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Efficacy of Hericium erinaceus in the Treatment of Resistant Chronic Active HP Gastritis

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Abstract

Chronic gastritis (CG) is a worldwide prevalent disease frequently caused by the bacterium Helicobacter pylori (HP). Recent studies suggest that Hericium erinaceus (HE) induces clinical and histological improvements in patients with CG, as well as a pronounced antimicrobial effect against HP infection. Preclinical cellular findings also suggest an antineoplastic role in gastric carcinogenesis. The aim of this study is to examine this effect in patients with treatment-resistant chronic HP gastritis using four case studies.

Four female patients were enrolled in the study after providing written consent. Inclusion criteria: histological evidence of gastric HP infection following eradication therapy, adverse effects during the last eradication therapy, elevated HP IgA antibodies.

All patients (age: 49 years, range 33–56 years) met the inclusion criteria. HP-IgA antibodies were elevated in all patients prior to therapy (median: min;max = 27; 22.7; 31 U/ml – normal range < 20 U/ml), while HP-IgG antibodies were normal (25; 20.6; 31 U/ml – normal range < 35 U/ml). In one patient (47 years old) with a positive family history of gastric cancer, the CA72-4 tumor marker was markedly elevated prior to therapy (18.4 U/ml – normal range < 6 U/ml); however, gastric histology revealed only chronic active HP gastritis and no cancer cells. All patients were treated with HE (1.8 g daily - divided into 3 doses) for 3 months. All patients were free of symptoms after 3 months; HP IgA antibodies now were within the normal range (17:14; 18.3 U/ml), and HP IgG antibodies were elevated (36.6:34; 42 U/ml). In the patient with the elevated CA72-4 marker, the value decreased to 5.6 U/ml during therapy (normal range < 6 U/ml) and remained within the normal range at 6 U/ml even 6 months after HE therapy. In all 4 cases, histological examination during the follow-up gastroscopy after HE therapy revealed no further detectable HP colonization. No patient reported any side effects during HE therapy.

HE appears to effectively eradicate HP and normalize the CA72-4 marker in the treatment of chronic active HP gastritis. Further randomized, placebo-controlled studies would be desirable to demonstrate the efficacy of HE in the treatment of chronic active HP gastritis, as well as its excellent tolerability.

Introduction

Helicobacter pylori (HP) infection is widespread and affects approximately half of the world's population **[1, 2].** Chronic gastritis (GC) caused by H. pylori (HP) results from persistent chronic inflammation that leads to atrophic damage to the gastric mucosa, as the bacterium first activates the innate immune system and subsequently the adaptive immune system **[3].** This process leads to the formation of proinflammatory cytokines such as interleukin-1β (IL-1β) and tumor necrosis factor-alpha (TNF-α), which promote atrophic damage **[3].**Furthermore, HP can induce DNA damage through oxidative stress and epigenetic modifications via hypermethylation of tumor suppressor genes **[3].**Overall, these mechanisms contribute to an increased risk of developing gastric cancer **[4, 5]** and are the reason why HP has been classified by the International Agency for Research on Cancer

as a Group 1 carcinogen for gastric cancer **[6].**This requires immediate eradication, typically through a combination of multiple antibiotics and acid-suppressing medications (such as proton pump inhibitors), as recommended by leading international guidelines **[7, 8].**However, these therapies are associated with a certain risk of adverse events such as intestinal cramps and bleeding, as well as arrhythmias **[9, 10].**Therefore, there is an urgent need to identify a non-toxic and safe new active ingredient derived from nature or food sources. Previous studies have shown that the mycelial polysaccharides of Hericium erinaceum (HE) exhibit a variety of biological effects, including immune system support, antioxidant and anti-inflammatory effects, and protection of the gastric mucosa **[11, 12].** It has been reported that extracts from HE influences non-resistant (susceptible) pathogenic bacteria **[13, 17],** particularly on Helicobacter pylori **[18, 19],** a human gastrointestinal pathogen that causes ulcers, among other conditions, and is implicated in gastric cancer **[4, 5].,**The aim of this study is to examine the effect of HE in patients with treatment-resistant chronic HP gastritis using four case studies.

