Vol. 50 (2025) No. 3&4 TIMOK MEDICAL GAZETTE Review article

OVARIAN TORSION - review paper

Nataša M. Janković (1), Miloš J. Janković (2), Sanja Dimitrijević (3)

1) PIROT HEALTH CENTER, WOMEN'S HEALTH CARE SERVICE; 2) DEPARTMENT OF INTERNAL MEDICINE, GENERAL HOSPITAL PIROT; 3) CENTER FOR GYNECOLOGY AND HUMAN REPRODUCTION, MILITARY MEDICAL ACADEMY BELGRADE

Summary: The largest number of ovarian torsions is seen in the reproductive period, around 71%, but it also occurs in fetuses and neonates, premenarchal girls, pregnant women, and postmenopausal women. Although the true incidence of torsion is still unknown, data show that torsion accounts for 2.7% of surgical interventions, making it the fifth most common condition requiring emergency surgery. To understand how torsion occurs, we must understand the anatomy of the supporting structures of the uterus and ovaries. The ovary is a mobile structure in itself, suspended from the pelvic wall by the infundibulopelvic ligament and attached to the uterus by the utero-ovarian ligament. Torsion occurs as a result of partial or complete rotation of the adnexal supporting structures, which leads to partial or complete obstruction of ovarian blood flow, resulting in ovarian ischemia, which leads to necrosis, local hemorrhage, and loss of function. Torsion can occur in women of all reproductive ages, but the highest percentage of ovarian torsions occurs in women in the reproductive period with adnexal changes such as functional ovarian cysts and benign tumors. The most common symptom is pain, which may be accompanied by nausea and vomiting. Physical examination may reveal low-grade fever, abdominal tenderness, abdominal pain, and a pelvic adnexal mass. Diagnosing ovarian torsion usually requires a combination of medical history, clinical examination, and imaging methods. Early diagnosis and surgical treatment are essential to preserve ovarian and tubal function and to prevent more serious morbidity..
Keywords: Ovarian torsion, ovarian cyst, benign tumor, acute abdomen

INTRODUCTION

The most common conditions that lead to acute pain in gynecological practice include ectopic pregnancies, pelvic inflammatory disease, ruptured ovarian cysts, ovarian torsion, torsion and degeneration of uterine leiomyomas, and spontaneous miscarriages. The largest number of ovarian torsions is seen in the reproductive period, around 71% [2], but it also occurs in fetuses and neonates, premenarchal girls, pregnant women, and postmenopausal women. Although the true incidence of torsion is still unknown, data show that torsion accounts for 2.7% of surgical interventions, making it the fifth most common condition requiring emergency surgery [2]. Another study found that 15% of surgically treated adnexal masses are in torsion [3]. Timely diagnosis is important both for preserving ovarian function and for preventing subsequent comorbidities..

PATHOGENESIS AND RISK FACTORS

To understand how torsion occurs, we must first understand the anatomy of the supporting structures of the uterus and ovaries. The ovary is a paired intraperitoneal organ with two primary functions: the production of sex hormones, and the development and release of the oocyte during ovulation, as well as the formation of the corpus luteum, which provides sufficient hormonal support to early pregnancy until placental function is established. What is specific and remarkable about the ovary is that it can increase its volume several hundred times during a woman's reproductive period without pathological clinical manifestations [4].

The ovary is a mobile structure, suspended from the pelvic wall by the infundibulopelvic ligament (also called the suspensory ligament of the ovary), through which the ovarian artery passes, and attached to the uterus by the utero-ovarian ligament (ligamentum ovarium proprium), through which the ovarian branch of the uterine artery passes. In addition to providing support, these ligaments also serve a nutritive role, as the blood vessels supplying the ovary run through them, ensuring dual vascularization of the ovary [5].

