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CASE REPORTS / CASE SERIES

Isolated cor triatriatum sinistrum and pregnancy: case report and review of the literature

Cœur triatrial gauche isolé et grossesse: présentation de cas et revue de la littérature

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Abstract

Purpose *Isolated cor triatriatum sinistrum (CTS) is a heart malformation in which a perforated fibromuscular membrane divides the left atrium into two chambers. When communication between these chambers is restricted, a patient may have signs and symptoms of mitral stenosis. The later stages of pregnancy are associated with tachycardia and increases in intravascular volume. We describe how this altered physiology may affect pregnant women with asymptomatic CTS. We also review the literature relating to pregnancy in patients with CTS.*

Clinical features *A 30-yr-old primigravida, at 40 weeks of gestation with pre-pregnancy diagnosed asymptomatic CTS, was admitted for delivery. She had no cardiac symptoms during pregnancy, and her vaginal delivery under epidural analgesia was uneventful. This cardiac malformation is infrequently described in pregnant women, but a literature review showed that the physiology of late pregnancy with increases in hemodynamic variables may result in cardiac decompensation.*

Conclusion *While our patient with isolated CTS and an unrestrictive intra-atrial membrane had an asymptomatic pregnancy and an uneventful labour, the literature review suggests that the increase in intravascular volume and heart rate that occurs during late pregnancy and after delivery may result in cardiac decompensation, even in patients with asymptomatic CTS.*

Résumé

Objectif *Le cœur triatrial gauche (CTG) isolé est une malformation cardiaque dans laquelle une membrane fibromusculaire perforée divise l'oreillette gauche en deux chambres. Lorsque la communication entre ces chambres est restreinte, le patient pourrait présenter les signes et symptômes d'une sténose mitrale. En fin de grossesse, on peut observer de la tachycardie et des augmentations du volume intravasculaire. Nous décrivons la façon dont cette physiologie modifiée pourrait affecter les femmes enceintes atteintes de CTG asymptomatique. Nous passons également en revue la littérature portant sur la grossesse chez les patientes atteintes de CTG.*

Éléments cliniques *Une primigeste de 30 ans, à 40 semaines de grossesse et chez laquelle un diagnostic de CTG asymptomatique avait été posé avant la grossesse, a été admise à l'hôpital pour l'accouchement. Pendant la grossesse, elle n'a présenté aucun symptôme cardiaque, et*

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son accouchement vaginal sous analgésie péridurale s'est déroulé sans complication. Cette malformation cardiaque n'est pas souvent décrite chez les femmes enceintes, mais une revue de la littérature a démontré que la physiologie de fin de grossesse, avec son hyperdynamie cardiovasculaire, peut entraîner une décompensation cardiaque.

Conclusion *Bien que notre patiente atteinte d'un CTG isolé et présentant une membrane intra-atriale non restrictive ait vécu une grossesse asymptomatique et un travail obstétrical sans complication, une revue de la littérature suggère que l'augmentation du volume intravasculaire et de la fréquence cardiaque survenant en fin de grossesse et après l'accouchement pourrait entraîner une décompensation cardiaque et ce, même chez les patientes atteintes d'un CTG asymptomatique.*

Cor triatriatum is a rare heart malformation that accounts for 0.1% of congenital heart defects.[1-4] It may be associated with other cardiac anomalies,[5,6] but only 30% of cases present as an isolated malformation. Several classifications have been proposed.[7-10] Isolated cor triatriatum sinistrum (CTS) is characterized by a septum-like fibromuscular membrane that divides the left atrium into proximal and distal chambers.[11] The pulmonary veins most frequently flow into the proximal chamber, and blood flows from the proximal to the distal chamber through a single opening or multiple openings in the membrane (Fig. 1).[12,13] Depending on the size of this communication, flow may be either unimpeded and clinically asymptomatic or restrictive and symptomatic.

