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Review article

Inferior vena cava resection and reconstruction: Technical considerations in the surgical management of renal cell carcinoma with tumor thrombus

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Abstract

The mainstay of treatment for renal cell carcinoma with invasion of the inferior vena cava (IVC) is complete surgical extirpation. Cases complicated by adherent or invasive tumor thrombus represent a special technical challenge due to the need for resection of a large segment of the IVC. The aim of this review is to describe the indications and surgical approach for radical nephrectomy with en bloc resection of the IVC with or without venous reconstruction. In addition, special attention is paid to the relevant anatomical and hemodynamic considerations related to the development of venous collateral pathways secondary to IVC obstruction.

Keywords: Renal cell carcinoma; Tumor thrombus; Inferior vena cava; Resection; Reconstruction

1. Introduction

Renal cell carcinoma (RCC) is known to spread via the lumen of the renal vein into the inferior vena cava (IVC) in up to 10% of the cases [1,2]. At present, surgery provides the only hope for a potential cure in these patients. However, the resection of RCC with tumor thrombus is technically demanding and has historically been associated with a high rate of perioperative morbidity and mortality [3,4]. This is especially true in cases complicated by venous wall invasion, which require tangential or en bloc resection of the IVC to achieve complete surgical removal [5–8]. These procedures are facilitated by the redirection of venous blood flow through collateral pathways, which develop during chronic obstruction to promote venous blood return to the heart. In cases of acute obstruction where such collateral pathways have not developed, reconstructive vascular procedures are required to ensure adequate venous drainage.

The purpose of this review is to describe our evolving surgical technique for the resection of RCC with tumor thrombus when IVC resection or reconstruction is required. The indications and technical issues related to these procedures are discussed according to our current practice so as to provide the reader with guidance for the management of these difficult cases. In addition, we provide a detailed discussion of the relevant anatomical and hemodynamic considerations in relation to the development of venous collateral pathways secondary to caval obstruction.

2. Clinical presentation

Obstruction of the IVC may be clinically silent or can present with a wide range of symptoms. Clinically, the alteration in blood flow may be heralded by the development of a venous collateral network, thus making the absence of symptoms the most frequently observed scenario. On the contrary, this condition may cause debilitating lower extremity edema (LEE), back pain, and venous stasis ulceration. This constellation of symptoms is sometimes referred to as the "IVC obstruction syndrome." Liver and

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kidney functions can also be disrupted if the intrahepatic vena cava or the renal veins are involved [9–11]. In a surgical series from our group, we observed no instances of LEE; however, we did commonly observe hepatomegaly, ascites, and jaundice (Budd-Chiari syndrome) secondary to venous flow interruption at the level of the major hepatic veins (MHVs) [12].

3. The venous collateral network

The marked variation in the clinical picture of the IVC obstruction syndrome is due to the variable formation of various abdominopelvic collateral venous pathways that result from chronic vascular obstruction [13,14]. An understanding of the underlying anatomy and hemodynamics of venous collateral pathways has important diagnostic and prognostic implications in patients with obstructing RCC tumor thrombi. Familiarization with these collateral pathways may also aid in evaluating the different treatment options.

3.1. Anatomy

Collateral systems can be arranged into 2 groups: deep and superficial. The deep azygos-hemiazygos system is the earliest channel available and plays a dominant role in venous decompression of IVC obstruction at any level. The vertebral venous plexus communicates in a bidirectional fashion with the IVC and the azygos-hemiazygos system via segmental tributaries (Fig. 1). Secondary systems of the deep collaterals comprise the gonadal, ureteral, and, to a lesser extent, the inferior mesenteric vein of the portal system.

The 2 primary components of the superficial venous network that develop during IVC obstruction are the paired lateral thoracic and internal thoracic systems. The distal tributaries of the lateral thoracic system are the superficial epigastric and circumflex iliac veins, which reach the axillary vein through the thoracoepigastric and lateral thoracic veins. The external iliac veins drain through the inferior and superior epigastric veins into the internal thoracic veins. Communications with the lumbar and intercostal plexuses allow for redistribution of the superficial flow to the primary deep systems and the IVC above the level of the obstruction. Although considered by many to be the least important of all the collaterals, they do take on significance after several weeks of the acute phase [14]. As the obliterating process progressively involves the more common collateral channels, the already known anastomosis between the superficial veins and the retroperitoneal branches of the superior mesenteric vein plays a more important role in venous decompression.

3.2. Hemodynamics

The determinants of the extent of collateral development are the location or level of the obstructed venous segment, the length of the obstruction, and the number of veins involved. These 3 elements individually and in combination cause an increase in venous resistance and thus determine the extent of collateralization.

Deep collateral channels may be adequate in the development of a fully compensated collateral circulation when the IVC is obstructed below the renal veins. In contrast, midlevel IVC occlusion results in congestion of the kidney and usually induces a less compensated state of obstruction. Collateralization from this level of occlusion has been shown to involve the portal system collaterals, wide dilatation of the vertebral veins, and perinephric and capsular drainage into the azygos-hemiazygos system [14] (Fig. 2).

In upper level obstructions, such as those involving the MHVs, communications between the IVC and superior vena cava develop from the portal systems both deep and superficial. Additionally, the vertebral plexus becomes widely dilated. In this setting, the finding of visceral congestion suggests inadequate collateralization.

4. Diagnosis and preoperative planning

An accurate assessment of the proximal extent of a given tumor is critical for preoperative planning. Most commonly, computed tomography is used to stage the extent of disease as well as to evaluate for the presence of venous collateral pathways (Fig. 3). Alternatively, magnetic resonance imaging may be used for this purpose. One distinct advantage of magnetic resonance imaging is its high sensitivity for detecting caval wall invasion [15].

To date, a number of systems have been described for the classification of tumor thrombi. Perhaps, the most commonly used for surgical considerations is the scheme described by Neves and Zincke [16]. For level III tumors, we routinely use our own modified classification system, which subdivides tumors (IIIa–IIId) based on their proximity to the ostia of the MHVs [17].

5. A rational approach to IVC resection

IVC wall invasion portends a poor prognosis, and therefore historically it was felt that patients requiring vena caval resection could only rarely be cured by an operation alone [18]. Accordingly, many authors recommended against performing this major surgical procedure given the anticipated short length of postoperative survival [19]. In 1991, however, a landmark paper by Hatcher et al. [20] changed this way of thinking after the authors demonstrated an improvement in 5-year survival from 26% (median 1.2 y) to 57% (median 5.3 y) with successful resection of the IVC invaded by tumor.