Fluid-structure interaction simulation of ureter with vesicoureteral reflux and primary obstructed megaureter

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Abstract. Two common abnormalities in ureters include primary refluxing megaureter (PRM) and primary obstructed megaureter (POM). The aim of this study was to represent the numerical simulation of the urine flow at the end of the ureter with vesicoureteral reflux (VUR) and POM during peristalsis. Methodologically, the peristalsis in the ureter wall was created using Gaussian distribution. Fluid-structure interaction (FSI) was applied to simulate urine-elastic wall interactions; and governing equations were solved using the arbitrary Lagrangian-Eulerian method. Theories such as wall elasticity, Newtonian fluid, and incompressible Navier-Stokes equations were used. Velocity fields, viscous stresses and volumetric outflow rate profiles were obtained through the simulation of the ureter with VUR and POM during peristalsis. In addition, the effect of urine viscosity on flow rate was investigated. When the bladder pressure increased, VUR occurred because of the ureterovesical junction (UVJ) dysfunction, leading to high stresses on the wall. In the POM, the outflow rate was ultimately zero, and stresses on the wall were severe in the obstructed section. Comparing the results demonstrated that the peristalsis leads to even further dilation of the prestenosis portion. It was also observed that the reflux occurs in the ureter with VUR when the bladder pressure is high. Additionally, the urine velocity during the peristalsis was higher than the non-peristaltic ureter.

Keywords: Primary obstructed megaureter, ureterovesical reflux, Arbitrary Lagrangian-Eulerian, fluid-structure interaction, Navier-Stokes equations, moving mesh

1. Introduction

Ureteral peristalsis can be considered as a series of waves of active muscular contraction that move at near-constant speed along the ureter which facilitate the urine flow \cite{1}. Peristalsis of the ureter has always been a significant issue in peristaltic transfer \cite{2,3}. Understanding the ureter peristalsis and its abnormalities may help in designing flow-aided devices such as valves and stents, which are key factors in eliminating these malfunctions. In addition to the peristalsis, the urine flow in the ureter initiates according to the pressure gradient between the renal pelvis and bladder \cite{4}. Urine discharges to the bladder through

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a one-way valve at the end of the ureter, called UVJ. Under normal circumstances, UVJ allows urine to enter the bladder while preventing urine reflux in high bladder pressure. When this valve is incompetent, the chance for urine reflux to the renal pelvis increases [5].

Megoureter is considered as an abnormally dilated ureter with any diameter larger than 8 mm [6]. According to the classification of Smith et al. [7], megoureters can be classified as obstructed, refluxing or unobstructed and not refluxing. The pathology of the megoureter can also be categorized as primary (intrinsic) or secondary (due to another urinary tract abnormality) [6]. Shokeir and Nijman [8] reviewed the most recent advances in the diagnosis and management of the megoureter. They indicated the POM as an abnormal ureter caused by a prevesical non-peristaltic ureter segment, which generates a dilatation at the prestenosis section. The prevesical stenosis segment cannot undergo the peristalsis because of a significant decrease in smooth muscle of ureter wall [5,9]. At the PRM, vesicoureteral reflux resulting from UVJ dysfunction causes dilation of the ureter [8]. The VUR, which is a prevalent disease in the field of pediatric urology, has been classified to 5 grades regarding the reflux intensity. For instance, grade 1 denotes reflux into the non-dilated ureter and grade 2 shows reflux into renal pelvis and calyces without dilation.

Studies in biology and medicine on the species of mammals have concentrated on the rate of ureteral peristaltic propagation [10], morphology of the normal ureter [11,12], ureteral lumen during peristalsis [13], and ureter muscle tissue reaction to drug stimulation [14]. Furthermore, Jørgensen [15] studied pathogenetic factors in VUR and showed that pressure load transmitted from bladder to the kidney without UVJ hindrance is mandatory to form VUR. Moreover, classification of megoureters [6–8], their diagnosis [8,16], and temporary treatment or management methods of them [8,17,18] have been investigated based on medical perspective. For instance, Lee et al. [17] introduced refluxing ureteral reimplantation as a safe surgery technique for temporizing with the obstructed megoureter.

