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Scientific letter

Severe hyperemesis gravidarum caused by *Helicobacter pylori*☆

Hiperemesis gravídica severa causada por Helicobacter pylori

32-year-old female, 9 + 3 weeks pregnant with earlier diagnosis of antiphospholipid syndrome, gestational hypothyroidism and four miscarriages, undergoing treatment with folic acid, levothyroxine, progesterone, acetylsalicylic acid and enoxaparin. She was admitted from the emergency department with a diagnosis of hyperemesis gravidarum, in spite of being treated with oral doxylamine/pyridoxine and metoclopramide. Despite symptomatic management with intravenous metoclopramide and methylprednisolone, the patient was unable to tolerate oral fluids and solids for 9 days after being admitted and parenteral nutrition was required. After this time, despite partial relief of symptoms, the patient continued to vomit 3–4 times a day. Given her poor progress, a *Helicobacter pylori* antigen stool test was ordered, with positive results. Eradication treatment was initiated in week 11 + 3. A regimen of amoxicillin 1 g/12 h, metronidazole 500 mg/12 h and omeprazole 20 mg/12 h was prescribed for 14 days. Following treatment, at her 15 + 5 week obstetrics appointment, the patient reported a clear improvement in her general health with no nausea or vomiting. A breath test and an antigen stool test were performed one month after completing treatment, with negative results. All ultrasounds and blood tests performed throughout the rest of the pregnancy were normal. The patient had a spontaneous vaginal delivery in week 39 + 5. The newborn had an APGAR score of 9/10, a birth weight of 3.275 g and did not need to be admitted or resuscitated. All post-natal check-ups were normal.

Hyperemesis gravidarum is characterised by more than 3 vomiting episodes a day, ketonuria and weight loss of 3 kg or more or 5% of the patient's pre-pregnancy weight.[1] It affects 0.3%–2% of pregnancies[2] and is associated with poor weight gain during pregnancy, low birth weight, restricted intrauterine growth, preterm delivery and low 5-min APGAR scores.[3]

Helicobacter pylori (H. pylori) is a spiral-shaped, gram-negative bacterium that colonises the human stomach thanks to its mobility and acid resistance. The prevalence of H. pylori infection in Spain is higher than 50%.[4]

A recent meta-analysis shows a significant association between H. pylori infection and hyperemesis gravidarum (OR: 1.348; 95% CI: 1.156–11.539; $P < .001$).[2] This association opens up the way for improved diagnosis and management of the disease, allowing the cause and not only the symptoms to be treated. The physiological immunosuppression caused by pregnancy probably contributes largely to susceptibility to H. pylori infection or its reactivation. Once the pregnant patient has the infection, it colonises the antral gastric mucosa, releases toxins that damage the mucosa and produces localised inflammation, increasing nausea and vomiting.[5]

H. pylori is also associated with other complications of pregnancy: preeclampsia (OR: 2.51; 95% CI: 1.88–3.34; $P < .001$), foetal growth restriction (OR: 2.28; 95% CI: 1.21–4.32; $P = .01$), gestational diabetes (OR: 2.03; 95% CI: 1.56–2.64; $P < .001$), miscarriage (OR: 1.5; 95% CI: 1.05–2.54; $P = .03$) and birth defects (OR: 1.63; 95% CI: 1.05–2.54; $P = .03$).[6]

The use of non-invasive methods is preferred for diagnosing pregnant patients. The C^{13} urea breath test is the recommended non-invasive test,[7] although this may give false negative results in patients taking proton pump inhibitors,[8] which are common during pregnancy. Therefore, antigen stool tests may be more useful in pregnant women. This test is recommended as an alternative to the breath test[7] and is also the non-invasive method most commonly used in Spain.[4] Serological tests are not routinely recommended.

Very few studies suggest specific eradication therapy regimens during pregnancy. It is important to consider the possible teratogenicity or foetal toxicity of treatments and whether pregnant women with severe symptoms are candidates. The most commonly used therapy is combination therapy or monotherapy with erythromycin, clarithromycin, metronidazole and amoxicillin, proton pump inhibitors and antihistamines.[9] The duration of treatment is 5–14 days. Clarithromycin should not be used during the first trimester due to the increased risk of miscarriage (OR 1.56; 95% CI: 1.14–2.13).[10]

As in our case, H. pylori infection should be investigated in patients with severe hyperemesis gravidarum that is refractory to symptomatic treatment as this allows the cause to be treated and may prevent other adverse effects during pregnancy. Screening for H. pylori infection at the patient's antenatal appointment may be of interest provided that the usual eradication therapy poses no risk to the foetus.

