CASE REPORT

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Tubo-ovarian abscess in an adolescent with uterine didelphys and prior cloacal repair: case report and literature review

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ABSTRACT

Tubo-ovarian abscess (TOA) is an uncommon complication of pelvic infection, particularly in patients with Müllerian anomalies. We report the case of a 16-year-old girl with uterine didelphys and a history of cloacal repair who presented with fever, abdominal pain, and a large multiloculated left adnexal mass. Laparoscopy revealed a TOA within a severely distorted pelvis, requiring extensive adhesiolysis, excision of the abscess cavity with adherent tubal remnants, and partial cystectomy with ovarian preservation. Histopathology confirmed a purely inflammatory process, excluding malignancy. This case highlights a non-obstructive mechanism for TOA, in which distorted pelvic anatomy and postsurgical adhesions predisposed to infection, underscoring the need for individualised evaluation and fertility-preserving management in this population.

Keywords: Adolescent, cloacal malformation, Müllerian anomalies, Tubo-ovarian abscess, uterine didelphys

Introduction

Müllerian duct anomalies comprise a heterogeneous group of congenital malformations that result from defective formation, fusion, or resorption of the paramesonephric ducts.[1,2] They are relatively uncommon in the general population, with an estimated prevalence ranging between 6% and 7%, but their incidence is likely underestimated due to the high number of asymptomatic cases.[3] Among the various types of anomalies, uterine didelphys is one of the rarest forms and is characterised by the presence of two uterine cavities with independent endometrial linings, most often associated with a double cervix and sometimes a longitudinal vaginal septum.[4] The population prevalence of didelphys uterus is reported to be between 0.3% and 5%.[5]

The clinical presentation of uterine didelphys is variable. Many patients remain asymptomatic, while others may present with dysmenorrhea, pelvic pain, dyspareunia, or complications related to pregnancy and fertility.[4] Importantly, this anomaly frequently coexists with renal malformations, reflecting the close embryological relationship between the Müllerian and Wolffian duct systems.[6]

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This association between Müllerian and urinary tract anomalies is particularly evident in patients born with cloacal malformations, a rare group of congenital defects (estimated incidence 1:50,000 live births) in which the uro-rectal septum fails to divide the cloaca during weeks 6-7 of gestation, resulting in a single perineal channel shared by the urinary, genital, and gastrointestinal tracts.[7,8] Up to 60% of these patients have a duplicated Müllerian system, and renal anomalies are identified in up to 83% of cases.[8,9] Surgical reconstruction of cloacal malformations in early childhood is among the most complex procedures in paediatric surgery and typically requires multiple staged interventions, resulting in extensive pelvic adhesions and distorted anatomy that may carry long-lasting gynaecological consequences.[10,11]

As these patients reach adolescence, the occurrence of adnexal masses poses a significant diagnostic and therapeutic dilemma. The differential diagnosis ranges from functional ovarian cysts to infectious Tubo-ovarian abscesses (TOAs) or, less frequently, neoplastic lesions.[12] The presence of complex features such as septations, thickened walls, or papillary projections on imaging raises the suspicion of malignancy and necessitates surgical intervention.[13]

In most published cases, TOA is described in association with obstructive malformations, such as the obstructed hemivagina and ipsilateral renal anomaly (OHVIRA) syndrome, where stasis of menstrual blood predisposes to ascending infection.[14-16] However, patients with a history of cloacal repair represent a distinct population in which non-obstructive mechanisms, namely post-surgical adhesive disease and altered pelvic anatomy, may independently predispose to adnexal infections.[17] This pathophysiological pathway remains poorly characterised in the literature.

Here we describe the case of an adolescent with uterine didelphys and a history of cloacal repair who developed a large multiloculated adnexal mass ultimately diagnosed as a TOA. We also review the literature to explore the relationship between cloacal anomalies, uterine malformations and adnexal infections, highlighting the challenges in diagnosis and fertility-preserving management.

Methods

This case report was prepared in accordance with the CAse REport guidelines.[18] Written informed consent for publication, including clinical details, imaging, and surgical video, was obtained from the patient and her legal guardians.

