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Helicobacter pylori – a risk factor for the development of the central serous chorioretinopathy

Helicobacter pylori jako czynnik ryzyka rozwoju centralnej surowiczej chorioretinopatii

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Summary: Purpose: To prove the influence of the *Helicobacter pylori* for the development of the central serous chorioretinopathy.
Material and methods: We examined 55 patients with central serous chorioretinopathy confirmed by fluorescein angiogram and 55 controls. Each patient provided venous blood sample for IgG anti – bodies to *Helicobacter pylori* by enzyme – linked immunosorbent assay technique (ELISA) and a stool specimen for *Helicobacter pylori* antigens.
Results: 44% in CSC patients were positive results of stool examine and only 29% in group control. In 67% of the patients we proved the presence of the antibodies IgG – anty *Helicobacter pylori* and in 47% controls. The difference was statistically significant.
Conclusions: *Helicobacter pylori* infection is statistically more frequently among the patients with CSC diagnosis than in healthy population.

Key words: *Helicobacter pylori*, central serous chorioretinopathy (CSC), the risk factors for the development of CSC.

Słowa kluczowe: *Helicobacter pylori*, centralna surowicza chorioretinopatia (CSC), czynniki ryzyka rozwoju CSC.

Introduction

Central serous chorioretinopathy (CSC) is a disease that affects young and middleaged adults, more often men than women. It is described as a neurosensory serous retinal detachment with sudden visual disturbances. The disease affects the macula region generally in the one eye. Bilateral and symmetrical presentation of the disease is reported to develop in only 10% of patients. Patients usually have mild visual loss. A lot of cases have a spontaneous recovery without therapy although the disease can become chronic with decompensation of the retinal pigment epithelium (RPE) and severe loss in visual acuity. Unfortunately the treatment of CSC is still unsatisfactory, probably because the pathogenesis of the disease is not clear.

The etiopathogenesis of CSC is still incompletely understood but the correlation with emotional stress periods is recognized probably through the way of the sympathetic – parasympathic activity leading to the development of defects in the RPE. Doubtless, the main reason of the development of CSC is vascular defects in choroidal circulation. The other known risk factors for the development of CSC are: using corticosteroids and sympathicomimetics, type A personality (1-6), vasomotorical vascular defects (7).

An association between CSC and *Helicobacter pylori* (HP) infection has been described.

HP is a gram – negative bacteria associated to multiple digestive and extra – digestive pathologies.

Our study suggest that HP may possibly be involved in the development of CSC and may be regarded as a risk factor for CSC.

Material and methods

We examined a group of 55 patients with diagnosis CSC – 19 women (34.5%) and 36 men (65.5%), age range 34-60 years, middle age 49.2, confirmed by fluorescein angiogram and 55 controls – 22 women (40%) and 33 men (60%), age range 30-60 years, middle age 46.7, deriving from hospitals' employees. Our patients not have been treated 6 months prior to the study with antibiotics, corticoids or sympathicomimetics drugs, they have not suffered any severe chronic disease, and the women have not been pregnant.

A total of individuals have been made a full ophthalmological exploration (visual acuity, Ishihara test, Amsler test, tonometry, eye fundus), including fluorescein angiography among the patients with CSC. The diagnostic criteria for CSC were: retinal pigment detachment in the fundus eye and CSC features in fluorescein angiography.

A control group has not a CSC history, past or present and has not a prior diagnosis of HP infection.

The HP study was with a non-invasive methods. Each patient provided venous blood sample for IgG antibodies to HP by enzyme – linked immunosorbent assay technique (ELISA) and a stool specimen for HP antigens.

The resultats was estimated using Chi² test and odds ratio.

Results

The prevalence of Helicobacter pylori active infection (positive results of stool examine) was 44% in CSC patients and only 29% in control group (Fig. 1, 2).

Using Chi² test $p = 0.11$, $p = 0.10$ is statistically significant. A difference of the frequency of antibodies IgG – anty

HP in the patients with CSC and in the control group was higher – 67% in th patients group and 47% in the control (Fig. 3, 4). In the Chi² test $p = 0.03$ and odds ratio = 2.29 it means that HP antibodies are over twice frequent among CSC patients than in controls. The difference is statistically significant.

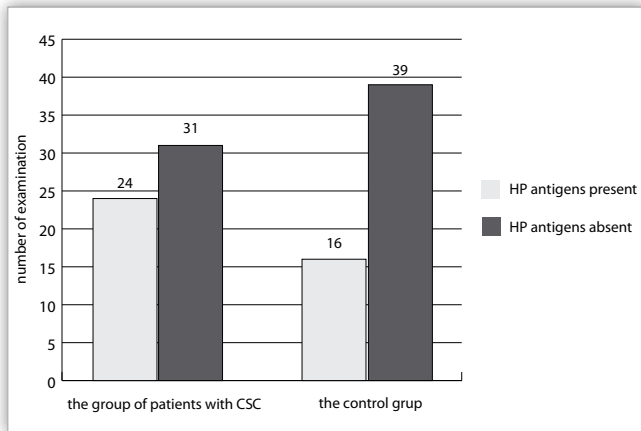


Fig. 1. Presence of HP antigens in the patients with CSC and in the control group.

Ryc. 1. Obecność antygenów HP u pacjentów z CSC i w grupie kontrolnej.

