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Review article

Imaging in the staging of renal cell carcinoma

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Abstract. Imaging is extremely important in determining the type of surgery undertaken in patients with proven renal cell carcinoma. In this review, the strength and limitations of each of the relevant techniques are outlined, highlighting particularly the correlation between the imaging findings and the pathological staging. Over the past decade, CT has become the most widely used technique for staging renal cell carcinoma, partly due to the very high overall accuracy of up to 90% that has been achieved. MRI appears to have a similar overall accuracy to CT, whereas ultrasound is less accurate than CT or MRI in the overall staging of tumours. However, ultrasound is extremely accurate in identifying and localising the clinically important tumour extension into the intra-hepatic vena cava and right atrium and if local surgical practice requires only a knowledge of venous invasion, a technically adequate ultrasound examination may suffice. All techniques are unreliable in detecting early perinephric spread.

Key words: Staging, Renal Cell Cancer

Introduction

After the diagnosis of renal cell carcinoma, staging is essential for appropriate management. Accurate determination of the extent of disease is necessary for assessment of tumour resectability and the appropriate surgical approach for excision of the primary tumour and, where present, tumour thrombus. Staging of renal cell carinoma depends on both the histological grade and the clinical staging of the tumour. The choice of the appropriate imaging technique for clinical staging will include the consideration of several factors. The accuracy, limitations and advantages of each technique are important considerations which are also influenced by local factors, such as the availability of specific modalities or the information required by individual surgeons prior to surgery.

Staging classifications

Two systems of clinical staging are in common use: the Robson classification (Table 1) and the TNM classification of the International Union Against Cancer (UICC) (Table 2). Table 3 shows the approximate correlation between these two staging systems. Most urological surgeons continue to refer to Robson's classification, which is essentially a surgical staging appraoch. This system includes the important staging variables that have survived scrutiny over the years [1]. Confinement within the renal capsule, penetration into the perinephric fat, invasion into the renal vein, lymph node metastases and distant spread are all important in determining prognosis (Table 4). The TNM system is more specific and correlates better with potential curability and prognosis.

At presentation about a third of patients have stage I (T1–T2) disease [2], 10% stage II (T3a) disease; 25% stage III disease and about 30% stage IV disease [3].

Staging and prognosis

A clear relationship exists between survival and the anatomical extent of tumour spread. Metastatic disease appears to be the strongest predictor of survival [6]. Results in the literature are inconsistent regarding the value of tumour size in predicting survival from renal cell carcinoma. The overwhelming evidence is that the use of tumour size as a staging variable is ineffective in the absence of involved lymph nodes or metastatic disease in patients without local invasion of perinephric fat or other structures [6, 7]. However, a clear correlation exists between tumour size and the development of metastases [8–10] and, because of this, the results indicating a poorer prognosis for larger tumours are corroborated by many studies [11–13].

Table 1. Staging of renal cell carcinoma [4]

Stage	
1	Tumour confined within capsule
2	Extracapsular spread to perinephric fat but confined to Gerota's fascia
3	Tumour involvement of renal vein, inferior vena cava or regional lymph nodes
4	Invasion of adjacent organs or distant metastases

Table 2. TNM staging system for renal cell carcinoma

Primary tumour (T)	
TX	Minimum requirements to assess primary tumour cannot be met
T0	No evidence of primary tumour
T1	Small tumour. Minimal renal and calyceal distortion or deformity. Circumscribed neovasculature surrounded by parenchyma.
T2	Large tumour with deformity and/or enlargement of kidney and/or collecting system
T3a	Large tumour involving perinephric tissues
T3b	Tumour involving renal vein
T3b	Tumour involving renal vein and infradiaphragmatic vena cava
T3d	Tumour involving infra- and supradiaphragmatic vena cava and renal vein
T4a	Tumour exentending into neighbouring organs or abdominal wall
T4b	Tumour extending intracardially
Nodal involvement (N)	
The para-aortic and para-caval nodes are the regional lymph nodes	
NX	Minimal requirements to assess nodal involvement cannot be met
N0	No evidence of regional lymph node involvement
N1	Single homolateral regional node involvement
N2	Involvement of multiple regional or contralateral or bilateral nodes
N3	Fixed regional nodes (assessable only at surgical exploration)
Distant metastases (M)	
MX	Not assessed
M0	No (known) distant metastases
M1	Distant metastases present

Staging and surgery

Initial work by Robson et al. [4] showed that survival rates are sginificantly improved by radical nephrectomy compared with simple or partial nephrectomy and since that time radical nephrectomy has been the preferred method of treatment for renal cell carcinoma. Thus, as the perinephric fat is routinely resected during a radical procedure, the accurate differentiation between stage I and stage II (T1–T3a) has not been essential. More recently, however, less aggressive surgery has been advocated for some patients with low-grade non-invasive tumours, especially in the presence of a solitary kidney or multiple lesions [14, 15]. For all these patients, the distinction between stage I and stage II tumours becomes extremely important. Furthermore, the diagnosis of stage II disease (T3a) does carry some prognostic significance [4]. Also, stage II disease includes spread, either direct or haematogenous, to the ipsilateral adrenal gland, so identification of disease within the adrenal gland is extremely important when partial nephrectomy is being planned [16].

