

workshops, and other such workshops, which are considered as “hot-spots” should be searched for. Most commonly they are in home settings or where young children have ready access, creating more chances of exposure to the children [2], as in our case. An initiative for “hot-spot” investigations and interventions in cottage industries is thereby mandated.

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## Splenic Infarction in *Plasmodium vivax* Malaria

This is in response to a recently reported case of splenic infarction due to vivax malaria [1]. We had a similar child, who on further evaluation was found to have an elevated titer of antiphospholipid IgM. We managed the child with low molecular weight heparin followed by oral warfarin.

In recent years, the association between infections and antiphospholipid syndrome has been reported in several epidemiologic and experimental studies that support the idea of infectious induction of aPL [2]. Witmer, *et al.* [3] reported two children with *Mycoplasma pneumoniae* pulmonary infection complicated by the development of splenic infarction and transient antiphospholipid antibodies. Among parasitic infections, malaria and leishmaniasis have been linked with the production of aPL. Avcin and Toplak [4] reviewed 100 antiphospholipid syndrome cases associated with infection and summarized clinical and experimental evidence on the association between aPL and infectious diseases, they also emphasized a possible association with immunizations. Consigny, *et al.* [5] found a high prevalence of serum cofactor independent aCL, which is a type of antiphospholipid antibody in 137

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individuals chronically exposed to *Plasmodium falciparum* or *vivax* infections.

We opine that antiphospholipid antibodies should be done in all unusual cases of thrombosis. Anti-phospholipid antibodies usually take 3-6 months to disappear, till that time the patients should be on anticoagulant prophylaxis.

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