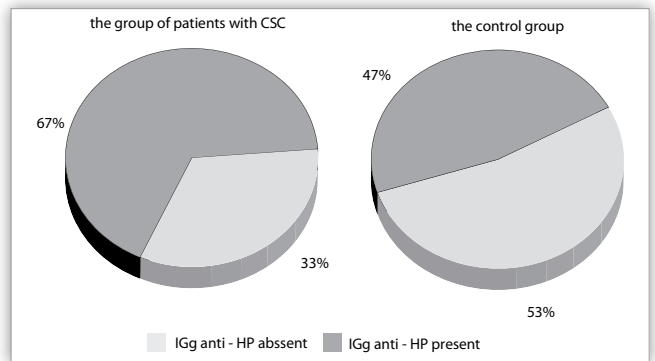


Fig. 4. A frequency of IGg anti – HP in patients with CSC and in the control group.

Ryc. 4. Częstość występowania IG anty HP u pacjentów z CSC i w grupie kontrolnej.

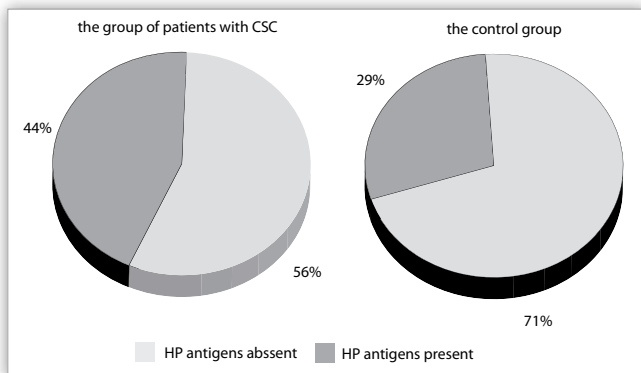


Fig. 2. A frequency of HP antigen in patients with CSC and in the control group.

Ryc. 2. Częstość występowania antygenu HP u pacjentów z CSC i w grupie kontrolnej.

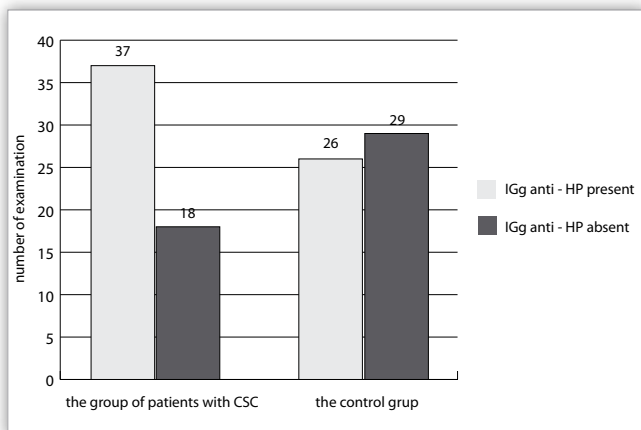


Fig. 3. Presence of IGg anti – HP in the patients with CSC and in the control group.

Ryc. 3. Obecność IG anty HP u pacjentów z CSC i w grupie kontrolnej.

Discussion

Typical CSC is a common disease that usually resolves with good vision after acute episodes. However in some cases recurrent of persistent disease may lead to permanent damage to the RPE and retina causing visual loss. The pathogenesis of CSC is unknown, but may be due to alterations in choroidal permeability with subsequent focal damage to the RPE. Risk factors of CSC are male gender, psychological stress, type A – personality, corticoid-steroid treatment and pregnancy.

A correlation between CSC and HP infection has recently been hypothesized. This association is still unclear. A possible explanation might arise from correlation between the HP infection and the development of atherosclerosis. Although the etiology of atherosclerosis is multifactorial, it has been documented that HP – cytotoxin – associated – gene A (CagA) may significantly increase the risk of its development. Anti – CagA antibodies may cross react with vascular wall antigens and begin an immunological cascade that causes arterial cell wall damage and leads to the development of atherosclerosis. Anti – CagA antibodies may destroy vascular endothelial cells and lead to the imbalance in vascular reactivity and permeability. Heat shock proteins (HSP) produced by HP make similar reaction with vascular wall antigens and have similar significance in making inflammation. An inflammation leads to defects in choroidal perfusion and to the ischaemia. Otherwise, the immunoglobulin – G (IgG) antibody response to the infection, can be a risk factor leading to the endothelial dysfunction. This theory of „molecular mimicry” might be an explanation of the prolonging the disease`s process, although the bacteria is eliminated (8-12).

Vascular endothelium regulates walls` permeability, hormones activations, coagulation and produces the factors regulating vascular spasm (7). The disturbances of endothelial cells` function may determine vasostenosis and ischaemical defects.

This process is probably responsible for changes in the retinal and choroidal tissue leading to the development of the CSC. The most probably is the focal occlusion of the choroidal microcirculation leading to the local ischaemia and local RPE damage.

If an auto-inflammatory etiology was solely responsible for the development of CSC, steroids would probably have been needed to suppress the inflammation. But it is documented that use of corticosteroid may cause the development of CSC (2).

However, independently of the kind of the pathogenesis of CSC, an association between HP infection and CSC has recently been documented and CSC could be an extra digestive expression of HP. Otherwise HP infection could be a risk factor of transformation the acute CSC in chronic and progresive process with decompensation of RPE and severe vision loss.

Conclusions

The results of this study show that the prevalence of Helicobacter pylori infection seems to be significantly higher in patients with CSC than in control group, and the difference is statistically important.

Helicobacter pylori infection may represent a risk factor for incidence of CSC.

CSC patients with accompanied HP infection have statistically more often a chronic form of CSC than other.

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