Torsion occurs as a result of partial or complete rotation of the adnexal supporting structures, during which the ovary and fallopian tube rotate around both the infundibulopelvic and utero-ovarian ligaments, resulting in partial or complete obstruction of ovarian blood flow [6,7,8]. The thin walls of the veins are more prone to complete occlusion compared to the muscular walls of arterial vessels. Continuous arterial inflow without venous outflow leads to edema with visible ovarian enlargement. Further vascular compression results in ovarian ischemia, leading to necrosis, local hemorrhage, and loss of function [9]. Most often, both the ovary and the fallopian tube undergo torsion simultaneously, although isolated torsion of the ovary or tube may occur, referred to as partial torsion. Torsion involving paraovarian or paratubal cysts has also been described [6]. The right ovary is more frequently affected than the left, possibly because the right utero-ovarian ligament is longer, and the presence of the sigmoid colon prevents torsion on the left side [8,10]. Bilateral asynchronous ovarian torsion is also possible, though rare [11]. The severity of symptoms and morphological ovarian changes depends on the type and degree of vascular occlusion. Based on anatomical features and clinical findings, we can define risk factors for adnexal torsion. Greater ovarian mobility is associated with torsion in premenarchal girls, who have elongated infundibulopelvic ligaments. In this population, more than half of patients have morphologically normal ovaries. After this premenarchal period, with puberty, the incidence of ovarian torsion decreases due to shortening of the infundibulopelvic ligaments. Risk factors in premenarchal girls may also include the presence of functional cysts or benign tumors, most commonly teratomas and cystadenomas [12,13,14].

Ovarian torsion has also been described in the fetal period (ultrasound may monitor cyst growth and secondary changes such as hemorrhage, calcifications, or resorption) and in neonates [15].

The highest percentage of ovarian torsions occurs in women of reproductive age with adnexal changes such as functional ovarian cysts and benign tumors [8,9,16]. Malignant tumors and endometriotic cysts are rarely the cause of ovarian torsion. In case series, the percentage of malignant ovarian tumors associated with torsion is reported to be below 3%. This is because such lesions cause peritoneal reactions and adhesions that fix the mass, thereby limiting its mobility [17]. More than 80% of patients with ovarian torsion have ovarian masses larger than 5 cm in diameter. The size of the ovarian mass correlates with the risk of torsion. In a series of 87 case studies, ovarian masses ranged widely from 3 to 30 cm, with an average of about 9.5 cm [18]. About 10–22% of ovarian torsions occur during pregnancy. The incidence is somewhat higher between the 10th and 17th weeks of gestation in the presence of ovarian masses larger than 4 cm. Ovulation, the corpus luteum, and ovulation induction in infertility treatment may cause ovarian hyperstimulation syndrome, with multiple large cystic ovarian changes that are prone to torsion. Polycystic ovary syndrome is also a risk factor [19]. On the other hand, in patients who have undergone a surgical procedure, the incidence of ovarian torsion is about 2–15%, typically due to strangulation of the ovarian pedicle around an existing adhesion. Recurrent torsion has also been described, and studies show that individuals who have experienced ovarian torsion once are at increased risk of developing torsion again—either of the same ovary ("salvage ovary") or of the contralateral ovary. [8].

Clinical presentation and clinical findings Ovarian torsion caused by the presence of an adnexal mass results in a variety of symptoms, clinical signs, and presentations. The most common symptom is acute, sharp pain in the lower abdomen or pelvis, accompanied by nausea and vomiting (70%) in women of reproductive age, in the presence of an adnexal mass or enlarged ovaries in PCOS or ovarian hyperstimulation, or in women with a history of prior ovarian torsion [17,18,20]. Some patients experience only nausea without vomiting. Abdominal pain is most often intermittent, colicky in nature, with gradual intensification and relief, although it may also be continuous. The pain arises secondarily due to occlusion of the vascular pedicle and is refractory to analgesics. It may radiate to the inguinal region or flank. Premenarchal patients may report diffuse abdominal pain, as they often find it difficult to localize the discomfort. In this group, vomiting is the most common symptom in the absence of adnexal pathology—this represents a vagal reflex response due to peritoneal irritation. In neonates, torsion may present with feeding difficulties, abdominal distension, vomiting, and irritability. Ovarian torsion without infectious pathology may also be accompanied by low-grade fever. The low-grade fever is explained by necrotic changes in the torted ovary and occurs in 2–20% of patients. Physical examination may reveal low-grade fever, abdominal tenderness, abdominal pain, and a pelvic adnexal mass. A further diagnostic

challenge is that 30% of patients—especially those in the premenarchal period—may have neither abdominal pain nor abdominal tenderness. [17,18,21-24].