Methods

Four female patients with histological evidence of persistent active HP gastritis following eradication therapy, who had experienced side effects during their most recent eradication therapy and had elevated serum HP IgA antibodies, were informed about the possibility of HE treatment and enrolled in the study after providing written informed consent. To confirm successful eradication of HP, either mucosal infection with HP was assessed histologically before 3 months during HE treatment as well as HP antibodies IgA and IgG according to the results of Hayashi et al. **[20].** The following serum parameters were measured before and during 3 months of HE treatment:

Helicobacter pylori IgA and IgG (SERION ELISA classic, Virion/Serion, Würzburg): Detection of H. pylori-specific IgG and IgA antibodies was performed using an indirect enzyme-linked immunosorbent assay (ELISA). After binding of the antibodies to the antigen-coated microtiter plate and detection via an alkaline phosphatase conjugate, the optical density was determined photometrically at 405 nm. The antibody concentration (U/ml) was calculated based on the sample absorbance using a manufacturer-specific logit-log standard curve. The test system exhibits a diagnostic sensitivity of >99% and a specificity of >94% for IgG (for IgA, >94% and >85%, respectively), based on comparison with biopsy-based reference methods.

CA 72-4 (Roche): CA 72-4 is measured using a sandwich-format electrochemi-luminescence immunoassay (ECLIA) that employs biotinylated and ruthenium-labeled antibodies. After the immune complex binds to streptavidin-coated microparticles, it is magnetically captured on an electrode. Final quantification is achieved by inducing voltage-dependent chemiluminescence, the light intensity of which is directly proportional to the antigen concentration.

All patients were treated with 3 x 2 capsules of BIO-Hericium powder (300 mg per capsule in hydroxypropyl methylcellulose), Tyrolean Glückspilze, Austria, per day. A follow-up gastroscopy to confirm successful HP eradication was performed after 3 months.

Results

The 4 study participants-median age 49 years (min; max = 33; 56 years) suffered from episodic stomach pain for a median of 6.5 years (min; max = 4; 10 years), caused by a histologically confirmed HP infection. All participants received at least one course of bismuth quadruple eradication therapy (BQET). All four participants discontinued this BQET due to intestinal cramps and bleeding; in one case, cardiac arrhythmia also occurred during BQET. At study enrollment, all participants had histologically confirmed active HP gastritis. In all participants, the activity of the HP infection was confirmed by elevated IgA antibodies against HP (median: min; max – 27: 22.7; 31 U/ml – normal < 20 U/ml). Surprisingly, despite the long duration of infection (median: min; max – 6.5: 4; 10 years) and completed BQET, the IgG antibody against HP was within the normal range (median: min; max – 25: 20.6; 31 U/ml – normal < 35 U/ml). In one patient with positive family history of gastric cancer, the tumor marker CA72-4 was elevated at 18.4 U/ml (normal range < 6 U/ml) prior to treatment. All other participants had CA72-4 levels within the normal range. (Table 1). HE treatment was well tolerated throughout the entire treatment period (3 months) without any side effects. In the follow-up gastroscopy after 3 months under continuous HE treatment, no further histological evidence of HP infection could be detected. The normalization of IgA antibodies during HE treatment (median: min; max – 17: 14; 18.3 U/ml – normal range < 20 U/ml) confirmed the absence of HP activity, and the increase in IgG antibodies against HP following treatment (median: min; max – 36.6: 34; 42 U/ml – normal range < 35 U/ml) confirmed successful eradication due to HE. The CA72-4 tumor marker returned to normal levels during HE eradication in the 47-year-old participant with positive family history of gastric cancer (before: 18.4 – after: 5.8 U/ml – normal range < 6 U/ml). In all other participants, CA72-4 remained within the normal range during therapy (median: min; max – before 0.7: 0.6; 1.2 U/mL / after 0.7: 0.6; 0.9 U/mL – normal < 6 U/mL [Table 1]). The 47-year-old participant with positive family history of gastric cancer continued HE treatment for 12 month. Reevaluation of histological and serological HP activity were still negative. After 12 month of HE treatment CA72-4 stayed within normal range as well (before: 18.4 – 3m after: 5.8 U/ml – 12m after: 6,0 U/ml – normal range < 6 U/ml).

