



# Numerical simulation of hemodynamics in membranous obstruction of the suprahepatic inferior vena cava based on a subject-specific Budd-Chiari syndrome model

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## ABSTRACT

**Background:** This study was performed to determine the hemodynamic changes of Budd-Chiari syndrome when the inferior vena vein membrane is developing.

**Methods:** A patient-specific Budd-Chiari syndrome vascular model was reconstructed based on magnetic resonance images using Mimics software and different degrees (16%, 37%, and 54%) of idealized membrane were built based on the Budd-Chiari syndrome vascular model using Geomagic software. Three membrane obstruction Budd-Chiari syndrome vascular models were established successfully and fluent software was used to simulate hemodynamic parameters, including blood velocity and wall shear stress.

**Findings:** The simulation results showed that there is low velocity and a low wall shear stress region at the junction of the inferior vena cava and the branches of the hepatic veins, and swirl may occur in this area. As the membrane develops, the size of the low velocity and low wall shear stress regions enlarged and the wall shear stress was increased at the membrane region. There was a significant difference in the mean values of wall shear stress between the different obstruction membrane models ( $P < 0.05$ ).

**Interpretation:** Hemodynamic parameters play an important role in vascular disease and there may be a correlation between inferior vena cava wall shear force changes and the slow development process of the inferior vena cava membrane.

## 1. Introduction

Budd-Chiari syndrome (BCS) has been defined as an obstruction of the hepatic venous outflow tract in the absence of right heart failure or constrictive pericarditis (Burra et al., 2016). BCS may also be called hepatic venous outflow tract obstruction. The obstacle causing BCS may be located in the small or large hepatic veins or on the suprahepatic portion of inferior vena cava (IVC), but does not include sinusoidal obstruction syndrome/hepatic veno-occlusive disease (Valla, 2017). There is no clear evidence for a difference in incidence of BCS between the West and East. Throughout the world, nearly all cases of BCS appeared to be caused by hepatic venous obstruction with or without involvement of parts of the IVC. However, in China, associated involvement of the IVC may be more common. As we studied, up to 70% of the 1148 BCS patients hospitalized in the Affiliated Hospital of Xuzhou Medical University had stenosis involving with the entire

length of the IVC or merely a short portion, and appeared as a membrane or “web” (Okuda, 2002; Zhuang et al., 2011). The location of the membrane is often at retrohepatic region. However, the mechanism underlying IVC membrane has not yet to be elucidated.

It is reported that hemodynamics played an important role in the pathogenesis of cardiovascular disease, so the study on inferior vena cava hemodynamics is of great clinical significance (Bale-Glickman et al., 2003; Stroud et al., 2002). At present, clinical measurement and model experiments are the two common methods to study the BCS hemodynamics. The methods used in clinical measurements include Doppler, magnetic resonance imaging (MRI), computerized tomography (CT), and digital subtraction angiography (DSA). But these methods cannot illustrate the dynamic development process of IVC membrane (Buckley et al., 2007; Zhang et al., 2015). Model experiments mainly refer to animal experiments. However, due to the complexity of the BCS occurs, to our knowledge there is no report about BCS

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animal model has established (Aydinli and Bayraktar, 2007). Moreover, as animal models are usually different from humans, it is difficult to obtain a convincing conclusion capable of explaining the hemodynamic in the BCS blood vessel from animal models. In addition, owing to the compensatory effect of blood vessel, there is no symptom in the early stage of the IVC membrane development and the hemodynamics change is always not elucidated.

Recently, computational fluid dynamics (CFD) have been extensively used in biology and biomechanics. Numerous experimental and computational studies have investigated the influence of flow on the vascular disease. At present, hemodynamic studies mainly focus on cerebral aneurysms (Munarriz et al., 2016), atherosclerosis (Hoi et al., 2011), portal hypertension (Morales-Ruiz et al., 2015) and other arterial vascular diseases. Studies showed that high shear stress may damage the vascular wall (Reneman et al., 2006), in contrast, in low wall shear stress regions, where mass exchange between the flow and the vascular wall may reduce (Alaraj et al., 2015). Increased flow rate causes dilatation of the artery, while decreased flow rates causes thickening of the intima to reestablish this normal value of the wall shear. Thus, hemodynamics may play an important role in the development of vascular diseases. In addition, it was observed vascular lesions tend to be localized at sites of branching, bifurcations, and artery curvature. Coincidentally, the position of the membrane tends to be located at sites of retrohepatic IVC where blood vessels converge together. Based on this, hemodynamics seems to be also important to the vein disease, especially the BCS with IVC membrane. Therefore, the impetus for this study is to determine the relationship between hemodynamics and membrane growth in BCS, using CFD simulation based on patient-specific model obtained from MRI images.