Studies in the field of biomechanics have concentrated on the mechanical properties of the ureter [19–21], the mathematical analysis of peristalsis in the urinary tract [22,23], and other organs [24,25]. Griffiths [26] studied the dynamics of the upper urinary tract and analyzed the impact of bladder pressure and peristalsis on pressure/flow relations. In addition, Vahidi and Fatouraee [27] simulated urine flow in the ureter assuming the simple geometry and boundary conditions. Lozano [28] modeled the peristaltic flow experimentally using dimensional analysis to investigate different subjects such as flow rate magnitude and solid particle transport during the peristalsis.

Adopting methods involving the direct solution of Navier-Stokes equations have recently gained popularity since they do not set any restrictions on problems of moving boundaries and complicated geometries, particularly those confronted with biology flows. For instance, Peskin [29] proposed the immersed boundary method to model blood flow in heart valves. In this method, which is a direct numerical methodology for handling the moving boundaries, moving boundary is presented with a singular source term in Navier-Stokes equations. Furthermore, Teran et al. [30] investigated the peristaltic pumping and irreversibility of a Stokes in a viscoelastic fluid by immersed boundary method. An alternative procedure for simulating the ureteral peristalsis is applying the FEM to solve the governing equations using Arbitrary Lagrangian-Eulerian (ALE) formulation [31]. In the ALE technique, the mesh moves arbitrarily to optimize the shapes of elements, while the mesh on the boundaries and interfaces of the domains can move along with materials to track the boundaries and interfaces of a multi-material system, precisely. Thus, the ALE approach is appropriate for solving the fluid-structure problems, where fluid mesh undergoes large deformations near the structure. Vahidi et al. [32] presented a two-dimensional model to simulate the urine flow and peristalsis in a healthy ureter using FSI method. The results represented that the shear stresses were high near the contraction wave. In another study, Vahidi and Fatouraee [33] utilized the FSI method and ALE formulation to analyze the urine bolus peristaltic
transportation. They studied urine pressure distribution, shear stresses on the ureteral wall, and the effect of pressure gradient between the kidney and the bladder. They also showed that improper function of ureteropelvic junction leads to urine backflow during start-up of the peristalsis.

Despite all these studies, no mechanical model of POM or a ureter with VUR has been provided yet. In this paper, two models are proposed for the ureter terminal: 1) refluxing ureter in the case of UVJ dysfunction; 2) POM. Assuming the actual characteristics of the experimental data and elastic wall, peristaltic propagation and its effects on the urine flow are investigated with axisymmetric models. Urine-elastic wall interaction is properly modeled using the FSI. The main aim is to model and analyze abnormal ureters whose diagnosis and treatment are important nowadays.

2. Methodology

2.1. Geometry of the models

Two models including ureter with VUR and POM were considered with limit lengths of 30 mm and 40 mm, respectively. The ureteral diameter including the ureter wall ranges from 4 mm to 10 mm [32]. As shown in Fig. 1, the normal diameter of ureter is assumed 6 mm and the thickness of the muscular coat is taken 1 mm. Ureters with any diameter larger than 8 mm are considered as a megaureter [6]. In this study, it is assumed 10 mm in the dilated segment and 0.5 mm in obstructed section. In addition, the length of the prevesical obstruction is assumed 20 mm as can be seen in Fig. 2.

