

Subtitle:

COMPARATIVE STUDIES IN THE PATHOLOGY AND HOST PHYSIOLOGY OF MALARIAS. PLASMODIUM INUI MALARIA.

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Preceding reports of this series described the pathophysiology of Plasmodium coatneyi and gibbon malaras. This report is concerned with the infection caused by P. inui in Macaca mulata. P. inui is of interest as potential model for human quartan malaria which it resembles in morphology and schizogonic behavior. Another point of affinity is that P. inui is capable, at least under experimental conditions, of infecting man (Coatney et al, 1966).

While the data are complete for the P. inui study they have not, as yet, been fully analyzed. In view of this only a summarization of our findings is given in this report.

Methods

The strain of parasite used in this study was isolated from a naturally infected M. irus that came from S. Thailand (see Ann. Rep., 1966). It is probably P. inui var. shortii. The methods of study followed that for P. coatneyi (Desowitz et al, 1967).

Results

As in P. coatneyi malaria there was a wide variation in intensity of infection. The most severe infections were produced in splenectomized rhesus monkeys although only in one instance did the animal die. A typical course of infection across the acute phase (first 40 days) is shown in fig. 1. It will be seen that prior to the haemolytic phase there is, early in the infection, a haemoconcentration phase. This was also described for P. coatneyi infections but preliminary results indicate that the period of haemoconcentration may be somewhat more prolonged in P. inui malaria. During the primary parasitaemia in splenectomized rhesus there was evidence of hepatic and renal pathophysiology. Transaminases (SGOT), BUN and creatinine were all elevated. These elevations were usually of a transient nature and the serum chemistries returned to normal by the 25th day despite a continuing moderate parasitaemia. Cholesterol also was lowered during the acute phase when a pronounced anemia was evident. However the hypocholesterolaemia was not as marked as that in gibbon malaria (Miller et al 1967).

A number of infections have now been followed for one year or more which has permitted a study on the effect of chronic P. inui. Unlike P. coatneyi in which only a very scanty parasitaemia was found after about the 60th day, P. inui often persisted in moderate numbers throughout the entire observation period of a year or more. Two animals sustained a parasitic recrudescence between the 150th and 200th days. As a consequence of this attack there was marked evidence of renalhepatic pathology. The renal pathology was of special interest since it showed some similarity to nephrotic syndrome i.e., elevated BUN, creatinine and cholesterol and concomittant proteinuria (fig. 2). It has been postulated that chronic P. malariae infections may cause nephrotic syndrome. There has been no direct evidence for this and the hypothesis has been based on epidemiologic observations. If further experiments confirm that chronic P. inui may also cause a similar pathology then a highly useful model would be available.

Fig. 1. Course of parasitaemia, haematology and serum chemistries during the course of a *P. inui* infection in a splenectomized rhesus

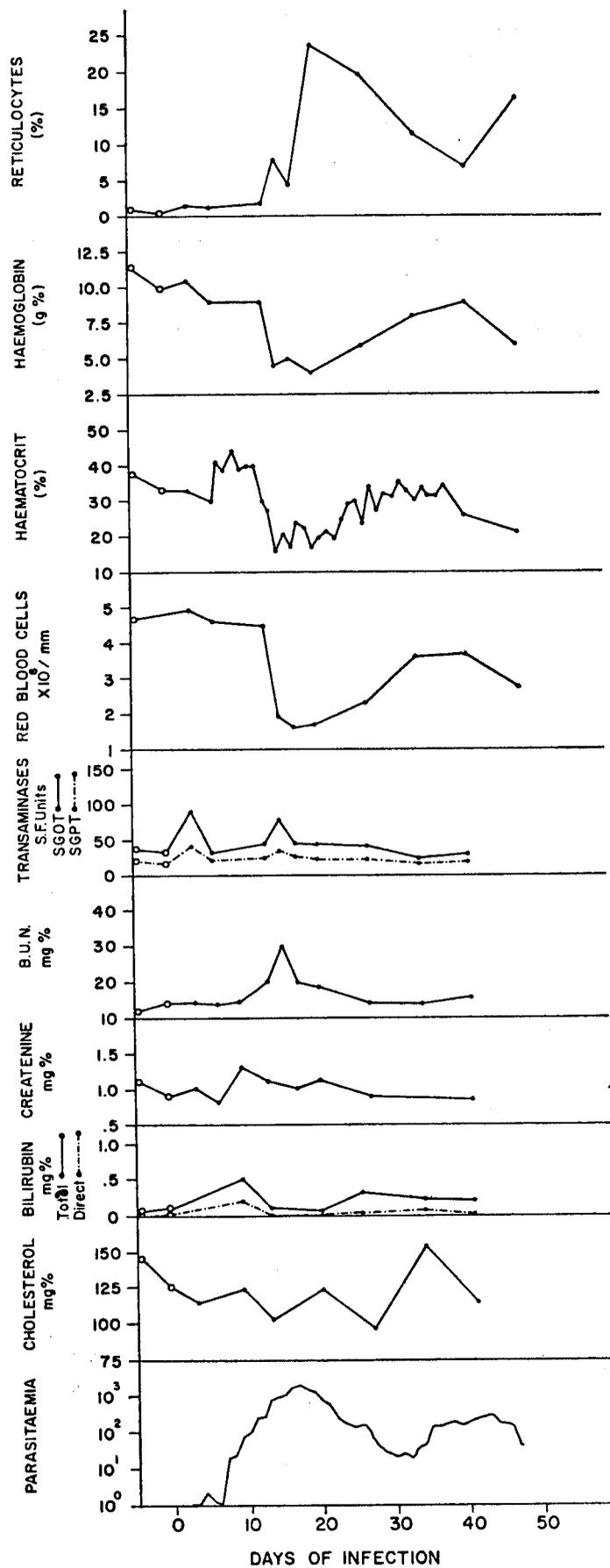


Fig. 2. Haematology and serum chemistries during a late parasitaemic recrudescence in a *P. inui* infected rhesus.