Discussion

In response to growing antibiotic resistance, increasing therapy dropouts due to severe side effects and therapy failure, there is increasing interest in natural products as complementary treatments. Phytochemicals such as terpenoids, flavonoids, alkaloids and polyphenols have shown promising anti-*H. pylori and* anti-inflammatory effects in both *in vitro* and

in vivo models **[21].** These compounds inhibit bacterial urease activity, reduce adhesion to gastric cells, modulate immune responses and protect mucosal integrity. Especially, flavonoid-rich extracts containing quercetin, catechin, and rutin exhibit multiple mechanisms including urease inhibition and oxidative stress reduction **[22].** This use of phytotherapy not only addresses the challenge of resistance and protection against gastric cancer but also offers no side effects **[23].**

HE provides numerous essential nutrients and constituents such as polysaccharides, proteins, lectins, phenols, isoindolinones, hericenones, erinacine terpenoids, and sterols, several of which possess various pharmacological properties **[25, 27].** HE extracts showed to be effective in stimulating the synthesis of immune system components **[25, 28].** which contribute to inhibiting tumor cell growth **[29].** Polysaccharide fraction derived from HE has also induced immunomodulating and anti-tumor effects **[29]** and anti-gastritis activity **[30].** Flavonoid extracts from HE fruiting bodies were reported to exhibit growth inhibitory effects on H. pylori by in vitro and/or in vivo studies **[31-33], [Figure 1].**

In our clinical cases, HE has demonstrated a histologically and serologically verifiable eradicating effect on HP and appears to possess antineoplastic properties. HE has a protective effect on the gastric mucosa, with the polysaccharide HEP-1 being the most important active ingredient **[34].** HEP-1 can improve blood flow to the gastric mucosa and strengthen its defensive function by accelerating the regeneration and repair of the gastric mucosal epithelium **[35-36].** HEP-1 can enhance the gastric mucosa's defensive capacity, and its molecular mechanism of action may improve the gastric mucosa's resistance to external stimuli as well as internal stimuli such as HP by promoting the activity of antioxidant enzymes, inhibiting the production of inflammatory factors, increasing the levels of eNOS-derived NO in gastric tissue, and upregulating the concentrations of EGF and VEGF **[37].** This mechanism explains the disappearance of HP in the histological mucosal findings following HE therapy in our study. In cell culture studies, HE demonstrates antineoplastic potential in gastric cancer, which is primarily mediated by two components: erinacin S and A **[38, 39].** Erinacin S induces a specific signaling pathway via reactive oxygen species, leading to the phosphorylation of focal adhesion kinase (FAK)/protein kinase B, also known as AKT, as well as p21-activated kinase 1 (PAK1) **[38].** This signaling pathway promotes the trimethylation of histone H3 and facilitates the production of various molecules, including the TNF-related apoptosis-inducing ligand and Fas ligand receptors, which trigger the caspase-3, -8, and -9-mediated apoptotic cascade **[38]**. Similarly, in gastric cancer cell models, modulation of the FAK/AKT/PAK1 signaling pathway by erinacin A can regulate two key proteins: microtubule-associated scaffold protein 2 and the 14-3-3-sigma protein **[39] [Figure 2].** This modulation negatively affects cell cycle regulation, particularly in the G2 and M phases, and exerts an antineoplastic effect. This provides an explanation for the normalization of the CA72-4 tumor marker during HE therapy in our case study.

Conclusion

HE treatment seems to be an effective alternative for patients with chronic active HP gastritis who are intolerant to conventional BQET; it can successfully eradicate HP from the gastric mucosa without side effects and exhibits antineoplastic properties. The small sample size limits the statistical power of our study and restricts the generalizability of our findings. Future research on HE to eradicate HP should focus on conducting rigourous clinical trials, standardising extract quality, and studying safety and pharmacokinetics. Large randomized clinical trials should be done to validate the findings and to demonstrate a protective effect against gastric cancer in long-term studies.

Table 1: Demographics and results before and after HE treatment. HE = Hericium erinaceum, SE = side effects, HP = Helicobacter pylori, BQET = bismute quadruple eradication therapy, AK = Antibody, IgA = Immunoglobuline A, IgG = Immunglobuline G, U/ml = Units per milliliter

age (years)	symptoms (years)	histology (SE of eradication) before	HP-IgA-Ak (norm: <20 U/ml) before	HP-IgA-Ak after	HP-IgG-Ak (norm: <35 U/ml) before	HP-IgG-Ak after	histology (SE) after	CA72-4 (norm: <6 U/ml) before	CA72-4 after
♀47	episodic gastric pain (7)	HP positive discontinuation of BQET (cramps, bleeding)	28	18	31	36	HP negative (none)	18,4	5,8
♀56	episodic gastric pain (10)	HP positive discontinuation of BQET (cramps, bleeding)	22,7	16,3	20,6	37,2	HP negative (none)	1,2	0,9