2.2. Fluid model

Fluid flow in both ureter with VUR and POM are simulated axisymmetrically with limit length. Density and viscosity of urine were assumed to be 1050 Kg/m³ and 1.3 mPa.s, respectively [32]. The Navier-Stokes
equations for a Newtonian incompressible fluid are as follows:

\[ \nabla \cdot \vec{V} = 0 \]  

(1)

\[ \rho \dot{\vec{V}} + (\vec{V} \cdot \nabla) \vec{V} = \nabla \cdot \left[-pI + \tau\right] + F \]  

(2)

where \( \rho \), \( \vec{V} \), and \( \rho \) are fluid density, velocity vector, and pressure vector, respectively. Also, \( F \) indicates any external forces applied to the fluid. The \( \tau \) represents deviatoric stress tensor that is usually expressed as

\[ \tau = \mu \dot{\gamma} \]  

(3)

where \( \mu \) denotes the fluid viscosity and \( \dot{\gamma} \) is the strain rate vector. For the incompressible viscous fluids, the strain rate tensor is defined as

\[ \dot{\gamma} = 2\varepsilon = \nabla \vec{V} + \nabla \vec{V}^T \]  

(4)

where \( \varepsilon \) indicates the rate of deformation tensor.

2.3. Elastic wall model

The ureter wall is assumed homogenous and linear elastic material with a constant density of 1000 Kg/m\(^3\). Young’s modulus of ureter wall is taken 5 KPa using the stress/stretch relations reported by Fung [21] in his experimental study on the ureteral tissue. The mathematical model of the ureter wall was solved using Lagrangian formulation, which is expressed as:

\[ \rho_s \frac{d^2 \vec{V}_s}{dt^2} - \nabla \cdot \sigma_s = F_v \]  

(5)
where $\rho^s$, $\vec{V}^s$ and $\sigma^s$ are solid density, solid displacement component, and stress tensor, respectively. $F_v$ indicates any volumetric force applied to the elastic material.

### 2.4. Fluid-structure model

For solving FSI problems, kinematic and dynamic coupling conditions should be considered on the common boundary of fluid and solid. The kinematic coupling condition that means no slip on this boundary is expressed as follows:

$$\vec{V}_f = \vec{V}_s$$

where $\vec{V}_f$ and $\vec{V}_s$ represent fluid velocity and solid velocity, respectively. This equation indicates equilibrium of the velocity and displacement magnitude for both fluid and solid at the interface boundary.

Regarding the dynamic coupling condition, the forces at the interface boundary are balanced, as

$$\sigma \cdot n = \Gamma \cdot n$$

where

$$\Gamma = -pI + \mu(\nabla \vec{V} + \nabla \vec{V}^T)$$

$n$ represents unit vector normal to the interface boundary of the fluid and solid.

### 2.5. Numerical simulation and boundary conditions

An appropriate method for solving the FSI problems is ALE. In the ALE delineation, the nodes of the computational mesh may be transferred to the continuum in Lagrangian manner, or maintained fixed in Eulerian fashion or moved in an arbitrarily specific way to provide a continuous rezoning potentiality [34].

Given the spatial coordinate $x$ and velocity $v$ for the fluid domain, the solid is defined likewise by the coordinate $X^s$ in the primary solid domain $\Omega^s_0$. Additionally, $x^s(X^s, t)$ and $v^s$ denote its spatial coordinate and velocity in the present solid domain, respectively. Hence, the difference between the present and primary coordinates is represented by the solid displacement $u^s$.

$$u^s = x^s - X^s.$$  

In fact, the FSI involves two components: 1) the interposition of fluid grid velocity $V(x, t)$ and the structural nodal velocities $V^s(x^s, t)$; 2) the inclusion of the FSI force $f^{FSI,s}(X^s, t)$ in the fluid domain $f^{FSI}(x, t)$.