The diagnosis and surgical correction of more complex variants of CTS usually occur in childhood because the morbidity and mortality in untreated patients are very high.[4] When diagnosed in adults, CTS is typically a variant with relatively unrestricted communication in the intra-atrial membrane and is an incidental finding.[14,15] Nevertheless, because CTS represents an obstructive lesion that limits the ability of the heart to respond to increased demand, even the asymptomatic form may become symptomatic in states of increased cardiovascular demand.[1,16,17] For example, late pregnancy is associated with increased intravascular volume and heart rate, and this physiologic change can convert the clinical presentation of CTS from asymptomatic to symptomatic.[1,7,17-19]

We describe a woman with an asymptomatic isolated type of CTS who had an uncomplicated vaginal delivery with epidural analgesia. We also review the literature relating to pregnancy in patients with cor triatriatum.

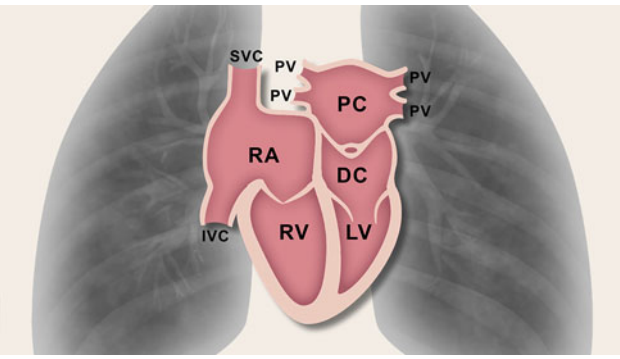

Fig. 1 Illustration of isolated form of cor triatriatum sinistrum. Left atrium is divided with interatrial membrane into proximal chamber (PC) and distal chamber (DC), and the blood flow occurs via a single opening in the membrane (shown also in the Video available as Electronic Supplementary Material). IVC = inferior vena cava; LV = left ventricle; PV = pulmonary vein; RA = right atrium; RV = right ventricle; SVC = superior vena cava

Case description

The need for permission to publish this single case report was waived by the Institutional Review Board at University Hospital Merkur, Zagreb, Croatia, and the patient provided written informed consent for the details of her case to be published.

A 30-yr-old primigravida was admitted at 40 weeks of gestation with regular contractions and intact membranes. At the age of 24 yr, she was diagnosed with isolated asymptomatic CTS during a respiratory tract infection when auscultation revealed the presence of a grade 3/6 left parasternal systolic heart murmur. At that time, she reported an excellent state of health and was fully employed in a physically demanding occupation. She was referred to a cardiologist for evaluation, and echocardiography showed that the right atrium and ventricle and the left ventricle were normal; however, the left atrium was divided into two chambers by an accessory membrane with a single 1.4-cm^2 opening (Fig. 2). This membrane separated the left atrium into a slightly hypertrophic proximal chamber that received the entire blood inflow through mildly dilated pulmonary veins and a distal chamber that contained the atrial appendage and the mitral valve. There were no other cardiac anomalies. Blood flow through the accessory membrane was turbulent (flow, 1.7 $min \cdot sec^{-1}$; mean diastolic gradient, 6.5 mmHg). Systolic and diastolic pulmonary pressure was 35/15 mmHg (measured via right heart catheterization). No surgical therapy was offered at that time.

During pregnancy, at the age of 30 yr, she had no cardiac symptoms and no follow-up evaluations by a cardiologist for her CTS. Toward the end of her pregnancy, at the 37th week of gestation, her cardiologist performed echocardiography and confirmed the presence of CTS with

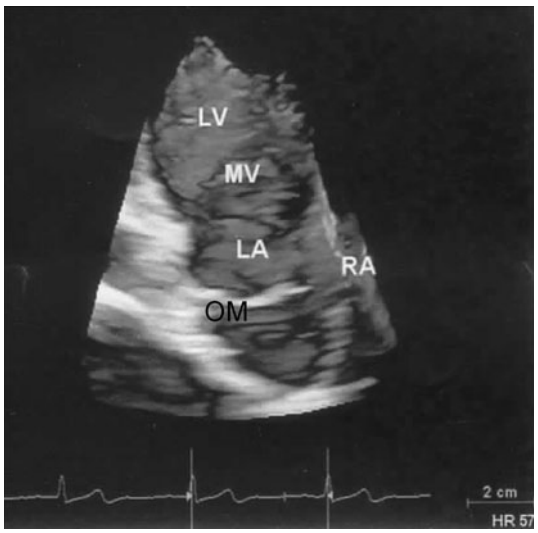

Fig. 2 Parasternal long-axis view of the heart. LA = left atrium; LV = left ventricle; MV = mitral valve; OM = obstructive membrane; RA = right atrium

