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Case Report

Extensive omental-to-adnexal venous collateralization in a patient with history of tetralogy of Fallot and vena cava inferior agenesis: a case report

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ABSTRACT

Tetralogy of Fallot (ToF) is a complex congenital cardiac anomaly increasingly associated with long-term extracardiac vascular adaptations. While arterial collaterals in ToF, such as major aortopulmonary collateral arteries, are well documented, venous collateral pathways remain underreported. Congenital agenesis of the inferior vena cava (IVC), a rare anomaly with a prevalence of 0.0005–1%, typically results in extensive venous collateralization through the azygos, gonadal, and retroperitoneal systems. Omental collateralization, however, has not been described in this context. Herein this report presents a 34-year-old woman with surgically repaired ToF who underwent laparoscopic appendectomy for acute appendicitis. Intraoperatively, extensive dilated venous collaterals were observed coursing from the omentum toward both adnexa. The ovaries appeared enlarged with multiple cystic nodules. Postoperatively, persistent pelvic pain prompted further evaluation. Contrast-enhanced computed tomography (CT) revealed congenital agenesis of the infrarenal IVC with collateral drainage via parauterine and omental venous plexuses. The left ovary demonstrated multiple cystic lesions up to 4.2 cm, consistent with chronic venous congestion rather than acute gynecologic pathology. Transvaginal ultrasound confirmed resolving cystic changes and varicose parauterine and omental plexuses. This case illustrates a previously undescribed omental–ovarian collateral circuit in the setting of IVC agenesis, coinciding with repaired ToF. The anomaly likely contributed to chronic pelvic pain and polycystic ovarian morphology, underscoring the clinical significance of venous remodeling in congenital vascular disease. Recognition of such variants is critical, as they may mimic pelvic masses on imaging and complicate surgery through increased bleeding risk. Awareness of rare collateral pathways expands the spectrum of venous adaptations and highlights implications for surgical, radiological, and reproductive management.

Keywords: Tetralogy of fallot, Venous collaterals, Omentum, Pelvic congestion, Agenesis, Vena cava inferior

INTRODUCTION

Tetralogy of Fallot (ToF) is a complex congenital cardiac malformation characterized by four anatomical features: a ventricular septal defect, pulmonary stenosis, overriding aorta, and right ventricular hypertrophy.[1] Thanks to improvements in early surgical repair, survival rates into adulthood now exceed 90%, shifting clinical focus towards long-term complications related to altered cardiovascular physiology and secondary vascular remodeling.[2]

While arterial collaterals in ToF are well described, particularly major aortopulmonary collateral arteries (MAPCAs), venous collateral pathways in the abdomen and pelvis have scarcely been reported.[3]

In this context, congenital agenesis of the inferior vena cava (IVC) deserves particular attention. Agenesis or hypoplasia of the IVC is a rare developmental anomaly, reported in 0.0005–1% of the general population.[4-6] It typically induces extensive venous collateralization involving the azygos–hemiazygos, gonadal, and retroperitoneal systems. The omentum, with its rich venous network, provides an additional but underrecognized route for collateral formation under chronic vascular stress.[7]

We report the case of a young woman with surgically corrected ToF who underwent laparoscopic appendectomy for acute appendicitis. Intraoperatively, extensive omental collaterals draining into the bilateral adnexal veins were observed. These were most likely the direct consequence of IVC agenesis, rather than her repaired ToF. The vascular anomaly coincided with a polycystic ovarian appearance on imaging, raising important questions about underlying pathophysiology and clinical relevance.

CASE REPORT

The 34-year-old patient with a history of ToF surgically repaired in childhood. She had experienced nonspecific lower abdominal pain for several years. She presented to the emergency department with acute right lower quadrant pain and was diagnosed with acute appendicitis based on clinical and laboratory findings.

