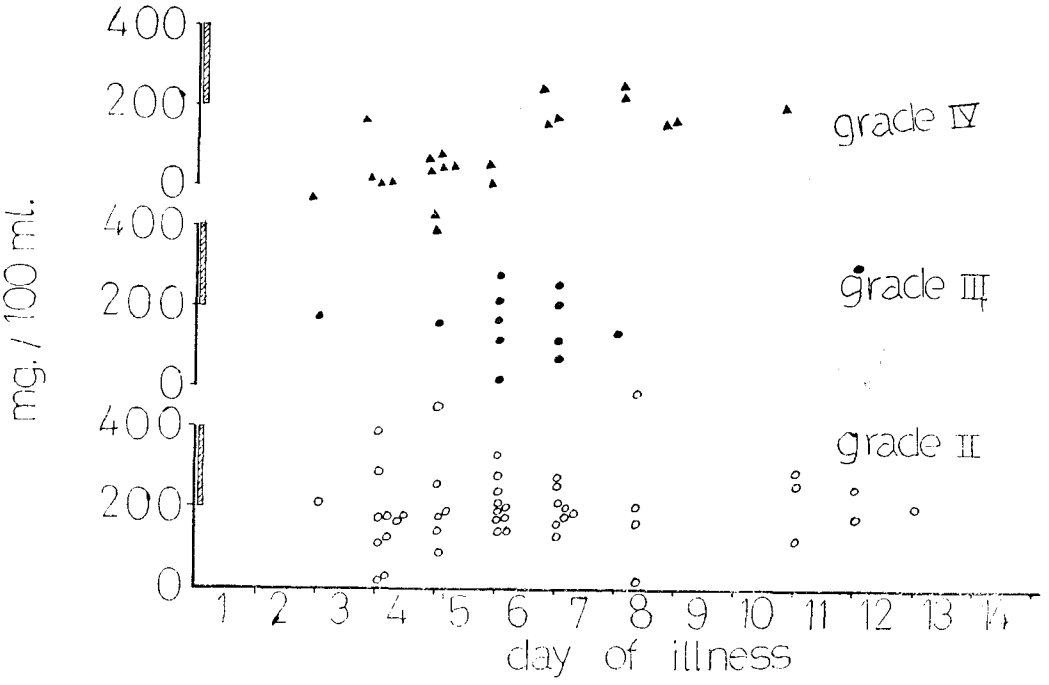

Fig. III. Fibrinogen determination in DHF Patients in respect to clinical severity grading.

III. Coagulopathy

Fibrinogen level was shown to be depleted in dengue virus infected patients; paralleling the degrees of clinical severity (Fig. III). A search for evidence of intravascular coagulation was accomplished by routine coagulation study, determination of fibrin degradation products and Hageman factor. The results of coagulogram showed only mild alteration of prothrombin time (PT), partial thromboplastin time (PTT) tests. Fibrin degradation product (FDP) by an large was slightly elevated above the normal level (Fig. IV) (normal value of FDP = 0 - 0.5 %). The result of Hageman factor studied in a few cases also showed a low level of Hageman factor. (Fig. V) All these findings point in favor that there is a mild to moderate degree of intravascular coagulation occurring in dengue hemorrhagic patients. In few severely bled patients, attempts to control bleeding by transfusing fibrinogen, concentrated platelets, fresh plasma and whole blood were mostly unsuccessful, which indicates a requirement of extraexplanation for the mechanism of such bleeding than the defective clotting mechanism per se.

