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Anti-*Toxoplasma gondii* antibodies in patients infected with hepatitis B virus

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Toxoplasma gondii (*T. gondii*) is closely related to other Coccidia and has certain similarities to malarial parasites. The parasites were first discovered on a North African rodent called the *Ctenodactylus gundii*, hence, the species was named as *gondii*. Although serologic evidence indicates a high rate of human exposure to the organism, the disease itself is relatively rare. *Toxoplasma gondii* can infect many vertebrates as well as humans, but the definitive host is the house cat and other members of the Felidae family. This organism is an obligate intracellular parasite, which are found in humans in 2 forms. The actively proliferating trophozoites or tachyzoites are usually seen in early, more acute phases of infection. The resting forms or tissue cysts are primarily found in muscle and brain, probably as a result of the host immune response.[1] *Toxoplasma* infections can be acquired postnatally and are categorized into 4 groups: (a) lymphadenitis, fever, headache and myalgia, with a possibility of splenomegaly and a brief erythematous rash (b) typhus-like exanthematous form with myocarditis, meningoencephalitis atypical pneumonia and possible death (c) retinochoroiditis, which may be severe, requiring enucleation (d) central nervous system involvement, which is usually fatal.[2] *Toxoplasma gondii* is transmitted parenterally, flourish in states of immunosuppression and most of *Toxoplasma* infections are asymptomatic. The large number of people who are serologically positive for *T. gondii* suggest that the majority of infections are benign, with most people exhibiting few (cold or light case of flu) or no symptoms. In a small percentage of cases, symptoms may range from mild to severe results.[2]

In Turkey, hepatitis B virus (HBV) is still a serious health problem. The prevalence of HBV carriage is 2-10%. Hepatitis B represents syndromes of hepatocellular necrosis, inflammatory and regenerative responses associated with little or no liver disease or with acute hepatitis. Patients with HBV demonstrate various cellular and humoral immunity disorders. Immunosuppression seems to increase HBV replication.[3] It may be thought that toxoplasmosis may lead to more frequent and more severe diseases in patients with HBV and may change the course of the disease. Therefore, we planned this study to determine the seroprevalence of anti-*T. gondii* antibodies in patients infected with HBV.

One hundred patients (57 males and 43 females; mean age: 46.5 ± 10.2) with HBV were selected and followed up by Ege University Medical Faculty, Gastroenterology department. All selected patients had positive hepatitis B surface antigen (HBs Ag) and they have been followed for at least 6 months for HBV. We also selected 50 healthy volunteer blood donors as control group (31 males and 19 females; mean age: 40 ± 6.7). Blood samples were taken from the brachial vein of all patients under sterile conditions. The sera were separated after centrifugation at 1000 x rounds per minute for 10 minutes and stored at –20 ^{o}C until the analysis.

Serologic techniques. Enzyme linked immunosorbent assay (ELISA). The sera were diluted and assessed semi quantitatively. For this, samples were diluted up to 1/64, 1/256, 1/1024 and 1/4096 to determine immunoglobulin M (IgM) antibodies and up to 1/256, 1/1024, 1/4096, 1/8000 and 1/32000 to determine immunoglobulin G (IgG) antibodies. The sera were read at 405 nm wavelength ELISA reader (Titertek II). The mean absorbance values of negative controls were added to the standard deviation of the absorbance values. Those that were obtained above the cut-off were accepted as positive and compared with the values expressed by these control sera to assess the suspected sera. Accepted as significant titers with regard to active disease were 1/1024 for IgG and above and 1/256 and above for IgM.

Immunofluorescent assay (IFA). Particle antigens were prepared and serum samples were diluted and assessed semi quantitatively. The dilution of the sera within the scope of the study was 1/16, 1/64, 1/128, 1/256, 1/512, 1/1024 and 1/4096 for both IgG and IgM. The results obtained were assessed by a fluorescent microscope (Nikon) at 490 nm stimulation, 510 nm barrier filter wavelength and x 200 magnification. Accepted as significant titers with regard to active disease were 1/256 for IgG and above and 1/16 and above for IgM. All samples that were obtained for seropositive of IgM were also determined by micro ELISA IgM immune capture kit purchased from MEDDENS commercial manufacturer for confirmation. This technique was performed following the manufacturer's instructions.

Statistical analyses. Student t-test was used for the statistical analyses and was carried out by the statistical package for social sciences V.10 for Windows.

Seventy-eight (78%) cases in the patients group and 24 (48%) healthy volunteer blood donors (control group) were found to be positive for IgG antibodies (**Table 1**).

The percentage of anti-*T. gondii* IgG positive antibody in patients with HBV (78%) was found to be significantly greater than the healthy volunteers (48%) ($p<0.05$). Three patients were positive for IgM antibodies (3%) in the patient group, although all subjects in the control group were seronegative. The percentage of anti-*T. gondii* IgM positive antibody in HBV patients (3%) was found to be greater than the healthy volunteers (0%) but the differences between the groups were not statistically significant ($p>0.05$).