Diagnosis

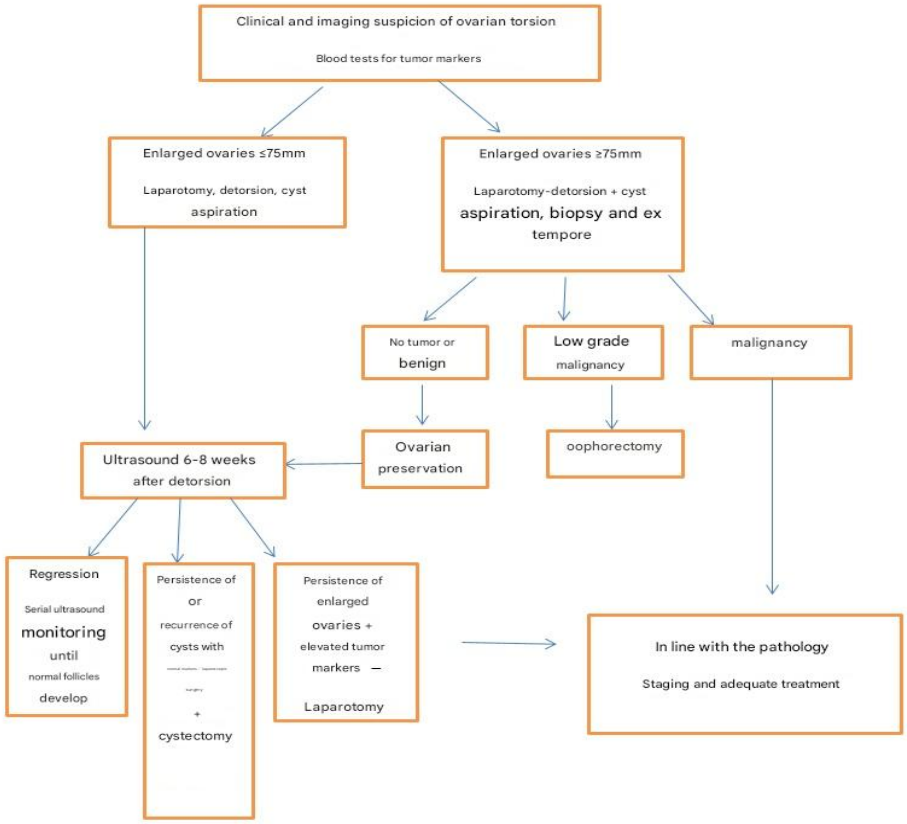
The diagnosis of ovarian torsion most often requires a combination of anamnestic data, clinical examination, and imaging methods. The first approach to the patient is the physical examination and taking the medical history. Anamnestic data may indicate a recent diagnosis of an adnexal mass, recurrent abdominal pain, and low-grade fever. In children aged 2–14 years, with high sensitivity and positive predictive value, the Bolli score can be applied. The Bolli score includes only the patient's clinical data but not imaging methods and identifies three useful clinical variables on the basis of which the ovarian torsion score is established: the age of the child, the duration of pain, and vomiting. –Number of points – number of years, minus three points if vomiting is present, plus one point if the duration of pain is longer than 12 hours. The cut-off value of the Bolli score in girls is 11.5, a lower score indicating a higher probability that ovarian torsion is present. [25,26].

LABORATORY TESTING should include hematocrit, leukocyte count, human chorionic gonadotropin (HCG), electrolytes, and inflammation parameters—C-reactive protein (CRP) [2,22]. Laboratory analyses may be completely normal, may indicate anemia in the case of corpus luteum rupture, or leukocytosis and elevated CRP due to tissue necrosis and consequent inflammation. The level of interleukin 6 is also elevated and indicates increased oxidative stress in torsion, but it is also a nonspecific sign of inflammation and is not routinely performed in our clinical practice [27,28]. Determining tumor markers has not proven to be sufficiently sensitive or specific, although the elevation of certain tumor markers may indicate the nature of the torted adnexal mass. The physical exam is focused on abdominal palpation in order to detect a tumor mass and assess the presence of peritoneal irritation. Imaging studies are the most important. Ultrasound is the first-line diagnostic tool [29,30].

