INTRODUCTION

Mondor’s disease is a rare, benign, and self-limiting inflammatory condition that affects the thoracoabdominal vasculature. Several predisposing factors are known, including breast surgery. This condition was first described by the French surgeon Henri Mondor in 1939 and has since been known as Mondor’s disease or syndrome. To date, its physiopathological mechanism remains poorly understood. Postoperative diagnosis of Mondor’s disease is important to ensure that the assistance and treatment provided to patients are adequate and reassuring.

CASE REPORT

A 54-year-old woman of average height and with a body mass index of 24.4 underwent mastopexy and breast augmentation with a 200mL textured prosthesis in October 2008. The results of preoperative examinations were normal, including hemography, coagulography, urinalysis, and electrocardiography to evaluate the surgical risk, and chest radiography. Mammography showed both breasts classified as category 2 (BI-RADS®) with benign radiological findings. Breast ultrasonography was also performed and showed normal breasts with moderate adipose substitution.

According to the patient’s medical records, she had undergone two caesarean sections and one hemorrhoid surgery, both successfully. It should be stressed that the patient smoked an average of 20 cigarettes per day.

Five weeks after surgery, the patient presented pain in the right anterior abdominal wall, running in a paramedian line on the right, below the surgical scar. A few days later, a thick fibrous cord had formed and mild skin hyperemia could be observed. The pain and cord were more evident when the patient raised the ipsilateral upper limb and/or the ipsilateral breast (Figure 1). The patient showed symptom regression and complete clinical remission within 3 weeks, with oral therapy consisting of...
of the nonsteroid anti-inflammatory drug nimesulide at 200 mg/day. In addition, the patient was asked to rest and topically apply a wet warm compress. The patient was also encouraged to quit smoking.

![Figure 1. Appearance of the fibrous cord and superior epigastric vein during the postoperative period, after mastopexy and breast augmentation surgery.](image)

**DISCUSSION**

The first description of compromised superficial vessels of the thorax and abdomen dates from 1869 and was performed by Fagge. The French surgeon Henri Mondor further described this condition in 1939; since then, the pathology became known as Mondor’s disease or syndrome. The physiopathological mechanism of this condition remains unclear, but it is currently known to involve the thrombosis of the superficial thoracoabdominal vasculature, with the thoracoepigastric, lateral thoracic, and superior epigastric veins being the most frequently involved.

The causes of this disease include trauma, surgery, breast cancer, tight clothes and compressive garments, excessive physical activity, vigorous sexual activity, biopsy or resection of the axillary or sentinel lymph nodes, and hepatitis C. All of these causes are related in some way with Virchow’s triad, that is, vasculature lesion, blood stasis, or hypercoagulability.

Despite compromising the vasculature, Mondor’s disease can also affect the lymphatic system, and some immunohistochemical markers have been identified for differentiation, such as CD31 and D240. However, any clinical application of these findings is scarce, as the treatment of the symptoms remains unchanged in both cases.

Mondor’s disease has also been described in other body regions such as the penis, neck, upper limbs, and inguinal region. Few reports indicated the development of this condition in the postoperative period of aesthetic surgery.

Complementary examinations performed for the diagnosis of Mondor’s disease include mammography and ultrasonography. Mammography can detect a limited number of findings with relevance to the diagnosis. In most cases, it only reveals superficial dilated tubular structures or alternatively, no alterations are reported. However, it is extremely relevant for the diagnosis of concomitant breast cancer, which can be the cause of the disease. The reported frequency varies between 1% and 12.7%. The ultrasonographic result can be normal or, alternatively, may reveal a superficial tubular hypoechoic structure that if associated with Doppler studies, can disclose interrupted blood flow. Laboratory tests are not required.

Clinical examination is of crucial importance, and the most commonly reported symptom is pain, followed by one or more superficial fibrous cords. The symptoms are aggravated when the patient performs movements of the affected region, which is characterized by hyperemia.

Inflammation lasts from 2 weeks to 6 months, and the pain can persist from 2 to 6 weeks.

Management of Mondor’s disease focuses on the symptoms, with the use of non-steroid anti-inflammatory drugs as the only treatment option. The use of corticosteroids, antibiotics, vaccines, anticoagulants, and even manual distraction procedures was reported, though all without any evidence of therapeutic benefits.

**CONCLUSION**

We can conclude that identification of Mondor’s disease in affected patients is important, despite its being a rare condition. Mondor’s disease is self-limiting and rarely requires any invasive approach for its remission, requiring only conservative procedures. Therefore, this report serves as a reassuring reminder for surgeons and patients that Mondor’s disease usually progresses favorably toward remission in most cases.

**REFERENCES**