Toxoplasmosis is a protozoan disease that is widespread all over the world and demonstrates varying clinical manifestations.[1] Determination of its incidence in the society and the establishment of these risk play a significant role in taking the necessary precautions against this disease. Previously, seropositivity rates of specific IgG antibodies for *Toxoplasma* were reported as 23.1% in Izmir Region and 36% in Kayseri Region in Turkey.[4] This present work is the first study with controls that addressed the prevalence of anti-*T. gondii* antibodies among the patients with HBV infection in Turkey. This study is also the first investigation of *Toxoplasma* seroprevalence in patients with HBV.

Table 1 - The percentage of anti-*T. gondii* IgG and IgM antibodies in hepatitis B virus on patients and control group.

	Age Mean ±SD	IgG positive n	(%)	IgM positive n	(%)
Patients	46.5 ± 10.2	78	(78)	3	(3)
Controls	40 ± 6.7	24	(48)	-	-

IgM - immunoglobulin M
IgG - immunoglobulin G

Turkey is in the moderately endemic region (2-10%) for hepatitis B. The proportion of HBV infections attributable to occupational exposures in the cohort, which includes some subjects born in HBV endemic areas and others who may be at risk for non-occupational HBV infection, is an important factor. Liver injury and extra hepatic disorders are caused by cell-mediated and humoral patterns of response to HBV infections. Whereas, both humoral and cellular immune responses are needed for effective viral clearance, the cellular immune response appears to be primarily involved in the pathogenesis of the disease. Therefore, cytokines play a crucial role in the natural clearance of HBV. They have been used as possible therapeutic agents for chronic hepatitis B. Cytokine responses are characterized as T-helper one (TH1), which induce HBV specific cytotoxic T lymphocytes and virucidal cytokines such as tumor necrosis factor alpha and interferon-λ or TH2, which induce antibody responses to viral antigens.[5] The present results revealed higher percentage of positivity for *T. gondii* IgG antibodies in patients with HBV (78%) compared to the controls (48%) with a statistically significant difference. These findings may be due to the fact that patients with HBV are immunocompromised which increase their susceptibility to this infection.

The patients with HBV may well form a risk group for *Toxoplasma*. The reasons as to why both the antibody positivity and titers have been found to be at high rates are still not clear. We know that

toxoplasmosis presents a special problem in immunosuppressed host, wherein reactivation of a latent toxoplasmosis may be developed. Therefore, patients with HBV should be screened for *Toxoplasma* and parasitologic surveys of HBV patients should be periodically performed to prevent the possible dissemination of toxoplasmosis.

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Hypovolemic cardiac arrest after dental extraction. *An unexpected high-flow maxillar arteriovenous malformation*

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Maxillar arteriovenous malformations (AVMs) of the maxillofacial region sometimes give rise to dental emergencies and may cause disfigurement, morbidity and even death. The most common clinical presentation is expansion of the buccal cortex, gingival bleeding from around the necks of mobile teeth or severe hemorrhage after dental extraction, gingival biopsy, or eruption of a tooth.[1,2] The vascular and hemodynamic nature of the lesion is important in determining the treatment and providing a favorable prognosis. Preoperative embolization and intervention by different therapeutic surgical modalities became the treatment of choice in the majority of cases.[3] This report describes a case of life threatening post extraction hemorrhage occurring in hypovolemic shock.

A 6-year-old girl was referred to the emergency department for assessment of post-extraction hemorrhage. On the day of presentation, she underwent extraction of the third molar tooth at a hospitals' dental office under local anesthesia. After the extraction, severe hemorrhage began and the dentist made a digital pressure at the site of the hemorrhage. However, the patient and her parents were agitated as the hemorrhage was constant. Hypotensive shock then occurred. Pulse and blood pressure (BP) was not achieved. A 24 G cannula was placed and entubated with 5 mg midazolam. Hemoglobin (Hb) count was 4 g/dl and so blood transfusion (400 cc) was administered. However, the tamponade was not able to stop the hemorrhage so she was referred to Hacettepe University Hospital emergency department. On examination, vital signs indicated hypotension (BP 60-40 mm Hg) and tachycardia (heart rate 140 beats/min), a mild hypoxia was noted despite the spontaneous breathing (oxygen saturation was 94% on room air). Neurologic examination was not carried out due to midazolam. Laboratory tests revealed a Hb count of 9.2 g dl^{-1} and hematocrit of 26%. Arterial blood analyses: hydrogen ion concentration 7.2, partial pressure of carbon dioxide 43.5, base excess -7.9 and laboratory data analyses were within normal limits. Anesthesia and maxillofacial surgery department examined the patient and decided to make an angiography. A cerebral and facial angiography was carried out under general anesthesia and revealed high flow arteriovenous malformation fed by right maxillary artery. Embolization, which consist in occluding the vessels contributing to the lesion (right maxillary artery) with polyvinyl alcohol particles (1 vial). An embolization of the right maxillary artery and arteriovenous malformation was performed. A complete occlusion of the malformation and cessation of bleeding were achieved after the embolization. On arrival in the anesthesia reanimation unit, blood results revealed a Hb of 11.7 g.dl^{-1} and the trachea was extubated uneventfully on the sixth hour after the embolization. Twelve hours later, surgery was performed by curettage of the collapsed vascular anomaly at the maxillary sinus and maxillary bone. There was no excessive bleeding (60 cc). She was discharged in good condition 3 days later with Hb