During laparoscopic appendectomy, an extensive dilated tortuous venous vessels coursing from the omentum toward both adnexa was encoutered. The ovaries appeared enlarged and exhibited multiple cystic nodules. The appendectomy proceeded without complications, and the patient was discharged in stable condition.

Following surgery, her chronic lower abdominal discomfort persisted. Several weeks later, she re-presented to the emergency department with ongoing pelvic pain. A contrast-enhanced abdominal computed tomography (CT) scan was obtained, revealing a prominent left adnexal mass composed of multiple thick-walled cystic structures measuring up to 4.2 cm. These findings were interpreted as likely enlarged corpus luteum cysts with underlying inflammatory changes or possibly tubo-ovarian abscesses. Importantly, the scan confirmed the prominent venous collaterals involving the parauterine and omental plexuses, consistent with chronic venous congestion. The CT further demonstrated congenital agenesis of the infrarenal IVC, with absence of the right femoral and iliac venous confluence. The omental veins thus represented part of the collateral network compensating for this anomaly (Figure 1).

A subsequent transvaginal ultrasound confirmed normal uterine morphology. The left ovary measured 79×59 mm, contained a collection approximately 52 mm in diameter, and a smaller follicular cyst of 19 mm, suggesting resolving inflammatory or cystic lesions. Varicose parauterine and omental venous plexuses were noted, correlating with our findings seen during surgery and on CT imaging (Figure 2).

Gynecological evaluation concluded that the inflammatory process had likely regressed, and no acute pathology warranted intervention. The chronic venous congestion was considered a likely consequence of the anomalous omental-ovarian collateral circulation.

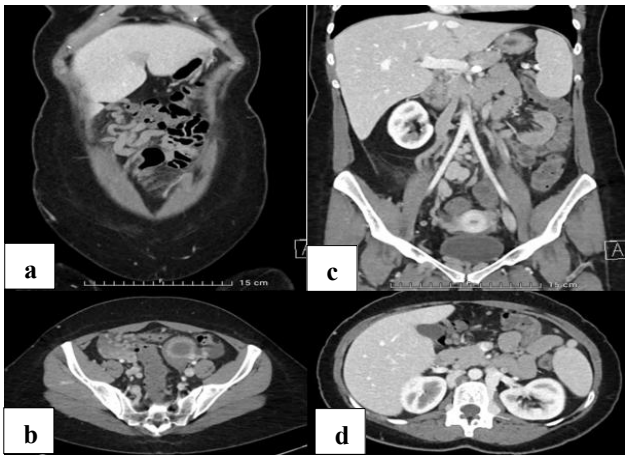

Figure 1: (a) Coronal section demonstrating the omental conglomerate located paramedian to the right beneath the abdominal wall, (b) paramedian right view showing the omental conglomerate draining the right ovary, (c) absence of the inferior vena cava, and (d) shows development of an extensive lumbar collateral network as an alternative venous pathway.

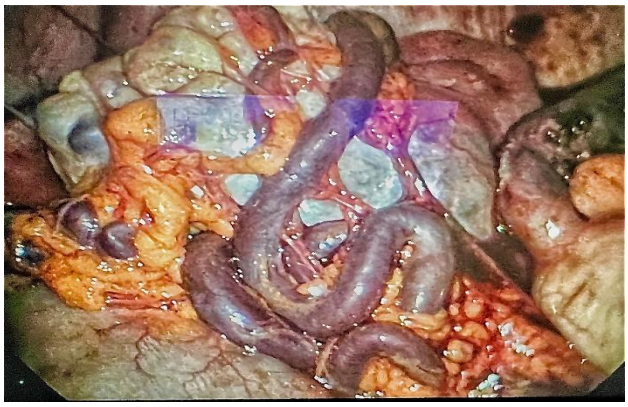

Figure 2: Intraoperative view showing omental varices draining towards the right ovary. The inflamed appendix is seen on the right.