For the literature review component, we adhered to the quality standards for narrative reviews as defined by the Scale for the Quality Assessment of Narrative Review Articles.[19] Relevant publications were identified through a comprehensive search of PubMed, Scopus, Embase and Google Scholar, complemented by cross-checking the reference lists of retrieved articles. We used combinations of the following search terms: "Tubo-ovarian abscess", "Müllerian anomaly", "uterine didelphys", "cloacal malformation", "cloacal repair", "pelvic abscess", and "adolescent". No language restriction was applied. All articles describing Tubo-ovarian or pelvic abscess in the context of Müllerian anomalies or cloacal malformations were considered eligible for inclusion.

Case Presentation

A 16-year-old female presented with fever and acute abdominal pain localized to the left iliac fossa.

Surgical and Medical Background

Her medical history was remarkable for a complex cloacal malformation repair with a 4.5 cm common channel, uterine didelphys, complete longitudinal vaginal septum, normal sacrum, and left vesicoureteral reflux. Neonatal management included loop sigmoidostomy and vesicostomy on the first day of life. On the seventh day of life, emergency laparotomy was performed for jejunal perforation, which was repaired by primary closure; appendectomy was performed concomitantly. At three months of age, surgical revision was required for colostomy prolapse. Definitive cloacal repair was undertaken at ten months of age at a tertiary colorectal referral centre and comprised posterior sagittal anorectovaginourethroplasty (PSARVUP) with laparotomy, extended total urogenital mobilisation, vaginal septum resection, vaginal flap vaginoplasty, and redo vesicostomy. Colostomy and vesicostomy closure were completed at fifteen months. The subsequent course was complicated by obstructive ileus at three years of age, requiring emergency laparotomy with resection of a large segment of small bowel. At five years, endoscopic subureteric injection was performed on the left side to address progressive renal damage secondary to vesicoureteral reflux. At seven years, a neo-appendicostomy with caecal flap (Malone procedure) was fashioned to allow antegrade colonic enemas for the management of faecal incontinence.

At the time of presentation, bowel continence was maintained through regular antegrade colonic enemas via the appendicostomy, and bladder emptying was achieved by clean intermittent self-catheterisation. Despite subureteric injection, left renal dysplasia with impaired function had developed as a consequence of chronic vesicoureteral reflux, with compensatory hypertrophy of the contralateral kidney. Menarche occurred at 12 years of age, and menstrual cycles had since been regular in frequency (28-30 days) and duration (4-5 days), with moderate flow and no dysmenorrhoea. There was no history of intermenstrual bleeding, cyclical pelvic pain, or symptoms suggestive of haemocoels. The patient did not use vaginal dilators; gynaecological follow-up had not identified vaginal stenosis requiring dilation. A structured safeguarding assessment was performed as part of the multidisciplinary evaluation: the patient confirmed that she had never been sexually active, and no safeguarding concerns were identified. Recurrent urinary tract infections with Escherichia coli had been documented over the preceding years.

Clinical Findings and Imaging

The patient presented to the emergency department with an approximately 4-day history of fever and progressively worsening pain in the left iliac fossa. On admission, she was febrile (38.9 °C), with localized tenderness in the left lower quadrant and a palpable adnexal mass. Laboratory tests demonstrated neutrophilic leucocytosis and markedly elevated C-reactive protein (CRP >30 mg/dL), while tumour markers (CEA, CA 19-9, CA 125) remained within normal limits. Urine cultures grew *Escherichia coli*, while vaginal swabs were sterile.

Vaginal examination revealed a patent vaginal introitus without stenosis and adequate vaginal calibre. A longitudinal vaginal septum was identified but was thin, non-obstructing, and allowed free passage to both cervical canals. Two distinct cervices were visualised, both appearing macroscopically normal, with no purulent cervical discharge.

Approximately one year before admission, a pelvic magnetic resonance imaging (MRI) documented a uterine didelphys with double cervices and a longitudinal vaginal septum. At that time, a large multiloculated cystic lesion was identified in the left adnexa (about 10 cm in size), helical in configuration, composed of multiple communicating cystic spaces and enclosing the left ovary. No mural nodules or papillary projections were detected. The differential diagnosis included a markedly distended hydrosalpinx vs. a large inclusion cyst.

Several months later, a contrast-enhanced abdominal computed tomography confirmed persistence of the left adnexal cystic lesion, described as a tubular, helical structure with mild homogeneous wall enhancement, displacing the ovary laterally. No ascites or lymphadenopathy were present.