a mean diastolic pressure gradient of 13.0 mmHg. Electrocardiography showed sinus rhythm at 56 beats·min^{-1}. There were no formal preoperative consultations with an anesthesiologist. In the delivery room, a lumbar epidural catheter was inserted, and analgesia was accomplished with a 6-mL bolus of 0.25% bupivacaine followed by continuous infusion of 0.125% bupivacaine at 8 mL·hr^{-1}. Contractions were augmented by an oxytocin infusion. Five hours later, a healthy baby was delivered. The patient's epidural analgesia was not associated with hypotension, and she received 500 mL of lactated Ringer solution during the delivery course with maximal heart rate of 120 beats· min^{-1}. From a cardiac standpoint, she remained asymptomatic during the three-day hospitalization. She remained asymptomatic one year following the delivery, and echocardiography found a mean diastolic pressure gradient of 6.4 mmHg.

Discussion

We describe an uneventful pregnancy and vaginal delivery with epidural analgesia in a patient with an asymptomatic form of isolated CTS. The clinical course in our patient differed from reports that provide evidence that pregnancy may trigger the initial onset of cardiac symptoms in women with pre-pregnancy asymptomatic CTS (Table).[1,7,17-19]

To evaluate the experience with cor triatriatum during pregnancy, we performed a MEDLINE database search from January 1, 1946, to July 1, 2012 with the following MeSH terms: *cor triatriatum*, *pregnancy*, *pregnancy complications*, *pregnancy outcome*, *pregnancy high-risk*, *labor*, *obstetric labor*, *labor onset*, *labor stage first*, *labor stage second*, *labor stage third*, and *delivery obstetric*. The following keywords were used: *cor triatriatum*, *triatriatum*, *polyatria$*, *triatria$*, *subdivided left atria/atrium*, *pregnan$*, *gravid$*, *labor*, and *delivery*. (The dollar sign, $, is the truncation symbol to capture inflective forms of terms, e.g., *pregnant*, *pregnancy*, *pregnancies*.) The same search was run in the EMBASE database covering January 1, 1988, to July 1, 2012. Searches were not limited by language or publication type (articles or letters).

In response to increased metabolic demand, physiologic cardiovascular changes occur in pregnancy (increases in heart rate, 15%-25%; intravascular volume, 45%; and therefore also cardiac output, 40%-50%) to reach maximum levels by the 32nd week of gestation.[20] This increased demand peaks during delivery when the heart rate may exceed 160 beats·min^{-1} during the pushing phase, regardless of the use of epidural analgesia.[20] Furthermore, immediately after delivery in a healthy woman, uterine autotransfusion acutely and additionally increases intravascular volume, resulting in up to an 80% increase in cardiac output.[16,20-22] These changes in hemodynamic variables may pose a substantial risk for parturients with CTS and especially those with restrictive flow across the intra-atrial membrane. Therefore, CTS patients who were symptomatic before pregnancy,[1,23] or even asymptomatic in previous pregnancies,[12] may be at risk for cardiac decompensation during and after delivery.

The pathophysiology of decompensation of CTS is similar to that of mitral stenosis.[24] The principal difference is that the obstructive site is at the intra-atrial accessory membrane instead of at the mitral valve. In the case of mitral stenosis, the patient may become symptomatic when the area is reduced to less than 2 cm^2 and becomes critical at less than 1 cm^2. Increases in heart rate and intravascular volume in a patient with CTS, as with mitral stenosis, worsen the cardiopulmonary signs and symptoms. As the heart rate accelerates, the diastolic filling time is insufficient to fill the left ventricle, and pressure builds up in the proximal left atrial chamber, leading to pulmonary hypertension, pulmonary edema, and right ventricular failure. A chronic pressure increase in the atrial chamber may lead to dilation and atrial fibrillation, which further impede blood flow across the accessory membrane because of the loss of atrial kick.[25] The propensity for cardiac decompensation can be even more pronounced in a preeclamptic parturient due to increased pulmonary capillary permeability;[2,19,26,27] therefore, the signs and symptoms of preeclampsia must be recognized early, and aggressive treatment and surveillance should be undertaken in these patients. It is noteworthy that our patient remained asymptomatic despite pregnancy-induced changes in cardiovascular dynamics, as evidenced by the increase in the diastolic pressure gradient from