In the pediatric population, transabdominal ultrasound with a full bladder is the initial imaging method for evaluating torsion. The sensitivity of transabdominal ultrasound in the pediatric population is 92–93%, with a specificity of 96–100%. In adult women, transvaginal ultrasound shows excellent specificity but variable sensitivity, ranging from 35–85% [31]. What is monitored on ultrasound is ovarian volume, presence of edema, presence of an adnexal mass, presence of free fluid, and color Doppler of ovarian or tumor mass blood vessels. The presence of a difference in ovarian volume with its displacement is a pathognomonic sign of ovarian torsion. Another sign is the presence of edema of normal ovarian tissue. In the literature, it is described as the presence of peripheral follicles with hyperechogenic halos in the ovary without cystic changes or tumors — strings of pearls. The presence of ovarian edema should not be mistaken for the presence of a solid ovarian tumor.

The torted ovary may be rounder and enlarged compared to the contralateral one due to swelling of vascular and lymphatic vessels. There may be normal, reduced, or completely absent blood flow through the vessels of the torted ovary [31–34]. The whirlpool sign is a highly sensitive and specific sign for the diagnosis of ovarian torsion. The whirlpool sign indicates the twisted vascular pedicle, and Doppler sonography reveals circular blood vessels within the mass [32]. Finally, a small amount of free fluid may be present in the pouch of Douglas [31]. The greatest diagnostic challenge is torsion without twisting of the ipsilateral ovary. It has been shown that 31% of all torsions are incomplete adnexal torsions. A useful sign of torsion involving only the tube but not the ovary is the presence of three or more cysts in one row [35]. The combination of free fluid in the pelvis, an enlarged ovary, and vascular abnormalities increases the sensitivity and specificity of ultrasound findings. CT of the abdomen and pelvis shows high sensitivity and specificity in the evaluation of suspected torsion [36] and may show an enlarged ovary, its displacement and pulling of the uterus to that side, thickening of the cystic mass, ascites, thickened walls of the tube [36,37]. The definitive diagnosis is made in the operating room by direct visualization of the specimen..

Algorithm 1. Algorithm for management in cases of clinical and imaging suspicion of ovarian torsion.



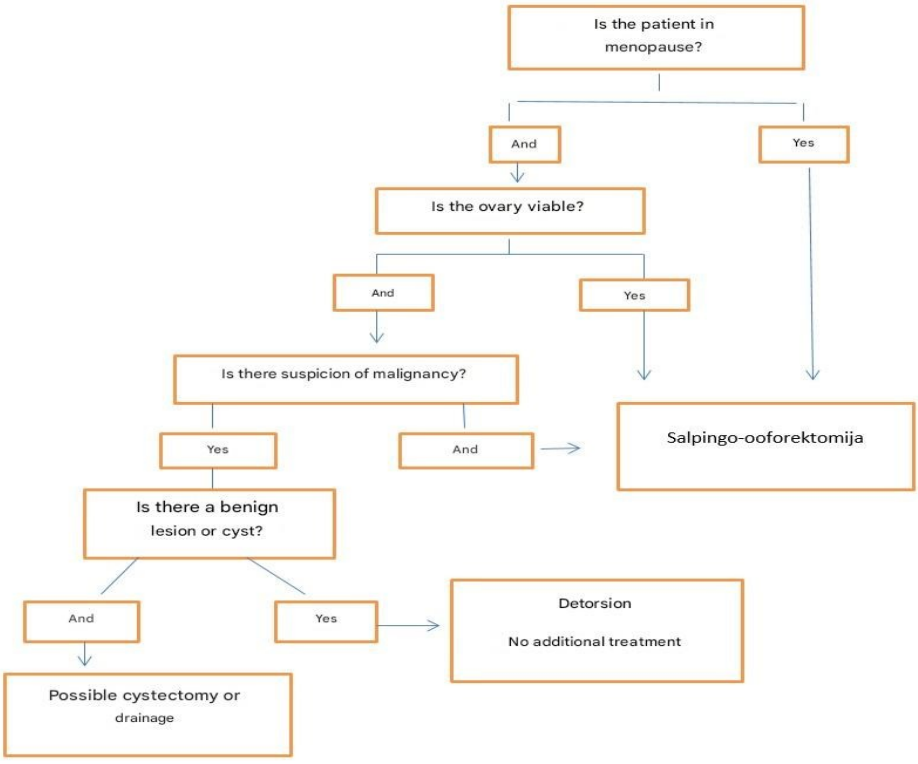
Treatment and assessment of ovarian viability

Treatment involves surgical management and at the same time confirmation of the diagnosis. Early diagnosis and surgical therapy are necessary in order to protect ovarian and tubal function and to prevent more serious morbidity. Minimizing the total time during which the ovary is in ischemia is a key component of therapy, but the time required for ovarian necrosis to occur is unclear. [37,38].