The following equation represents the FSI force for an incompressible structure solved by a Lagrangian description.

$$f^{FSI,s}_i = -(\rho^s - \rho^f) \frac{\partial^2 u^s_i}{\partial t^2} \bigg|_{\Omega^s} + \sigma^s_{i,j} \bigg|_{\Omega^s}$$

where $\sigma^s_{i,j}$ stands for the derivative of Cauchy stress in the solid domain and $\Omega^s$ denotes the solid region. The Dirac delta function, $\delta$ is responsible for distribution of the FSI force from the structure into the fluid domain

$$f^{FSI}(x, t) = \int_{\Omega} f^{FSI,s}(X^s, t) \delta(x - x^s(X^s, t)) \, d\Omega.$$  

(11)
Reproducing Kernel Particle Method (RKPM) is adopted as an appropriate delta function to apply the two above-mentioned components of the FSI delta function. Similarly, the same Dirac delta function is utilized to couple the velocity fields from both fluid and solid domains

\[ v_s^i(X_s, t) = \int_{\Omega} v_i(x, t)\delta(x - x^s(X_s, t)) \, d\Omega. \]  

(12)

To simulate the peristaltic force applied to the elastic wall, a Gaussian distribution along the length of the ureter was employed, which is expressed as follows:

\[ \exp\left(-\frac{(Z_S - (Z_0 + V_0 t/W))^2}{2}\right) \]  

(13)

where, \( V_0 \) denotes the constant velocity of peristaltic propagation, \( Z_0 \) starting point of the force, \( W \) the Gaussian distribution width, and

\[ Z_S = \frac{Z}{W} \]  

(14)

where \( Z \) shows the \( z \)-component of the cylindrical coordinate system.

The Gaussian distribution is 1 mm wide and moves at the constant velocity of 2 cm/s equal to the average peristalsis velocity in the ureter [35]. For engaging and ending the force applied to the elastic wall, the smooth Heaviside function was used. When the peristalsis force was applied to the elastic wall onsets, it takes 0.05 s to reach the maximum value and it ends at 1.3 s from the start. The obstructed section in the POM cannot undergo the peristalsis. Hence, for simulating this defect, the peristalsis ends when the prevesical non-peristaltic segment arrives. The walls in both models were fixed to prevent the wall motion along the longitudinal direction. The inlet pressure was assumed 80 Pa, but different outlet pressures were applied to the outlet boundary for the pressure gradient analysis.

The physically controlled mesh containing 15144 triangular elements and 714 quadrilateral elements near the interface boundary was applied to the models in normal ureter. In the POM model, the mesh includes 9471 triangular elements and 668 quadrilateral elements near the interface boundary. Increasing the number of elements just extended the solving process and the results were the same. Hence, mesh independency was achieved (Figs 3–4).

3. Results and discussion

The urine flow in an open ureter with VUR was investigated. The peristalsis plays an important role in transferring the urine from kidney to the bladder, which was simulated using the FSI method. Furthermore, a sample of POM was modeled. The main goals of this paper are analyzing urine flow in ureter with VUR, predicting the possibility of urine reflux from bladder to the ureter, studying the urine viscosity effect on its velocity, studying the POM model, and comparing its attribute with the normal ureter.

3.1. Validation

The present ureter model is validated against previous studies. As pointed out earlier, Vahidi et al. [32] simulated a finite-length axisymmetric tube to study the model parameters effects on ureteral pressure/flow relations. The reported urine outlet velocities and the present investigation results in the case of non-peristaltic flow are shown versus the distance from the ureteral axis in Fig. 5 while Young’s modulus is
taken at 5 kPa. It is mandatory to mention that the ureter diameter is not constant and becomes smaller approaching the bladder. Thus, the ureter radius is taken 6 mm in our major model to study the UVJ malfunction and reflux in the ureterovesical area.

The results obtained in this paper are in good agreement with those of the past researches (Table 1). Griffiths and Notschaele [22] developed a simple mechanical framework in one minipig ureter to understand the propagation of the isolated bolus. One part of their study was the investigation of a relation between the mean flow rate in the bolus and the mean velocity of its leading edge. They used a roller to determine the mean velocity of leading edge over the length of the ureter. In another study, Griffiths [35]
Fig. 5. Validation of the present study through comparison with Vahidi et al. [32]. Outlet velocity vs. ureter radius in a non-peristaltic ureter. Related specifications were $\Delta P = 0.3$, Young’s modulus $= 5$ kPa, ureter radius $= 3.5$ mm.