DISCUSSION

This case illustrates a unique venous collateral circuit between the omentum and bilateral adnexal veins in a patient with surgically corrected ToF and associated agenesis of the infrarenal IVC, accompanied by polycystic ovarian morphology and chronic pelvic pain. Such venous collaterals have not been previously described in the

context of congenital heart disease, raising questions about their pathogenesis and clinical implications.

The pathophysiology of venous collaterals in ToF patients is complex. Collateral vessels typically develop as compensatory mechanisms in response to chronic venous hypertension or vascular obstruction.[8,9] While arterial collaterals such as MAPCAs are well documented and studied for their implications in pulmonary blood flow, venous collateral pathways tend to receive less attention. Among known venous collaterals, azygos continuation of the inferior vena cava and portocaval shunts are described in association with congenital heart disease, including ToF).[10,11] However, an omental-ovarian venous collateral circuit has not been reported in the literature. Congenital agenesis or hypoplasia of the IVC is a rare but clinically significant vascular anomaly, with prevalence ranging from 0.0005% to 1% of the general population, and up to 5% in young patients presenting with unprovoked deep vein thrombosis (DVT).[4-6] It results from failure in the complex embryological development of the IVC during weeks 4–8, involving the posterior cardinal, subcardinal, and supracardinal veins. The condition is often asymptomatic and discovered incidentally during imaging, surgery, or after a thrombotic event. It promotes venous return via collateral pathways—including the azygos–hemiazygos system, retroperitoneal venous plexuses, and gonadal or pelvic veins—that can manifest as varices or congestion.[4-6,12]

The omentum's vascular network is known for its plasticity and capacity to form collaterals under ischemic or congestive conditions.[13] The presence of extensive dilated veins connecting the omentum to the adnexal veins suggests chronic adaptive remodeling in response to altered central venous pressures or localized venous hypertension. Such adaptations may result from the altered hemodynamics following ToF repair or from residual circulatory abnormalities.

In our patient, the omental–ovarian varices are most plausibly attributed to compensatory collateral flow due to IVC agenesis, rather than being directly related to ToF. Nonetheless, the coexistence of ToF and IVC agenesis underscores the propensity of patients with significant congenital vascular malformations to harbor multiple anomalies. The chronic venous congestion likely contributed to the observed polycystic ovarian morphology, via impaired ovarian drainage, follicular stasis, and stromal edema.[14,15]

Clinically, recognizing IVC agenesis is essential: it can mislead radiological interpretation, complicate surgical planning due to the risk of bleeding from collaterals, and may affect reproductive health through venous congestion. Moreover, affected individuals may be at risk for DVT and thus require vigilant assessment and potential long-term anticoagulation. This case highlights key clinical considerations. Surgeons should be aware of venous collateral networks in patients with congenital heart disease, as unrecognized dilated collaterals can increase bleeding risk and complicate surgery. Careful preoperative imaging and planning are essential.

Radiologists should also recognize these vascular variants, as dilated collaterals may mimic cystic or inflammatory pelvic masses, potentially leading to misdiagnosis. Multimodal imaging with Doppler ultrasound and contrast-enhanced CT or magnetic resonance imaging (MRI) can improve accuracy.

Finally, while the impact of chronic ovarian venous congestion on fertility remains unclear, it warrants attention, as prolonged venous stasis could affect ovarian function and endometrial receptivity. Further studies in congenital heart disease survivors are needed to clarify these effects.

CONCLUSION

We present a rare and previously undescribed case of extensive omental–ovarian venous collaterals in a patient with repaired ToF, associated with congenital agenesis of the infrarenal IVC, polycystic ovarian morphology, and chronic lower abdominal pain. The unique collateral pathway expands the spectrum of venous adaptations in congenital vascular anomalies and highlights its clinical significance for surgical, imaging, and reproductive clinicians. Awareness of this variant is crucial to avoid diagnostic errors and surgical complications. Further research is necessary to elucidate the pathophysiological mechanisms and long-term clinical outcomes related to such venous anomalies.

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