Table Summary of reports of pregnancy associated with cor triatriatum since 1946 ($n = 14$)*

Source and Year	Pre-pregnancy/Pregnancy Cardiac History	Signs/Symptoms/Evaluation	Delivery and Anesthesia	Outcome
Thorin *et al.*,[2] 1995	22-yr-old, first pregnancy; no cardiac symptoms before or during pregnancy; hypertension and proteinuria at 38 weeks; at 40 weeks hypertension and progressive exertional dyspnea	Immediately postpartum, sudden onset of shortness of breath (pulmonary edema preceded by generous hydration); ECG: right atrial hypertrophy; ECHO: CTS type A (1 or 2) (small secundum type ASD), mean gradient across OM, 4.5 mmHg	Spontaneous rupture of membrane at 41 weeks; urgent Cesarean delivery for pathologic cardiotocogram (epidural anesthesia)	3 months postpartum, uneventful repair (resection of intra-atrial membrane, closure of ASD, and mitral annuloplasty)
LeClair *et al.*,[16] 1996	25-yr-old, first pregnancy; no symptoms before or during pregnancy	Immediately postpartum, shortness of breath (SpO_2, 70%), tachycardia, tachypnea, crackles both lungs; chest radiogram consistent with pulmonary edema which resolved with therapy; discharged without diagnosis	Cesarean delivery for cephalopelvic disproportion (spinal anesthesia)	Discharged on postoperative day 5; subsequent ECHO: CTS with intra-atrial membrane opening, 1.5 cm^2, mean gradient, 12 mmHg, and PHTN; surgical repair of small ASD (subtype A1 or A2?), uneventful postoperative course
Sajeev *et al.*,[12] 2003	33-yr-old, fourth pregnancy; no complications or symptoms of heart disease during previous 3 pregnancies	Asymptomatic during fourth pregnancy but heart murmur triggered evaluation; ECHO: CTS type A, mean gradient, 8 mmHg	Not reported	Rest of pregnancy uneventful; uncomplicated delivery, no complications long term after delivery
Davlouros *et al.*,[18] 2011	35-yr-old, second pregnancy; no cardiac symptoms before or during pregnancy	Immediately postpartum, acute pulmonary edema; CTS type A, mean gradient across OM, 7.9 mmHg	Not reported	Patient declined surgery and was discharged on medical therapy
Bai *et al.*,[23] 2010	20-yr-old, first pregnancy; asymptomatic before pregnancy	At 20 weeks exercise intolerance, orthopnea, paroxysmal nocturnal dyspnea; ECHO: CTS type A, mean gradient across OM, 17 mmHg, PHTN, 65 mmHg	Vaginal delivery at term uneventful (epidural analgesia)	At 21 weeks, urgent corrective surgery with cardiopulmonary bypass; discharged home postoperative day 4, uneventful continued pregnancy
Sentilhes *et al.*,[1] 2004	31-yr-old, first pregnancy; asymptomatic before pregnancy	After 3 months of pregnancy progressive dyspnea, rapid AF, auscultation grade 2/4 holosystolic murmur; ECHO: CTS type A	At 37 weeks, spontaneous vaginal delivery (epidural analgesia)	Therapy with digitalis, glycosides, β-blocker, LMWH (all stopped 5 days before delivery); rest of pregnancy uneventful; uncomplicated delivery
Tasca *et al.*,[28] 2007	34-yr-old, first pregnancy; at age 18 years palpitations and unconfirmed "congenital heart defect"	Asymptomatic before and after pregnancy; referred to cardiology during pregnancy because of history of "congenital heart defect"; ECHO: mildly obstructive CTS type A, mean gradient, 2.7 mmHg	Cesarean delivery at 36 weeks per obstetrician's decision (type of anesthesia unknown, indication unknown)	Rest of pregnancy uneventful; uncomplicated delivery; stable condition 2 yr after delivery
Gavand *et al.*,[25] 2011	41-yr-old, first pregnancy; diagnosed with cor triatriatum at age 17 years with AF; asymptomatic with physical activity; treatment (amiodarone) stopped	At 23 weeks PHTN (57 mmHg), nocturnal orthopnea, AF, severe anemia (6.4 $g \cdot dL^{-1}$), NYHA class III; ECHO: CTS type A, maximal gradient across OM, 30 mmHg	Cesarean delivery at 32 weeks due to maternal symptoms (general anesthesia)	Discharged 10 days after uneventful delivery; 5 months later, corrective cardiac surgery; postoperative period uneventful; ECHO evaluation after 2 months: stable condition