Table 1
Some important findings of experiments and simulations

<table>
<thead>
<tr>
<th>Investigators</th>
<th>Specifications</th>
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<tbody>
<tr>
<td>Griffiths and Notschaele [22]</td>
<td>Flow rate in the bolus depends on the velocity of the leading edge. When the mean velocity of leading edge was 2 cm/s, the mean flow rate was equal to 0.05 ml/s.</td>
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<tr>
<td>Griffiths [35]</td>
<td>The average carrying capacity was apparently about 2 ml/min. The average ureteral unsticking pressure was 7 cm H$_2$O.</td>
</tr>
<tr>
<td>Vahidi et al. [32]</td>
<td>Urine flow rate was about 0.15 ml/s when the maximum height of peristaltic was 1.12 mm. Related parameters were: wave motion velocity $= 2$ cm/s, pressure gradient $= 0.3$ pa and Young’s modulus of 5 KPa.</td>
</tr>
<tr>
<td>Present study</td>
<td>Flow rate changed to variation of the bladder pressure. Urine flow rate during the peristaltic motion ($t = 1$ s) was equal to about 0.075 ml/s, while pressure gradient was 0.3 pa. Related parameters were: wave motion velocity $= 2$ cm/s and Young’s modulus $= 5$ KPa. At the POM, outflow rate was approximately zero.</td>
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investigated the effect of bladder pressure on pyeloureteral pressure/flow relations, theoretically and experimentally. They demonstrated that the renal pelvis was well isolated from the variation in the bladder pressure at flow rates below 2 ml/min. However, at higher flow rates, bladder pressure changes were transmitted to the pelvic. Table 1 shows some specifications of several studies. In addition, flow rate value obtained in each of them is presented.

3.2. Fluid velocity

As the peristaltic motion in the ureter starts, the urine average velocity increases up to about 13 times. Figure 6 illustrates the distribution of velocity magnitude during the peristaltic wave motion when the
pressure gradient is 0.3 Pa. The maximum speed was in the front region of the peristaltic wave. As can be seen in Fig. 6, urine high velocity spreads to further areas by peristaltic wave advancement.

Figure 7 shows the urine velocity vectors at the end times of peristalsis when it discharges the urine to the bladder. In practice, there is the UVJ that closes when the bladder pressure rises and the bladder expands. This phenomenon prevents the urine reflux to the ureter. However, in some cases when the UVJ is incompetent, it cannot prevent the urine reflux. Figure 7 is related to the ureter with VUR that illustrates the urine reflux from the bladder in the final moments of peristalsis.

Due to the high bladder pressure at the end of peristalsis, urinary reflux occurred and urine returned to the ureter at a rate of about 5 times the normal speed of urine in the ureter during peristalsis. According to Fig. 7, reflux is limited to the contraction area and the velocity of backflow is slow at $t = 1.26$ s. The peristalsis causes more severe reflux approaching to end of the ureter ($t = 1.30$ s). This reflux, which results from the absence of intravesical ureter or other derangement of the UVJ, can lead to PRM. Actually, UVJ reflux takes place because of two main reasons: first, lack of UVJ hindrance versus urine flow back, and second, the high pressure of bladder transmitted to the ureter.

In the POM, urine moves with no regular pattern during peristalsis. The prevesical obstructed section is not able to transfer the urine flow properly because of the significant decrease in smooth muscle that accounts for a non-peristaltic condition. This non-peristaltic segment gradually leads to accumulation of
urine and ureteral dilation behind the obstructed section. Figure 8 illustrates the distribution of velocity magnitude vectors in the POM. The peristalsis moves on the wall to reach the obstructed segment. Not only did peristalsis not increase the speed of urine, but also it produced the irregular flows. In fact, peristalsis plays no effective role in transferring the urine at the POM.