Picture 1. Surgery of a torted ovarian fibroma
(Dr. Janković, General Hospital Pirot))

As long as the venous and lymphatic vessels are occluded, the patient may have symptoms for some time before the arterial vessels become occluded [30,40,41]. In a retrospective study of the pediatric population, the median time to save the ovary before detorsion was 10.8 hours. If detorsion is performed within the first 8 hours, the ovary is preserved in 40% of cases, and within the first 24 hours in 33% [42]. This finding is consistent with data showing that in women, the percentage of preserved ovaries is 30% if surgery is performed within the first 24 hours from the onset of symptoms [43,44]. Different studies show varying times from symptom onset to detorsion in order to preserve the ovary. Animal studies have shown that necrosis can occur 36 hours or more after occlusion. Pediatric and adult populations show good long-term outcomes after detorsion of either hemorrhagic or ischemic ovaries, with normal follicle production later in life in 90–94% of cases described [45,46]. There are two surgical treatment methods — laparoscopy and laparotomy. Laparoscopy represents a reasonable alternative. The benefits of laparoscopy include reduced need for analgesics, early mobilization, cosmetic advantage, and earlier discharge to home care. An additional advantage is that laparoscopic ovarian cystectomy is associated with a lower incidence of postoperative adhesions compared to laparotomy [47]. Laparotomy is recommended when a malignant process is suspected. What is essential is the assessment of ovarian viability and preservation of its function. The only way to assess the viability of the ovary is by gross visual inspection. Conventionally, a dark and enlarged ovary may be only in venous or lymphatic congestion and may appear nonviable, but there is a substantial probability that it is a viable ovary that can regain function after detorsion [46]. There are other methods to assess ovarian viability, such as injecting fluorescein and observing the flow under ultraviolet light [48]. Another method is ovarian bivalving, i.e., laparoscopically making an incision in the ovary with an electric hook (L-hook) after detorsion and observing whether there is blood flow at the cut surface. This also serves a therapeutic purpose by reducing pressure caused by venous and lymphatic congestion [49]. There is no precisely determined time for ovarian necrosis to occur. A definitive sign of ovarian necrosis is a gelatinous formation that disintegrates upon manipulation. What is expected from surgical treatment? Ideally, detorsion [50], detorsion with oophoropexy, ovarian cystectomy (recommended for benign cysts after detorsion), or salpingo-oophorectomy in cases of suspected malignancy, necrotic ovaries, and postmenopausal women.

Algorithm 2. Procedure in menopausal women



CONCLUSIONS:

- Ovarian torsion mainly affects women of reproductive age, but a significant percentage also occurs in the premenarcheal period, in pregnant women, and in postmenopausal women.
- • Ovarian torsion occurs due to complete or partial rotation of the ovary and fallopian tube, leading to obstruction of vascular flow.
- • Crucial factors for ovarian torsion include the presence of an ovarian mass in women of reproductive age.
- • Almost 90% of women experience abdominal pain that begins suddenly, is sharp, and intermittent in nature.
- • Up to 70% experience nausea and vomiting.
- • Nearly one-third of patients with torsion have no abdominal or pelvic pain on examination.
- • Although ultrasound is used as the primary diagnostic modality in the evaluation of ovarian torsion with high specificity, a normal ultrasound finding cannot effectively rule out the diagnosis.
- • CT of the abdomen and pelvis with IV contrast can be helpful in diagnosis.
- • In the evaluation, findings for reduced or absent ovarian enlargement, peripheral displacement of follicles, enlarged ovaries with follicular stroma, and thickened fallopian tube are considered significant

REFERENCE:

1. Franco PN, García-Baizán A, Aymerich M, Maino C, Frade-Santos S, et al. Gynaecological Causes of Acute Pelvic Pain: Common and Not-So-Common Imaging Findings. Life (Basel).2025;13(10):2025.
2. Hibbard LT.Adnexal torsion. Am J Obstet Gynecol 1985;152:456-61.
3. Bouguizane S, Bibi H, Farhart Y, et al. Adnexal torsion: a report of 135 cases. J Gynecol Obstet Biol Reprod. 2003;32(6):535–40.
4. Gibson E, Mahdy H. Anatomy, Abdomen and Pelvis, Ovary. StatPearls Publishing, Treasure Island 2019.
5. Ying J, Feng J, Hu J, Wang S, Han P, Huang Y, et al. Can ovaries be preserved after an ovarian arteriovenous disconnection? One case report and a review of surgical treatment using Da Vinci robots for aggressive ovarian fibromatosis. J Ovarian Res. 2019;12(1):52.
6. Huang C, Hong MK, Ding DC. A review of ovary torsion. Tzu Chi Med J. 2017;29(3):143-7.
7. Sanfilippo JS, Rock JA. Surgery for benign disease of the ovary. In: TeLinde's Operative Gynecology, 11th ed., Jones HW, Rock JA (Eds), Wolters Kluwer, 2015.
8. Beaunoyer M, Chapdelaine J, Bouchard S, Ouimet A. Asynchronous bilateral ovarian torsion. J Pediatr Surg 2004; 39:746-9.
9. Takeda A, Hayashi S, Teranishi Y, et al. Chronic adnexal torsion: An under-recognized disease entity. Eur J Obstet Gynecol Reprod Biol 2017; 210:45.
10. Huchon C, Fauconnier A. Adnexal torsion: a literature review. Eur J Obstet Gynecol Reprod Biol 2010; 150:8-12.
11. Raicevic M, Saxena AK. Asynchronus bilateral ovarian torsions in girls-systematic review. World J Pediatr 2017; 13:416-20.
12. Oltmann SC, Fischer A, Barber R, et al. Cannot exclude torsion – a 15-year review. J Pediatr Surg. 2009;44:1212–6.
13. Smorgick N, Melcer Y, Sarig-Meth T, et al. High risk of recurrent torsion in premenarchal girls with torsion of the normal adnexa. Fertil Steril. 2016;105:1561–5.
14. Breech LL, Hillard PJ. Adnexal torsion in pediatric and adolescent girls. Curr Opin Obstet Gynecol. 2005;17:483–9.
15. Heling KS, Chaoui R, Kirchmair F, et al. Fetal ovarian cysts: prenatal diagnosis, management and postnatal outcome. Ultrasound Obstet Gynecol 2002; 20:47-50.
16. Ssi-Yan-Kai G, Rivain AL, Trichot C, et al. What every radiologist should know about adnexal torsion. Emerg Radiol. 2018;25(1):51–9.
17. Tsafrir Z, Hasson J, Levin I, et al. Adnexal torsion: cystectomy and ovarian fixation are equally important in preventing recurrence. Eur J Obstet Gynecol Reprod Biol 2012; 162:203-5.
18. Houry D, Abbott JT. Ovarian torsion: a fifteen-year review. Ann Emerg Med 2001; 38:156-9.
19. White M, Stella J. Ovarian torsion: 10-year perspective. Emerg Med Australas 2005;17:231-7.
20. Sasso RA. Intermittent partial adnexal torsion after electrosurgical tubal ligation. J Am Assoc Gynecol Laparosc 1996; 3:427-30.
21. Huchon C, Fauconnier A. Adnexal torsion: a literature review. Eur J Obstet GynecolReprod Biol. 2010;150(1):8–12.
22. White M, Stella J. Ovarian torsion: 10-year perspective. Emerg Med Australas. 2005;17(3):231–7.
23. Sasaki KJ, Miller CE. Adnexal torsion: review of the literature. J Minim InvasiveGynecol. 2014;21(2):196–202.
24. Gasser CRB, Gehri M, Joseph JM, Pauchard JY. Is it ovarian torsion? A systematic lit-erature review and evaluation of prediction signs. Pediatr Emerg Care. 2016;32(4):256–61.
25. P. Bolli, S. Schadelin, S. Holland-Cunz, P. Zimmermann.Ovarian torsion in children:development of a predictive score. Medicine 2017;96(43) :e8299.
26. M. B. Pepys, G. M. Hirschfield. C-reactive protein: a critical update. J ClinInvest 2003;111(12):1805-12.
27. Cohen SB, Wattiez A, Stockheim D, et al. The accuracy of serum interleukin-6 and tumour necrosis factor as markers for ovarian torsion. Hum Reprod 2001; 16:2195-7.
28. Daponte A, Pournaras S, Hadjichristodoulou C, et al. Novel serum inflammatory markers in patients with adnexal mass who had surgery for ovarian torsion. Fertil Steril 2006; 85:1469-72.