♀33	episodic gastric pain (4)	HP positive discontinuation of BQET (cramps, bleeding, arrhythmia)	26	18,3	21	34	HP negative (none)	0,6	0,7
♀51	episodic gastric pain (6)	HP positive discontinuation of BQET (cramps, bleeding)	31	14	29	42	HP negative (none)	0,7	0,6

Figure 01: Anti-Helicobacter pylori mechanisms of plant derived compounds – especially Hericium erinaceum. NF-κB = Nuclear Factor kappa B, IL-8 = Interleukin 8, COX-2 = Cyclooxygenase-2, CagA = Cytotoxin-Associated Gene A, VacA = Vacuolating Cytotoxin A (24)

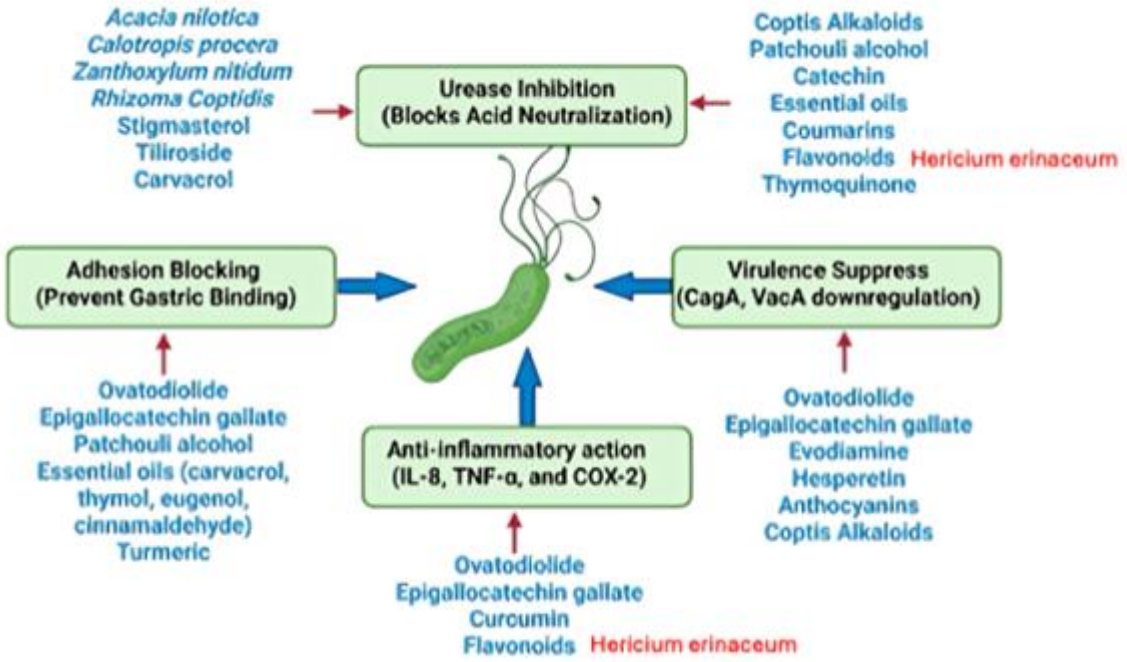
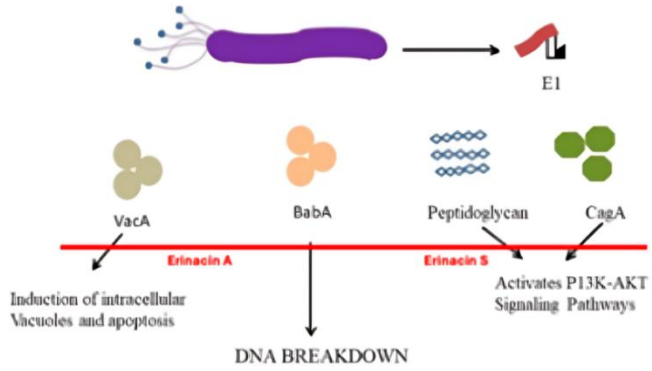


Figure 2: Carcinogic effect of Helicobacter pylori through different mechanisms blocked by Erinacin A and S (40)



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