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The liver as an organ at risk for *Toxoplasma* transmission during transplantation: myth or reality?

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Background: The parasite *Toxoplasma gondii* can be transmitted through solid organ transplantation, by the introduction of a cyst-containing organ from a donor with chronic infection into the recipient. Few cases of toxoplasmosis consecutive to liver transplantation are described, but they are usually severe because of the host immunosuppression following the allograft. Actually, the prevalence of cysts in the liver of seropositive donors is poorly documented in humans, thus prophylaxis guidelines to prevent toxoplasmosis during liver transplantation are not consensual. As hepatic *Toxoplasma* infection is difficult to explore in humans, this study aimed at characterizing hepatic cyst carriage in a murine model and at potentially defining a preferential hepatic localization.

Material/methods: The study was conducted on 19 female Swiss mice used for routine diagnosis of congenital toxoplasmosis at the University Hospital of Rennes. Mice were infected by intraperitoneal injection of placenta from mothers with a history of toxoplasma primary infection during pregnancy. Once toxoplasmosis was confirmed by a positive serology, mice were sacrificed, and organs (brain, heart and liver) were collected. Liver was divided into lobes (right median, left median, large, right lateral, left lateral, papillary process and caudal lobe) before DNA extraction. DNA was extracted by the phenol-chloroform method, and quantitative PCR was realized using an in-house method targeting the rep529 sequence.

Results: Although parasite loads were far lower in the liver than in brain or heart, the frequency of liver infection was surprisingly very high, as 95% (18/19) of mice had a positive toxoplasma PCR in at least one hepatic lobe. The mean number of positive lobes per mouse was $2,8 \pm 1,7$ (46 % of lobes). No relation was found between the liver weight and the hepatic parasite loads nor the number of infected lobes. There was no correlation with the cerebral parasitic load. There was no lobe with a higher frequency of positivity. However, parasite loads were higher in the papillary process than in any other lobes ($p < 0,05$).

Conclusions: Despite the fact that the heart is well-known as the main organ at risk for *Toxoplasma* transmission during transplantation, it appears clearly that the liver is also a target organ for the parasite. This hepatotropism is not restricted to a given anatomical site, as parasite DNA could be detected in any lobe. This absence of preferential anatomical location challenges the possibility to localize cysts by guided liver biopsy in humans, to verify the frequency of cyst carriage. However, in view of this finding, liver recipients with *Toxoplasma*-mismatch transplantation should benefit from systematic chemoprophylaxis, which is not the rule in all countries.