29. Taufiq DM, Bharwani NM, Sudderuddin SA, Rockall AG, Stewart VR. Adnexal torsion: review of radiologic appearances. Radiographics. 2021;41(2): 609–24.
30. Robertson JJ, Long B, Koyfman A. Myths in the evaluation and Management of Ovarian Torsion. J Emerg Med. 2017;52(4):449–56.
31. Bridwell RE, Koyfman A, Long B. High risk and low prevalence diseases: Ovarian torsion. Am J Emerg Med. 2022 ;56:145-150.
32. Moro F, Bolomini G, Sibal M, et al. Imaging in gynecological disease (20): clinical and ultrasound characteristics of adnexal torsion. Ultrasound Obstet Gynecol 2020; 56:934-43.
33. Adnexal Torsion in Adolescents: ACOG Committee Opinion No, 783. Obstet Gynecol 2019; 134:e56. Reaffirmed 2021.
34. Janković N, Janković M, Ristić Petrović A, Dimitrijević S. A rare disease which lead to avoidable ovary loss. Ovarian torsion-Case reports. Med. Pregl. 2025.(in press)
35. Pignataro JN, Schindler L. Isolated Fallopian Tube Torsion: Diagnosis and Management of a Gynecologic Emergency. Cureus. 2023;15(9):e46260.
36. Dhanda S, Quek ST, Ting MY, et al. CT features in surgically proven cases of ovarian torsion—a pictorial review. Br J Radiol. 2017; 90(1078):20170052
37. Schlaff W, Lund K, McAleese K, Hurst B. Diagnosing ovarian torsion with computed tomography. A case report. J Reprod Med. 1998;43(9):827–30.
38. Spinelli C, Piscioneri J, Strambi S. Adnexal torsion in adolescents: update and review of the literature. Curr Opin Obstet Gynecol. 2015;27(5):320–5.
39. Dasgupta R, Renaud E, Goldin AB, et al. Ovarian torsion in pediatric and adolescent patients: A systematic review. J Pediatr Surg. 2018;53(7):1387–91.
40. Cass DL. Ovarian torsion. Semin Pediatr Surg. 2005;14(2):86–92.
41. Cicchiello LA, Hamper UM, Scoutt LM. Ultrasound evaluation of gynecologic causes of pelvic pain. Obstet Gynecol Clin North Am. 2011;38(1):85–114.
42. Anders JF, Powell EC. Urgency of evaluation and outcome of acute ovarian torsion in pediatric patients. Arch Pediatr Adolesc Med. 2005;159(6):532–5.
43. Shalev J, Goldenberg M, Oelsner G, et al. Treatment of twisted ischemic adnexa by simple detorsion. N Engl J Med. 1989;321(8):546.
44. Oelsner G, Bider D, Goldenberg M, Admon D, Mashiach S. Long-term follow-up of the twisted ischemic adnexa managed by detorsion. Fertil Steril. 1993;60(6): 976–9.
45. Cohen SB, Oelsner G, Seidman DS, Admon D, Mashiach S, Goldenberg M. Laparoscopic detorsion allows sparing of the twisted ischemic adnexa. J Am Assoc Gynecol Laparosc. 1999;6(2):139–43.
46. Pansky M, Abargil A, Dreazen E, Golan A, Bukovsky I, Herman A. Conservative management of adnexal torsion in premenarchal girls. J Am Assoc Gynecol Laparosc. 2000;7(1):121–4
47. Lundorff P, Hahlin M, Kallfelt B, Thorburn J, Lindblom B.Adhesionformation after laparoscopy surgery in tubal pregnancy: a randomizedtrial versus laparotomy. Fertil Steril. 1991;55 (5):911-5.
48. McHutchinson LL, Koonings PP, Ballard CA, d'Ablaing G 3rd. Preservation of ovarian tissue in adnexal torsion with fluorescein. Am J Obstet Gynecol 1993; 168:1386-8.
49. Styer AK, Laufer MR. Ovarian bivalving after detorsion. Fertil Steril 2002; 77:1053-5.
50. Wang JH, Wu DH, Jin H, Wu YZ. Predominant etiology of adnexal torsion and ovarian outcome after detorsion in premenarchal girls. Eur J Pediatr Surg 2010; 20:298-301.