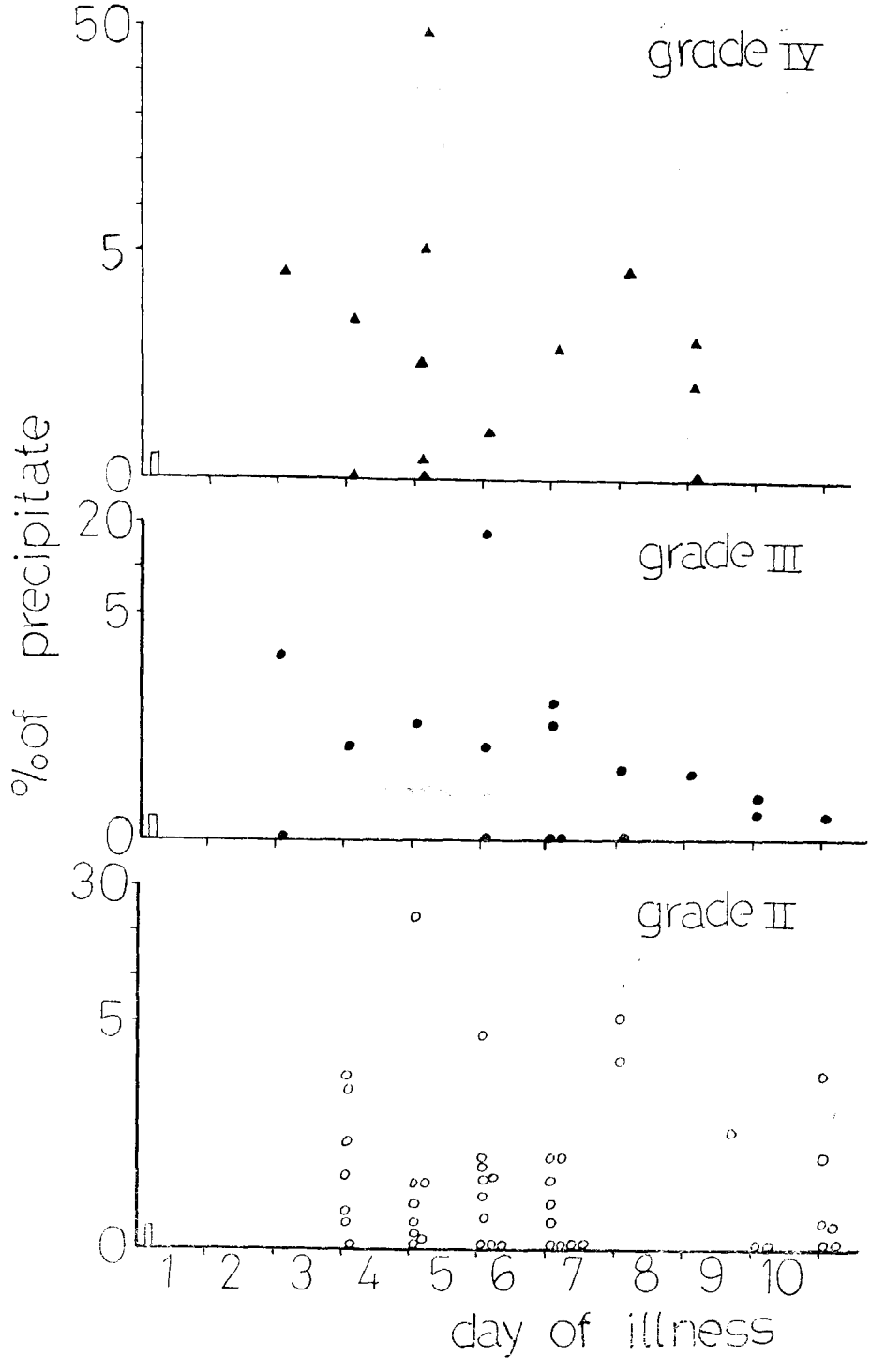

Fig. IV. Levels of fibrin degradation product in DHF Patients by mean of Tube Precipitin Technique.

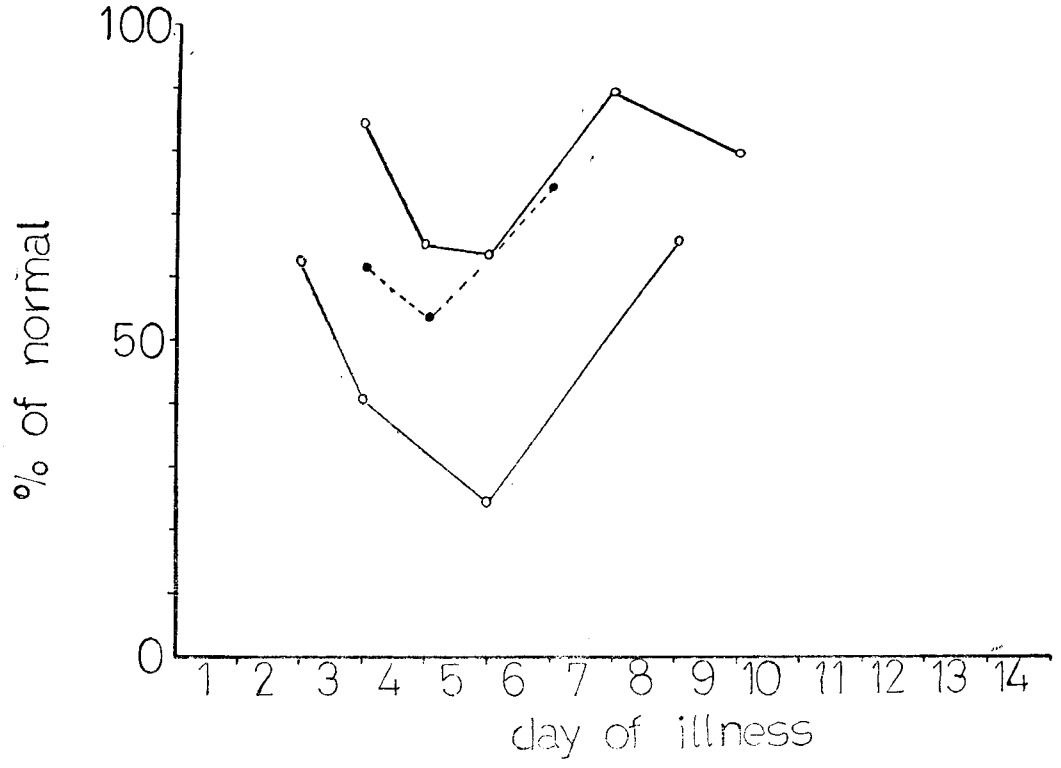

Fig. V. Hageman factor determination in DHF Patient

Reference

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2. Bhanchet, P., Tuchinda, S., Pitakpriwan, P., Pariyavatee, P., Djcrassi, I., Tuchinda, P., Chulacharitta, C.: The Control of Bleeding in Thai Hemorrhagic Fever by Transfusions of Fresh Pletelet Concentrated. Bulletin of WHO 35:75, 1966.