At the time of hospitalization, a repeat pelvic MRI demonstrated significant interval progression of the adnexal lesion compared with prior imaging. The mass had developed diffuse wall thickening, perilesional inflammatory fat stranding, and a 1.4 cm papillary projection showing intermediate signal intensity on diffusion-weighted imaging (Figures 1, 2). The left ovary, though encapsulated by the cystic structure, remained identifiable with preserved follicular morphology. On coronal T2-weighted sequences (Figure 3), the spatial relationship between the mass and the pelvic organs was clearly depicted: the two separate uterine horns of the didelphic uterus were visible inferiorly, symmetric in size and signal intensity, with no evidence of hematometra or haemocoels. Based on these findings, the lesion was classified as Ovarian-Adnexal Reporting and Data

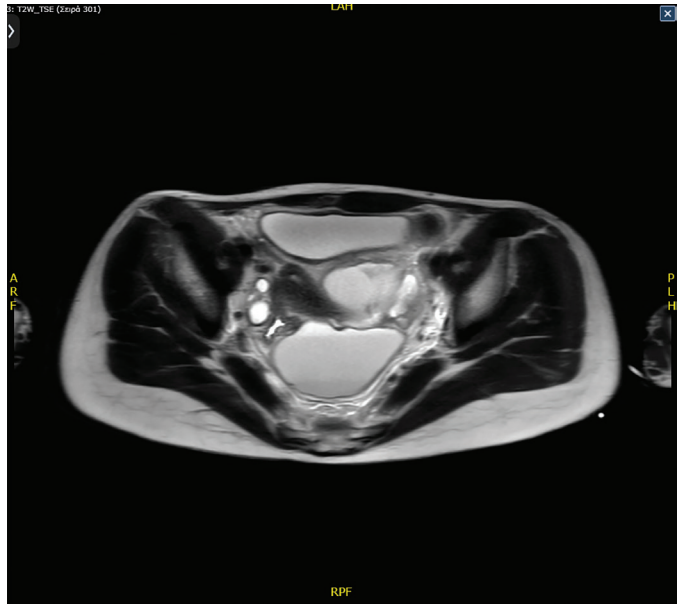

Figure 1. Axial T2-weighted pelvic magnetic resonance imaging obtained at the time of hospitalisation. The large multiloculated left adnexal mass is depicted with thickened walls and heterogeneous content consistent with Tubo-ovarian abscess. The bladder is visible anteriorly. The normal right ovary with preserved follicular morphology is identified in the right hemipelvis (contralateral side), providing comparison with the affected left adnexa.

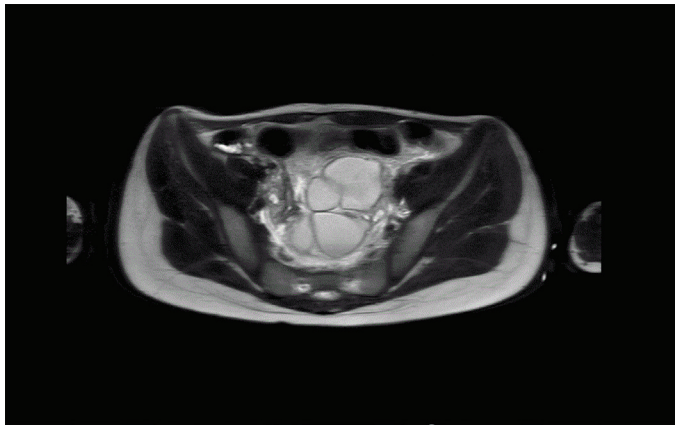

Figure 2. Axial pelvic magnetic resonance imaging (MRI) at the time of hospitalisation. The multiloculated mass demonstrates diffuse wall thickening and perilesional inflammatory changes consistent with Tubo-ovarian abscess formation. The multiple communicating cystic spaces of the lesion are clearly depicted.