Table continued

Source and Year	Pre-pregnancy/Pregnancy Cardiac History	Signs/Symptoms/Evaluation	Delivery and Anesthesia	Outcome
Higton *et al.*,[17] 2009	32-yr-old, third pregnancy; as a child diagnosed with "a hole in the heart," but no workup; in previous pregnancies breathlessness from mid-trimester into postpartum period attributed to physiology of pregnancy	At 20 weeks breathlessness; systolic murmur, loud II heart sound; cardiac MRI and ECHO: CTS type A, PHTN	Not reported	Not reported
Kanbara *et al.*,[29] 2005	26-yr-old, first pregnancy	At 21 weeks, symptoms of acute CHF; ECHO: CTS type A with aberrant inflow or left pulmonary vein below accessory membrane	Cesarean delivery (spinal anesthesia) at 38 weeks due to worsening cardiac symptoms	Uneventful delivery
Kaneko *et al.*,[30] 1993	30-yr-old, second pregnancy; cardiac symptoms (palpitations, chest pain, shortness of breath) during the first and second pregnancies; no diagnosis established in first pregnancy	Cardiac symptoms recurred during second pregnancy; ECHO: CTS type A	Vaginal delivery at 39 weeks	Uneventful delivery; 1 month after second delivery, episode of palpitations, shortness of breath, and chest pain, acute coronary syndrome
Mathew *et al.*,[19] 2004	28-yr-old, fifth pregnancy; uneventful	At 25 weeks' gestation, fifth pregnancy, severe preeclampsia, diagnosed with CTS subtype A2 (ostium secundum ASD)	Cesarean delivery (general anesthesia), emergency due to severe preeclampsia	Uneventful postdelivery course; discharged day 5 after delivery
Kokotsakis *et al.*,[31] 2011	32-yr-old, third pregnancy; uneventful	Immediately postpartum (third pregnancy), acute pulmonary edema; ECHO: PHTN, CTS type A, with redundant intra-atrial membrane prolapsing toward mitral valve and causing severe obstruction	Not specified	At age 33 yr, admitted to cardiology with heart failure, corrective surgery, excellent long-term outcome (NYHA class I)
Present case report, 2012	30-yr-old, first pregnancy; asymptomatic before and during pregnancy	Asymptomatic; at age 24 yr referred to cardiologist to evaluate loud systolic heart murmur; ECHO: CTS type A	Uneventful vaginal delivery (epidural analgesia)	Asymptomatic from cardiac standpoint during pregnancy, delivery, and postpartum; remained asymptomatic 2 yr after pregnancy

AF = atrial fibrillation; ASD = atrial septal defect; CHF = congestive heart failure; CTS = cor triatriatum sinistrum; ECG = electrocardiogram; ECHO = echocardiography; LMWH = low-molecular-weight heparin; MRI = magnetic resonance imaging; NYHA = New York Heart Association; OM = obstructive membrane; PHTN = pulmonary hypertension; SpO_2 = oxygen saturation as measured by pulse oximetry

* Lam's classification of cor triatriatum: type A (classic or isolated variant): proximal chamber receives all vein inflow, no ASD; subtype A1: ASD between proximal chamber and right atrium; subtype A2: ASD between distal chamber and right atrium; type B: pulmonary vein inflow into coronary sinus, which forms a variant of total anomalous pulmonary venous connection; type C: no connection between pulmonary veins and the proximal chamber

6.5 mmHg before pregnancy to 13.0 mmHg during late pregnancy, reflecting increased intravascular volume load.