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Conflicts of interest

The authors declare that they have no conflicts of interest.

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Pseudomonas monteilii nosocomial meningitis in a patient with an intraventricular catheter☆

Meningitis nosocomial por Pseudomonas monteilii *en paciente portador de catéter intraventricular*

A 78-year-old male was admitted to our hospital with an extra-axial space-occupying lesion suggestive of posterior fossa meningioma, which was later confirmed by nuclear magnetic resonance. Surgery was performed to remove the meningioma and to insert an external ventricular drain to remove excess cerebrospinal fluid (CSF) due to developing secondary hydrocephalus, with subsequent admission to the intensive care unit (ICU).

On day 13 in the ICU, the device was changed after the first device malfunctioned due to the presence of a clot blocking the ventricular portion of the catheter. On day 26, the patient experienced an accelerated decline in neurological function with a blood-like fluid observed in the drain, which was sent for microbiological study. Empirical antibiotic therapy was initiated with meropenem and linezolid.

Biochemistry testing of the fluid suggested a bacterial infection due to the presence of pleocytosis (1320 cells/ml), with 90% polymorphonuclear lymphocytes, low glucose levels (0.2 g/l) and high protein (1.5 g/l) and lactic acid (1.1 g/dl) levels.

A gram stain showed abundant polymorphonuclear leukocytes and gram-negative rods of variable length with no specific morphology. In view of these findings, it was decided to perform a molecular study using multiplex PCR (FilmArray®, BCID panel, bioMérieux), based on the recommendations of Micó et al.,[1] which was negative. Subsequently, a sample was cultured on blood agar, MacConkey agar and chocolate agar and incubated at 37 °C in aerobic conditions and 5% CO_2, respectively.

After 18 hours, growth in pure culture of non-pigmented mucoid colonies was observed on all three media (Fig. 1). The oxidase test was positive. The isolate was identified as *Pseudomonas fluorescens/putida* (99.9% probability) using a MicroScan Combo Panel, Type 71 (Beckman-Coulter, USA). It demonstrated sensitivity to standard doses of: meropenem, amikacin, tobramycin and colistin, and sensitivity with increased exposure to: piperacillin, ceftazidime, cefepime, ciprofloxacin and imipenem, based on EUCAST criteria.[2] Duplicate mass spectrometry (Microflex LT, Bruker Daltonics, USA) identified the isolate as *Pseudomonas monteilii*, with values of 2.25 and 2.10 using matrix only and values of 2.34 and 2.19 with pre-treatment with formic acid.

The final diagnosis was meningoencephalitis caused by *P. monteilii* as a result of infection of the drainage device and treatment was changed to ceftazidime, resulting in a rapid clinical improvement, which was confirmed by negative CSF culture results at 72 h.

The genus *Pseudomonas* is divided into three phylogenetic lineages and at least 19 groups and sub-groups. One of the most relevant groups is the *Pseudomonas putida* group, which comprises up to 15 strains, including *P. monteilii*,[3] which was first described in 1997.[4] Phenotypically it easily fits into this group based on Pickett's and Gilardi's identification schemes,[5,6] falling into the fluorescent group of non-fermenting gram-negative bacilli (GNB), together with *Pseudomonas aeruginosa* and *Pseudomonas fluorescens*. This phenotypic characterisation is still valid, despite the reclassification of *Pseudomonas* based on RNA/DNA homology studies and 16S rRNA gene sequencing-based characterisation.[7] Also, per routine clinical laboratory practices, *P. monteilii* can be quickly and safely identified using mass spectrometry.[8] *P. monteilii* is an environmental microorganism in healthcare settings and is often isolated from sink, tap and shower surfaces.[9] In this context, it must also be considered as a potential pathogen and, as such, it has also been cultured from clinical specimens such as bronchial aspirates, urine, stool, bile and blood.[4] Nevertheless, its role as a cause of central nervous system (CNS) infections has been rarely reported. In addi-

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