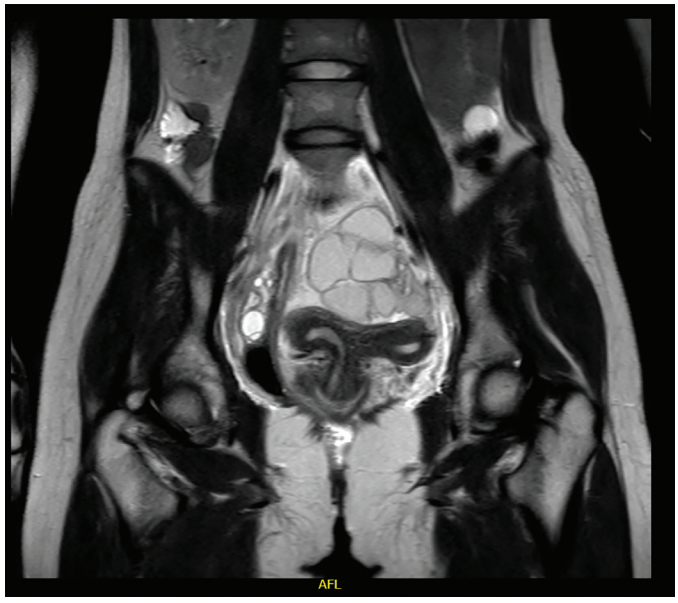

Figure 3. Coronal T2-weighted pelvic magnetic resonance imaging at the time of hospitalisation. The image demonstrates the large multiloculated cystic mass occupying the upper pelvis, with the two separate uterine horns of the didelphic uterus visible in the lower portion of the image. This view illustrates the spatial relationship between the adnexal lesion and the duplicated uterine anatomy and confirms the absence of hematometra in either uterine horn.

System 4 (O-RADS 4), raising concern for possible neoplastic transformation. No ascites or pathologic lymphadenopathy were detected.

Surgical Management

Empirical antimicrobial therapy was initiated with sequential intravenous regimens including cephalosporins (cefotaxime and ceftriaxone), ciprofloxacin, colistin, piperacillin-tazobactam, and oral doxycycline. Despite 10 days of treatment, the patient remained febrile, prompting surgical intervention. The decision to proceed to surgery rested on two convergent considerations: the failure of the presumed inflammatory process to respond to prolonged, culture-guided antimicrobial therapy, and the O-RADS 4 classification, which could not exclude malignancy and thus mandated surgical exploration with histological characterisation.

On the tenth day of hospitalisation, the patient underwent laparoscopic exploration under general anaesthesia (Supplementary Video). Given her history of cloacal repair and multiple abdominal surgeries, the procedure was technically challenging. Entry was achieved via Palmer's point, followed by placement of additional trocars.

Extensive adhesiolysis was necessary to access the pelvis. During exploration, a large Tubo-ovarian mass was identified, densely adherent to the sigmoid colon and pelvic sidewall. The ureters were carefully identified bilaterally to prevent injury. The sigmoid colon was mobilized to improve exposure of the adnexal region. The Tubo-ovarian complex was opened, releasing thick purulent material and inflammatory debris, which were thoroughly evacuated. Samples of intraperitoneal and cystic fluid were obtained for microbiological examination. Despite the macroscopic appearance, cultures were sterile.

Given the extent of inflammation and tissue destruction, complete anatomical identification of the left fallopian tube as a discrete structure was not achievable. The Tubo-ovarian complex was approached as a single inflammatory mass: the abscess cavity was opened, drained of purulent material and inflammatory debris, and its wall was progressively excised together with the adherent, necrotic tubal remnants that were inseparable from the abscess capsule. Partial cystectomy was then carried out using ultrasonic scissors, excising the majority of the remaining cyst wall while carefully preserving the left ovary and its vascular pedicle. Special care was taken to avoid injury to the ureter and bowel, given the distorted anatomy from previous surgeries. Haemostasis was secured with bipolar diathermy, followed by copious irrigation. A drain was placed in the pouch of Douglas.

The patient tolerated the procedure well, and the immediate postoperative course was uncomplicated.

Postoperative Course

The total hospital stays lasted 15 days. Following surgery and continuation of antimicrobial therapy, laboratory parameters showed progressive improvement. The initial neutrophilic leucocytosis and markedly elevated CRP decreased steadily, with normalisation of leukocyte counts and CRP levels approaching 5 mg/dL by the time of discharge. Haemoglobin levels transiently declined postoperatively to 8-9 g/dL, consistent with mild anaemia, but subsequently stabilised without the need for transfusion. At discharge, the patient was afebrile, asymptomatic, and in good clinical condition.