Our literature search identified ten parturients with cor triatriatum,[1,2,12,16-19,23,25,28] and review of the bibliography of these articles revealed three additional cases (Table).[29-31] Six of these 13 women were first diagnosed with CTS during pregnancy;[1,12,19,23,29,30] four of them presented during the early postpartum period;[2,16,18,31] one was diagnosed with CTS at the age of 17 yr (presenting sign was transient atrial fibrillation), but she was asymptomatic until severe symptoms developed during her first pregnancy at the age of 41 yr;[25] one was diagnosed with a "nonspecified cardiac defect" at the age of 18 yr, was asymptomatic throughout life, but underwent further cardiac evaluation during an uneventful pregnancy;[28] and one was diagnosed in childhood with a "hole in the heart" but had no further workup until she became symptomatic during her first pregnancy.[17] Altogether, three asymptomatic women had cor triatriatum diagnosed through evaluation of a heart murmur, and 11 were asymptomatic

before pregnancy and became symptomatic during pregnancy when the diagnosis was established. As with our patient, isolated CTS malformation was reported in 11 of the published cases, but two had CTS associated with small atrial septal defects. No report of a pregnant woman with cor triatriatum associated with other major cardiac defects was identified in our literature search. This may be attributed to the fact that CTS associated with other cardiac anomalies is symptomatic early in life, involves high morbidity and mortality, and the defects are surgically repaired in childhood.[4]

While pregnancy-induced changes in the cardiovascular workload are well tolerated by a healthy parturient, this may not be the case when an obstructive lesion, such as CTS, is present.[16] An animation showing cardiac decompensation associated with pregnancy, labour, and the postpartum period in an isolated CTS heart is shown in the Video (available as Electronic Supplementary Material). Cor triatriatum sinistrum increases the likelihood to become symptomatic after 20 weeks of gestation and more likely during delivery and in the postpartum period;[22,32] therefore, early identification of all asymptomatic patients with CTS (i.e., evaluate all "innocent" heart murmurs) is extremely important. It remains a matter of judgment (on the part of a cardiologist) whether CTS has the potential to decompensate in pregnancy. Consideration should be given to pre-pregnancy correction of CTS, except in women whose membrane perforations are very large and clearly do not restrict communication. If CTS is diagnosed during pregnancy, the woman should be regarded as high risk for the remainder of her pregnancy, and she should be observed closely for onset of symptoms that may indicate cardiac decompensation (shortness of breath and/or palpitations, irregular pulse). A high level of vigilance is needed because symptoms, such as easily fatiguing and palpitations, may be a part of normal pregnancy and, at the same time, may be early signs of cardiac decompensation in CTS.[17,30] Furthermore, it is possible to be asymptomatic in an uneventful pregnancy but symptomatic in subsequent pregnancies associated with preeclampsia.[17,26,27]

Medical treatment is directed toward lowering the heart rate (β-adrenergic blockers) and blood volume (diuretics). If atrial fibrillation develops, anticoagulant therapy is needed to reduce the risk of embolic events.[33] The diagnosis of acute heart failure and severe pulmonary hypertension during pregnancy may mandate emergent operation.[23] An open surgical approach using cardiopulmonary bypass is preferred over rarely performed percutaneous interventions for treatment of this type of cardiac anomaly.[34] The role of percutaneous balloon dilation in managing this condition remains to be determined.[4,35]

If emergency surgery is indicated during pregnancy, the anesthesiologist must follow the same hemodynamic principles used for intraoperative management of mitral stenosis (avoid tachycardia, maintain euvolemia, avoid hypotension [lateral tilt], and avoid an increase in pulmonary vascular resistance from hypoxia, hypercapnia, or certain drugs).[36] If regional anesthesia is contemplated for a vaginal delivery, as it was in our parturient and in other reported patients,[1,23] the use of diluted local anesthetics or opioids can minimize adverse hemodynamic effects. Nevertheless, in the majority of the reported cases, obstetricians chose Cesarean delivery,[2,16,19,25,28,29] which was performed successfully under epidural[2,25] or spinal anesthesia (Table).[16,29]

In summary, asymptomatic isolated CTS can acutely decompensate during pregnancy and the early postpartum period. Surgical repair of asymptomatic CTS with an echocardiographically proven restrictive opening should be considered before pregnancy. Although successful medical management of decompensated cor triatriatum during pregnancy is possible, if severe cardiac decompensation ensues, urgent surgical intervention may be required. Since these patients are at high risk of developing acute heart failure, their care should be managed in large medical centres with immediately available multispecialty expertise.

Conflict of interest None declared.

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