## 2. Methods

### 2.1. Data acquisition

A BCS patient with IVC membrane obstruction participated in the current study. The patient, a 46-year-old female, presented with a good prognosis and no apparent vascular anomalies after interventional therapy with elimination of the membrane obstruction effect. The MRI images used in the current study were obtained from the Department of Radiology (Affiliated Hospital of Xuzhou Medical University, China). A 3.0 T GE MRI scanner with an 8-channel abdominal TORSOPA coil was used to collect images. The scan range included the lower edge of the heart to the entire pelvic region. The MR scan parameters were set as follows: LAVA sequence; matrix, 288 \* 256; turning angle, 12°; bandwidth, 83.33; FOV, 40 cm; layer thickness, 0.8 mm; enhanced contrast DTPA-Gd with a total injection of 0.1 mmol/kg and an injection speed of 3 ml/s; enhanced scanning venous phase, 70 s; and delay period, 120 s. The images were stored as a DICOM 3.0 standard file format.

### 2.2. Vascular modeling

In this paper, a realistic or patient-specific suprahepatic IVC system was reconstructed using Mimics V10.0 (Materialize, Leuven, Belgium) using MRA images by converting medical image data into image-based 3D surfaces and a volume representation, as shown in Fig. 1. The construction process of the specific model involves the following three major steps: (1) non-invasive image acquisition; (2) imaging processing; and (3) 3D reconstruction to create a volumetric image representation. The MRA images were imported into Mimics software for the generation of 3D reconstructed images. The reconstruction process also involved smoothing, contrast enhancement, and automatic edge detection. A 3D vascular model was created by Mimics 3D module and exported to surface tessellation language (STL) for the next step (finite element meshes). Because the obstructed membrane is thin and difficult to reconstruct, an ideal membrane was created in the paper. The formation of the membrane into the IVC was performed in three steps.

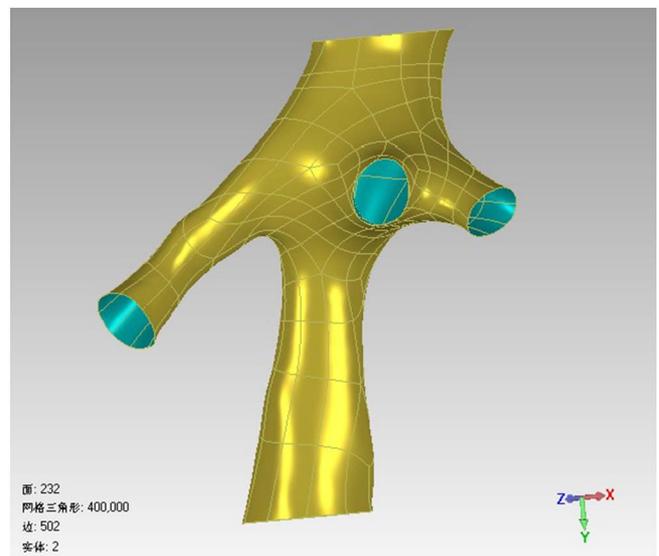


Fig. 1. Reconstructed model retrohepatic IVC system.

First, the BCS model was imported into the reverse engineering package of Geomagic Studio V12.0 (Geomagic, Inc., Research Triangle Park, NC, USA) and the location of the membrane was settled. Second, the membrane was generated by the software. Third, a Boolean subtraction operation between the membrane and the BCS geometry was performed. A merged model representing the reconstructed vessel and the membrane was thus obtained. The degree of membrane obstruction was controlled by the size of the membrane. The BCS model with a membrane is shown in Fig. 2. To study the effect of the membrane obstruction on the vascular blood flow, the position of the membrane was fixed 2 cm above the confluence of the hepatic vein and IVC, where the obstruction commonly occurs. Three IVC membrane BCS models with different degrees (16%, 37%, and 54%) of membrane obstruction were built. The Geomagic Studio created the necessary points, body edges, and faces for defining the finite element meshes, loads, and boundaries. Then the CAD module was generated for volume meshes and exported as IGES files for further hemodynamic simulation.

### 2.3. Hemodynamic modeling

The commercial finite element package, fluent 6.3.26 (ANSYS, Inc., Canonsburg, PA, USA), was used for CFD simulations. The blood flow through the vasculature is modeled as a homogeneous, incompressible, steady Newtonian fluid with a dynamic viscosity of 0.0035 P and a mass density of 1050 kg m<sup>-3</sup> (Qiao et al., 2016). The BCS blood vessel wall is assumed to be rigid and no slip condition was applied (Wang et al., 2014). As the flow is assumed to be Newtonian and laminar, 3D incompressible Navier-Stokes equations were used in the simulation (Caiazzo et al., 2015; Chen et al., 2015). The blood flow was solved by the finite volume method in fluent 6.3. The mass flow rate at the inlets was all set at 0.1 m/s, and the blood pressure was set to 0 Pa at the exit.

## 3. Results

### 3.1. Model without a membrane

The velocity distribution of the BCS model without a membrane is shown in Fig. 3. As Fig. 3 shows the velocity at the junction of the IVC and the branches of the hepatic veins (HVs) and swirl. Wall shear stress decreased in the junction area, but increased above the junction, as shown in Fig. 4. Coincidentally, the BCS membrane often occurred at the areas of velocity and wall shear stress change.

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