Histopathological examination of the excised specimen revealed fragments of a fibrous wall devoid of epithelial lining, containing inflammatory tissue with abundant neutrophilic polymorphonuclear leukocytes and foamy histiocytes, as well as haemorrhagic infiltration and dilated vessels. No evidence of malignancy was identified.

Discussion

This case illustrates that TOA can develop in adolescents with Müllerian anomalies through a non-obstructive pathway driven principally by post-surgical adhesive disease and altered pelvic anatomy rather than by menstrual outflow obstruction, thereby expanding the current understanding of the mechanisms linking congenital uterovaginal malformations to adnexal infection.

The literature on TOA in the context of Müllerian anomalies has been dominated by reports associated with the OHVIRA syndrome, also known as Herlyn-Werner-Wunderlich syndrome.[14-16,20-29] In this condition, the combination of an obstructed hemi-vagina and functional endometrium creates a closed cavity where menstrual blood accumulates, leading to haemocoels, retrograde menstruation, and a stagnant environment that facilitates ascending bacterial colonisation, with potential progression to pyocolpos, pyosalpinx, and ultimately TOA.[14,30,31] Retrospective paediatric and young adult series demonstrated that between 14% and 17% of OHVIRA patients presented with pelvic or intra-abdominal abscesses requiring surgical intervention.[27,32] A significant proportion of patients are diagnosed only years after menarche or even in adulthood, when complications such as TOA or pyometra become clinically evident.[15,33,34]

This obstructive model, however, does not apply to our patient and offers only a limited framework for understanding TOA that arises in the absence of menstrual outflow obstruction. Our patient shared some phenotypic features with OHVIRA, such as uterine didelphys, double cervix, and ipsilateral renal anomaly, yet the pathophysiology was entirely distinct. Menses were regular, and serial imaging consistently excluded hematometra, haemocoels, or any evidence of outflow obstruction. Rather, the development of TOA in this case must be understood in the context of her cloacal malformation and its surgical consequences.

Reconstruction of cloacal malformations is among the most complex undertakings in paediatric surgery, typically requiring PSARVUP, often with vaginal replacement, followed by staged procedures such as stoma reversal, bladder augmentation, and creation of catheterisable conduits.[7,11] In a review of 490 cases, Levitt and Peña[35] reported that 19% of patients required reoperation for complications including vaginal stricture or atresia, urethrovaginal fistula, and rectal prolapse. These multiple interventions inevitably result in extensive pelvic adhesive disease, which is a well-recognised cause of infertility, dyspareunia, bowel obstruction, and increased surgical complexity at subsequent operations.[36,37] In our patient, the dense adhesions between the Tubo-ovarian complex, sigmoid colon, and pelvic sidewall, encountered at laparoscopy, are entirely consistent with this pattern and likely contributed to the formation of a confined, poorly vascularised pelvic compartment, favouring abscess development.

Infectious complications, including pelvic abscess and pyometrocolpos, are in fact recognised sequelae of cloacal reconstruction. Fumino et al.[17] described a remarkably similar case of TOA developing after colonic vaginoplasty for a high cloacal anomaly in a 13-year-old sexually inactive girl and Sharma and Gupta[38] reported pelvic abscess among the complications of early vaginal replacement in cloacal malformations.

An anatomically sequestered compartment, however, still requires a source of infection, and here the urinary tract is the most likely candidate. Although our patient was not sexually active and no evidence of ascending genital tract infection was identified, she presented with a concurrent urinary tract infection caused by *Escherichia coli*. This finding is clinically relevant because patients with cloacal anomalies are particularly susceptible to recurrent urinary infections as a consequence of vesicoureteral reflux, incomplete bladder emptying, and the need for clean intermittent catheterisation,[8,9] all of which were

present in our patient. The direct anatomical proximity of the dysplastic left urinary tract to the confined adnexal space, together with the disruption of normal fascial planes from prior reconstructive surgery, likely facilitated local extension of urinary infection to the entrapped Tubo-ovarian complex.[12] It should be emphasised, however, that this contiguous urinary-to-adnexal route remains a plausible hypothesis rather than a proven mechanism. Urine cultures grew *Escherichia coli*, whereas the intraoperative peritoneal and cystic-fluid cultures were sterile, a finding most plausibly attributable to the prolonged multidrug antibiotic therapy administered preoperatively.[39] Direct microbiological continuity between the urinary and adnexal compartments could therefore not be established.

