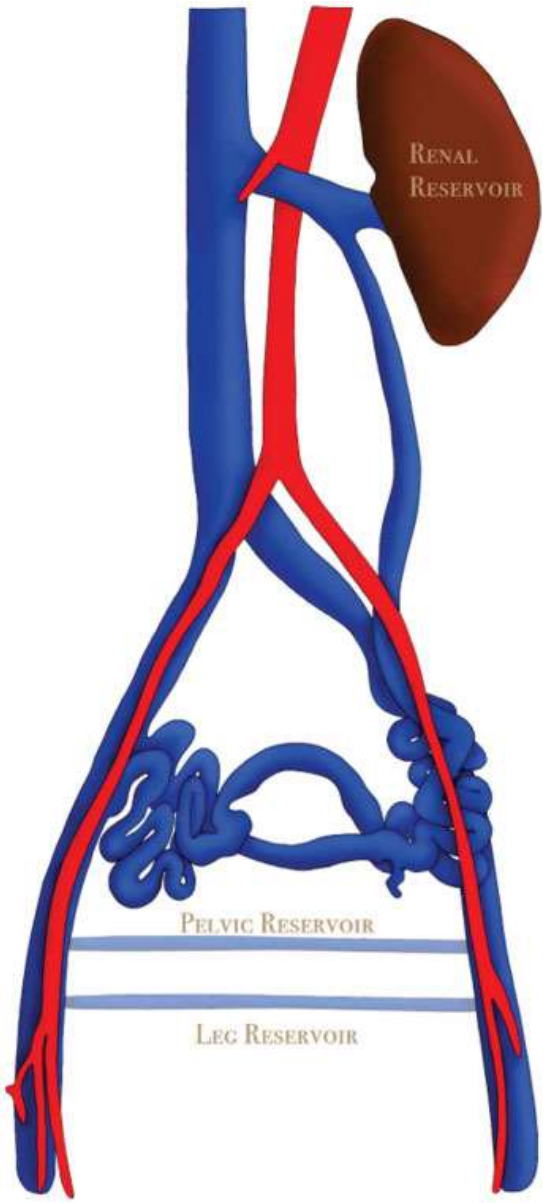

Fig. 1 Demonstrates an artist rendering of pelvic venous anatomy showing the pelvic reservoir and leg reservoir in relation to the anatomic configuration of the iliac veins, inferior vena cava and ovarian veins.

matrix metalloproteinases. These enzymes, when activated, cleave proteins responsible for cell-to-cell integrity in vein wall muscle layers and valvular structure. After enzymatic cleavage, and white cell infiltration secondary to endothelial dysfunction, increased vessel capacitance and worsening valvular incompetence occur. Through this cyclic mechanism, increased venous pressure (venous hypertension) develops in the pelvic viscera, particularly within the uterine walls and initiates activation of local nociceptors which may result in the clinical presentation of CPP. Additionally, in the pelvis, there are venous escape points (deep perforator veins) through which the pelvic venous plexus communicates with the superficial veins of the upper thighs, which themselves may become incompetent resulting in the lower extremity, vulvar, and perineal manifestations described earlier.[14]

Imaging Assessment

Since the clinical presentation of CPP from a venous cause may have overlapping features with other etiologies of CPP, diagnostic imaging is an important component of patient evaluation and helps direct future treatment strategies. In many OB/GYN practices, PeVD is considered a diagnosis of exclusion based on the frequency of other pathologies such as endometriosis and pelvic floor dysfunction. Current gynecological US guidelines do not seek to identify pelvic varices. However, imaging demonstrating pelvic varices may be important to identify patients who may benefit from endovascular treatment. Although dilated veins in the pelvis are a common finding on imaging studies occurring in up to 15% of women aged 20 to 50 years,[15] not all of these patients will have CPP. No clear imaging criteria to define PeVD have been published.

Imaging definitions for pelvic venous disease based on transfundal venography performed by Beard and colleagues have been modified to be utilized in noninvasive imaging strategies in attempt to identify the pathophysiology of PeVD.[15,16] The predominant factors involved in imaging diagnosis include ovarian vein diameter, and the presence or absence of these findings: ovarian vein reflux, pelvic variceal reservoir, iliac vein obstruction, and renal vein compression. The optimal imaging tool may vary based on the availability of the modality and local expertise but can be extrapolated across modalities with the knowledge that the optimal treatment is not yet clearly defined. The SVP consensus document proposes a major criterion for establishing the diagnosis of PeVD as the presence of varices in the ovarian or uterovaginal plexuses $\geq$ 5 mm in diameter regardless of what imaging technique is utilized.[17]

Ultrasound

Much of the abdominopelvic viscera can be evaluated accurately with transvaginal (TV) or transabdominal (TA) US techniques. Specifically, sonography can be used to look for uterine, ovarian, bowel, and vascular pathologies if performed with the appropriate protocols. Patients evaluated for CPP often undergo US examinations given its accessibility, cost-effectiveness, and convenience. However, most of these outpatient protocols do not include a vascular assessment. Additionally, limitations to venous evaluation are present, such as vein collapse from full bladder (required in TV OB/GYN protocol) and supine positioning. Although TVUS may depict the vessels more accurately, as mentioned few protocols include an assessment for the presence of varices and US technologists are directed by image acquisition protocols that include evaluation of uterus and ovaries only when ordered by primary care practitioners.

Sonographic imaging protocols in PeVD are designed not only to identify pelvic varices but to evaluate for the pathophysiology described above. Labropoulos et al recommended a standard PeVD TA sonographic evaluation, as this is the best