Urine accumulation and reflux behind the obstructed area gradually lead to dilated ureter. According to the velocity distribution in Fig. 8, although the maximum velocity magnitude in the megaureter is 40 mm/s, urine discharge is much less in comparison with the healthy ureter due to prevesical stenosis and lack of the peristalsis in the obstructed area.

The relationship between the output pressure and urine velocity magnitude at the end of ureter are shown in Fig. 9. In all models, the inlet pressure is assumed 80 Pa. For different output pressures, the urine outflow velocity is shown at the ureter with VUR. Urine velocity decreased due to peristalsis reduction at the final moments. Also, with the rise in bladder pressure, urine overall speed reduced. By passing the output pressure from input pressure, the reflux occurred gently at end of the ureter. Higher outlet pressures led to the reflux expanding in more areas of the ureter. This reflux in severe conditions can go up through the ureter to the renal pelvis and causes severe kidney infections.

Volumetric outflow rates of the ureter with VUR and POM are shown in Fig. 10 during the peristalsis at a specific time. They are obtained and illustrated for different pressure gradients. As seen in Fig. 10, the volumetric outflow magnitude declined by ΔP rising. Eventually, reflux took place during peristaltic at the pressure gradient of ~0.6 pa. In this case, peristalsis itself helps more backflow along the ureter. The urine outflow rate in POM was 0.0012 ml/s, much less in comparison with the healthy ureter. The obstructed section accounts for this low outflow rate value which leads to urine accumulation and megaureter.
Fig. 8. Velocity distribution and vectors in the POM during peristaltic motion at the prestenosis section.

Fig. 9. Comparison of the urine outflow velocity for different back pressures versus time. Inlet pressure is equal to 80 Pa.
Fig. 10. Urine volumetric outflow rate for different pressure gradients versus time. The magnitudes brought up in this figure determine variations of urine outflow rate during peristalsis ($t = 1$ s) and quantity of VUR reflux at $t = 1.28$ s. Large amount of VUR reflux occurs at $t = 1.28$ s. Peristalsis ends at $t = 1.3$ s.

Fig. 11. Variations of viscous stress along the FSI boundary at different time intervals in the case of $\Delta P = 0.3$ Pa, peristaltic velocity = 2 cm/s and Young’s modulus = 5 KPa. Times related to different moments of peristalsis are shown in the figure.
Viscous stresses

Viscous stress profiles on the interface boundary at different time intervals are obtained and shown in Fig. 11. The profiles represent viscous stresses in a limit length-ureter during the peristalsis. As can be seen in Fig. 11, at the initial times of peristalsis propagation (t = 0.15, t = 0.5, t = 0.8, t = 1.1, t = 1.26), viscous stresses were higher in contractive throat. However, they decreased gradually when the peristalsis approached the ultimate section of the ureter. The reason for this phenomenon is a sudden urinary acceleration at the onset of peristalsis and a noticeable difference in wave speed of wall contraction and the urine velocity near the wall. Therefore, the primary parts of the ureter wall tolerate more stresses. On the other hand, the stress intensity declined as urine velocity increased adjacent to the contraction wave. According to the profiles related to times t = 1.28 s and t = 1.30 s in Fig. 11, viscous stresses in the prevesical segment were applied to the wall in the opposite direction. These profiles correspond to the VUR in the ureter with abnormal UVJ function. These severe stresses resulting from backflow are one of the main components for ureteral dilation (PRM). Here, the outlet pressure was assumed 79.7 Pa.