Beyond the obstructive paradigm, a distinct and growing body of literature documents TOA in non-sexually active adolescents in whom menstrual outflow is unobstructed.[40-43] In the largest series to date, Hakim et al.[40] described sixteen non-sexually active adolescents with TOA (mean age 14.6 years), the majority of whom had an underlying comorbidity, including genitourinary tract anomalies, obstructed hemi-vagina, ipsilateral renal agenesis, or recent appendicitis, with *Escherichia coli* the organism most frequently recovered. Comparable non-obstructive mechanisms have been reported in individual cases, including contiguous spread from appendiceal inflammation with bacterial translocation to the adnexa,[41] while a recent systematic review of pelvic inflammatory disease in sexually inactive paediatric patients identified urinary tract infection, congenital anomalies and prior appendicitis as the most frequent associated conditions.[42] Contemporary reviews of paediatric and adolescent TOA similarly emphasise that the aetiological spectrum in this population differs substantially from that of sexually active adults, with contiguous and haematogenous routes predominating over ascending sexually transmitted infection.[43] Our patient conforms to this non-obstructive, comorbidity-driven profile: a repaired cloacal malformation with distorted pelvic anatomy, recurrent *Escherichia coli* urinary tract infections, and no evidence of menstrual outflow obstruction.

The management of TOA in patients with prior cloacal reconstruction poses specific challenges that differ from both OHVIRA-associated cases and de novo TOA in the general population. In classical OHVIRA, the cornerstone of treatment is vaginal septectomy with marsupialisation, which restores drainage, relieves pain, and prevents re-accumulation and infection,[15,16,23,25,27,30] with hemi-hysterectomy or salpingectomy reserved for delayed or refractory disease at the cost of future fertility.[21-23,28,30,32] In patients with post-surgical adhesive disease, the goal was to eradicate the infection while preserving adnexal structures, a priority of paramount importance given the already compromised reproductive anatomy. Options include laparoscopic drainage or partial cystectomy of multiloculated adnexal collections with adnexal preservation, reserving organ-removing procedures for necrotic or unsalvageable tissue.

In our case, surgery was technically challenging, necessitating laparoscopic adhesiolysis, meticulous dissection to preserve ureter and bowel integrity, and partial cystectomy with ovarian preservation. This level of surgical complexity is well documented in the cloacal anomaly literature, where pelvic adhesions from prior reconstructive surgery significantly increase operative difficulty and the risk of iatrogenic injury to adjacent structures.[11] Fertility preservation was particularly important in light of the limited reproductive potential documented in this population. In a literature review, Vilanova-Sanchez et al.[36] reported that pregnancy is achievable in women with prior cloacal or complex anorectal malformation repair, with 24 pregnancies documented in 16 patients; the majority conceived spontaneously. However, these pregnancies carried significant risks: 5 ended in spontaneous miscarriage and 2 in ectopic pregnancy. Among the 19 live births, two thirds were delivered prematurely and nearly all required caesarean section.[36]

In conclusion, this case demonstrates that post-surgical adhesive disease, potentially compounded by recurrent urinary tract infections, represents a distinct and under-recognised risk factor for TOA in patients with repaired cloacal malformations. We advocate for proactive gynaecological surveillance in all adolescents with prior cloacal repair, with particular attention to both obstructive and non-obstructive complications as these patients reach reproductive age. Multidisciplinary, fertility-preserving approaches remain critical to optimising both short-term clinical outcomes and long-term reproductive potential in these complex patients.

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Ethical approval: This study is an observational case report based on data collected during routine clinical practice. This type of study does not require approval by an Ethics Committee.

Informed consent: Written informed consent for publication, including clinical details, imaging, and surgical video, was obtained from the patient and her legal guardians.

Data sharing: Data sharing is not applicable to this article as no datasets were generated or analysed during this study.

Transparency: The manuscript is an honest, accurate, and transparent account of the study being reported; that no important aspects of the study have been omitted; and that any discrepancies from the study as planned (and, if relevant, registered) have been explained.

Supplementary Video: https://youtu.be/xWtWUYJRrSQ

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