stent and thoracic CT with intravenous contrast enhancement revealed no mediastinal hematoma or extravagation of contrast medium to the mediastinum.

The case currently presented is, to the best of our knowledge, the first report of long-term data for the TEVAR approach in children. Although TEVAR is a challenge in this group of patients and is associated with many serious complications, our case shows that in some instances, TEVAR application in the pediatric population is promising in terms of long-term safety and efficiency.

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Keywords: post-traumatic, pseudoaneurysm, endovascular, young patient

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Treatment with risperidone and venlafaxine of a patient with double-coded diagnosis of body dysmorphic disorder and delusional disorder somatic type

Dear Editor,

DSM-IV classifies body dysmorphic disorder (BDD) under the somatoform disorders and its delusional variant as delusional disorder somatic type (DDST)[1]. The differential diagnosis is difficult, especially in cases with delusions of dysmorphobia, the subset of DDST which seems nosologically closest to BDD. DSM-IV, recognizing the diagnostic confusion in these definitions**,** allows these two variants to be double-coded, and delusional patients may be assigned both diagnoses[1]. The pharmacotherapy of these disorders has not been extensively studied. Proposals include antidepressants[2], antipsychotics[3] or a combination of both[2]. We present a patient with a double-coded diagnosis, treated with risperidone and venlafaxine.

A 26-year-old male was referred to our consultation-liaison unit by the dermatology department. The last six months he was preoccupied with beliefs that the skin of his arms became ill-structured, as evidenced by the appearance of 'abnormal skin folding'. He had already consulted dermatologists and plastic surgeons and had undergone minor cosmetic laser treatment for deformity correction.

Besides the aforementioned beliefs**,** he presented low mood. Due to his preoccupation with the perceived defects, he was withdrawn and abandoned his job. No other psychiatric symptoms were present. He had no prior psychiatric or substance abuse history, or family history of mental disorders. Physical examination and laboratory tests including MRI were normal.

A diagnosis of BDD and additionally of DDST was made, since his beliefs were held with delusional intensity. Pimozide, which was initially administered, was switched to risperidone (2mg/day, gradually increased to 4mg/day), due to extrapyramidal side effects. Simultaneously, due to deterioration in his mood, venlafaxine was introduced, 37.5mg/day, gradually increased to 150mg/day. No side effects developed. Four weeks later the symptoms improved significantly. Six months later he was free of symptoms.

Our patient fulfilled DSM-IV criteria for both BDD and DDST. It was suggested that, rather being distinct, the two disorders may constitute one entity[1]. Pimozide, which was suggested as the treatment of choice for DDST[3], caused intolerable side effects. Therefore, risperidone was administered**,** which has been reported as effective in the treatment of this disorder[3]. Venlafaxine, which was primarily administered for our patient's low mood, was also reported as effective in the treatment of BDD[4]. Additionally, the effectiveness of an antipsychotic-antidepressant combination in the treatment of DDST is established[2].

This case indicates that combination of risperidone and venlafaxine is effective in the treatment of BDD with somatic delusions. Patients may first seek advice by dermatologists and plastic surgeons, who should be aware of this condition and promptly request psychiatric consultation, for proper treatment and to avoid unnecessary, inefficient or even harmful interventions.

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Key words: dysmorphophobia, delusional disorder somatic type, antipsychotics, antidepressants

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Low aqueous humor ghrelin levels in open-angle glaucoma patients may correlate with Helicobacter pylori-associated apoptotic mechanisms

Dear Editor,

Recent evidence indicates that the aqueous humor ghrelin levels are significantly lower in primary open-angle (POAG) and pseudoexfoliation glaucoma (PXG) patients compared with cataract controls[1]. This finding has been attributed to: 1) glaucomatous neuropathy per se; 2) glaucoma medications; or 3) a higher intraocular pressure level[1].

Concerning the first possible explanation, ghrelin, playing an essential role in the gastric mucosal defense mechanism, has been closely associated with gastritis and Helicobacter pylori infection (Hp-I), mainly by controlling the apoptotic processes induced by Hp lipopolysaccharide (LPS)[2]; apoptosis is involved in several important ocular and gastrointestinal diseases, including glaucoma, Hp-induced gastrointestinal and/or extraintestinal diseases, comprising autoimmune and neurodegenerative ones, also associated with glaucomatous apoptotic neuropathy. Recently, we showed a high prevalence of Hp-I in POAG and PXG patients, and Hp eradication may positively influence glaucoma parameters[3]. Hp-I may influence the pathophysiology of glaucoma by releasing various proinflammatory and vasoactive substances or by being involved in the apoptotic process[3].

Furthermore, it has been assumed that circulating ghrelin can affect aqueous humor ghrelin levels by crossing the blood-ocular barrier (BOB), based on previous studies showing that ghrelin can pass the blood-brain barrier (BBB) in a complex and highly regulated process[1]. In this respect, a series of factors has been implicated in inducing BBB disruption, including inflammatory mediators (e.g., cytokines and chemokines induced by Hp-I) and oxidative stress[3]. In addition, Hp-induced cytotoxin VacA exhibits chemotactic activities to the bone marrow-derived mast cells (BMDMCs) and induces BMDMCs to produce proinflammatory cytokines including tumor necrosis factor (TNF)-α acting at a distance; TNF-α is involved in BBB disruption through a mechanism involving matrix metalloproteinases upregulation. Apart from activated mast cells, vascular endothelial growth factor, interleukin (IL)-8, chymase or tryptase and mast cell growth factor linked to Hp-I, mast cells themselves can be stimulated by corticotropin-releasing hormone, secreted under stress, to release the aforementioned mediators, which disrupt the BBB; its disruption could play an important role in promoting immune cell infiltration and pathogens' entry to the brain[4]. Besides, specific antibodies are found in increased levels in glaucoma patients' sera, and when these antibodies access the brain, due to BBB disruption, they are capable of killing retinal cells, thereby contributing to glaucoma[3].

Given that Hp-I and its related inflammation scores are significantly associated with lower ghrelin levels and that ghrelin protects gastric mucosal cells against Hp LPS-induced apoptosis[4], it would be interesting to elucidate in the future the concept that the low aqueous humor ghrelin levels found in glaucoma patients possibly correlate with a responsible apoptotic mechanism induced or not by Hp-I.

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