Figure 12 shows viscous stress profiles on the interface boundary at different time intervals in the POM. At the time t = 0.8 s, peristalsis was still some distance away from the obstructed area. Therefore, the average magnitude of the viscous stresses was small, and only in the stenosed non-peristaltic area, it increased slightly. By the time t = 0.85 s, when the peristalsis was near the stenosed segment, urine backflow occurred because of the collision with the obstructed area. This backflow led to a high fluctuating...
Fig. 13. Outlet urine velocity during peristalsis ($t = 0.9$ s) along ureter radius with different viscosity values in the case of $\Delta P = 5$ Pa, peristaltic velocity = 2 cm/s and Young’s modulus = 5 KPa.

Fig. 14. Outlet urine velocity during VUR reflux ($t = 1.3$ s) along ureter radius with different viscosity values in the case of $\Delta P = 5$ Pa, peristaltic velocity = 2 cm/s and Young’s modulus = 5 KPa.

stresses in the prestenosis section. In practice, these severe stresses result in more dilation of the POM. As can be seen in Fig. 12, when the peristalsis reached to the obstructed area, not only did the viscous stresses grow in the prestenosis section but also it intensively grew in the stenosed section because of low
but severe urine flow. The peristalsis disappeared by the time \( t = 0.95 \) and the viscous stresses decreased gradually in the whole length of POM. Hence, it can be realized that peristalsis plays a negative role and results in more POM dilating.

### 3.4. Effects of viscosity

The behavior of the urine flow in the ureter depends on many parameters such as the wall characteristics and urine viscosity. Urine, which is predominantly water, behaves as a Newtonian fluid so it can be characterized by a single coefficient of viscosity. Urine viscosity is temperature dependent. However, in special conditions such as infection and blood presence, it can change drastically [36]. At the temperature of 37 °C, urine viscosity is ultimately 1.3 mPa.s. Here, three different viscosities were given to compare the outlet urine flow during peristalsis and reflux. As can be seen in Fig. 13, higher viscosity encompasses less outflow rate. Actually, viscosity increases the urine resistance, proportionally [37].

This is also the reason for slower reflux as shown in Fig. 14. At the high viscosity magnitudes, it takes longer for the urine to discharge. In severe pressure gradients, the effect of rising viscosity lessens. However, in low-pressure gradient, viscosity change leads to alternative flow rates. Here, the pressure gradient was assumed 5 Pa.

### 4. Conclusion

The aim of this study was to simulate two abnormal ureters by applying physiological parameters. The FSI method helped with the better simulation of fluid-elastic wall interaction. Consequently, it would be declared that proximal portion of the ureter is exposed to more stresses. However, in a ureter with UVJ dysfunction, by creating the reflux at the end of the ureter, the stresses increased in the opposite direction that could lead to PRM. Also in the POM, not only did the peristalsis not play any positive role in urine flow, but it also helped with more dilation of the prestenosis portion.

Adopting the FSI in simulations revealed the details of behavior of urinary tract peristalsis. In the past studies, ureteral peristalsis had been restricted to a rigid contact surface for applying the peristalsis wave. In this study, an axisymmetric Gaussian distribution was used to engage the peristaltic force along the ureter. This eliminated the predetermined wall displacement limitation. In addition, there had been no precise investigation of the abnormal ureters such as ureter with VUR and POM models, which were provided in this study. Although mammalian ureters vary widely in terms of size, shape, material properties, and loading conditions, the ureter characteristics are assumed close to the physiological parameters. Furthermore, the effect of the urine viscosity in flow was studied and the results represented that the urine velocity reduces when the viscosity rises. However, the urine reflux was more severe when the viscosity value was lessened. It should be emphasized that due to undeniable simulation limits, an accurate simulation of urine flow in abnormal ureters might help with a better diagnosis and treatment. There are still many unsolved problems in this field. For instance, interactions between two parts of the urinary system, the system of peristalsis pacemaker, and the effect of inconstant peristalsis velocity, have not been investigated yet. Furthermore, the presented model can be used to study the effect of non-Newtonian fluids on peristalsis. In addition, more sophisticated models are needed to understand the abnormal ureter’s behavior in more detail.
Conflict of interest

None to report.

References