Table 3. Staging renal cell carcinoma: Robson's classification versus TNM

Robson	Disease extent	TNM
I	Tumour confined to kidney (small, intrarenal)	T1
	Tumour confined to kidney (large)	T2
II	Tumour spread to perinephric fat but within Gerota's fascia	T3a
IIIA	Tumour spread to renal vein or cava	T3b
IIIB	Tumour spread to local lymph nodes (LN)	N1–N3
IIIC	Tumour spread to local vessels and LN	T3b, N1–N3
IVA	Tumour spread to adjacent organs (excluding ipsilateral adrenal)	T4
IVB	Distant metastasis	M1a–d, N4

Table 4. Survival and anatomical extent of renal cell cancer [5]

Anatomical extent of tumour	% survival	
	5 year	10 year
Within renal capsule	65	56
Renal vein (RV) alone	66	49
RV + perinephric fat	50	33
RV + regional nodes	0	0
Perinephric fat alone	47	20
Regional nodes alone	33	17
Invading nearby structures	0	0

Lymph node involvement is important to detect preoperatively, usually occurs at the same time as bloodborne spread and carries a poor prognosis. Local lymph node involvement may acutally be an ineffectual barrier to tumour spread because the incidence of distant metastases is 50 % higher in patients with infiltrated lymph nodes [17]. Although lymph node resection is recommended by some surgeons [18], there is little evidence that lymph node clearance improves survival [19] and it is not universally performed.

Tumour thrombus occurs in 20 % of patients at presentation with renal cell carcinoma and the accurate assessment of its extent is vital to planning the proper surgical approach. Tumour thrombus in the renal vein only, can be dealt with by the routine ligation of the renal vein to prevent tumour embolization. Tumour in the inferior vena cava (IVC) necessitates a midline abdominal incision through which the inferior vena cava may be cleared. A key aspect of delineating thrombus within the IVC is demonstration of its upper extent. If the thrombus extends above the level of the hepatic veins, intraoperative cardiopulmonary bypass combined with a thoraco-abdominal approach is necessary to remove the entire tumour thrombus [20].

Detection of all sites of metastases can be important [21]. Nephrectomy has been shown to improve survival if only one organ contains metastases, particularly if in

bone [22]. The incidence of regression induced by nephrectomy in multiple metastases is, however, low (0.3 %) and the risk of mortality significantly greater [23]. Thus, in patients with multiple metastases or extensive lymph node infiltration, the treatment is usually palliative [20].

Imaging in staging

The interpretation of any imaging used to stage renal cell cancer must include evaluation of tumour size, shape, its interface with the renal parenchyma, perinephric extension, local and regional lymph node enlargement, spread into contiguous organs, involvement of the adrenals and local or distant metastatic spread [22]. Imaging techniques that are currently used for the local staging of renal cell carcinoma include CT, MRI, ultrasonography (US) and occasionally inferior venacavography [20]. Over the past decade CT scanning has become the most widely used imaging technique for the diagnosis and staging of renal cell carcinoma [20, 21]. US is also used to stage renal cell carcinoma, but in a relatively high proportion of cases overlying bowel gas precludes adequate visualization of the renal vessels, the infrahepatic IVC and the retroperitoneum [24, 25]. US is also inferior to CT or MRI in detecting muscle invasion [26]. Increasingly, MRI is proving to be a valuable method for staging and seems to be at least as accurate as CT [27, 28].

Computed tomography

The reported overall accuracy rate for CT in staging renal cell carcinoma ranges between 72 % and 90 % [29, 30]. Robson's stage I [T1–T2) tumours are defined on CT as lying entirely within the kidney with an intact renal capsule (Fig. 1). Stage II tumours (T3a) are diagnosed by extension of tumour *spread into the perinephric space.* The most specific sign of this spread is the presence within the perinephric space of a discrete mass measuring at least 1 cm in diameter (Fig. 2). Although the specificity of this finding is 98 % for stage II (T3a) tumour spread, its sensitivity is only 46 % [30] as the finding is absent in the majority of patients with perinephric extension. Renal capsular invasion is difficult to diagnose unless the tumour obviously extends into the perinephric space. Recognized signs on CT include an indistinct tumour margin, blurring of the renal outline, thickening of the perirenal fascia and strands of soft tissue spreading into the perirenal fat resulting in "webs" or "wispy" densities (Fig. 3). False-positive diagnoses do occur and in up to 50 % of patients with stage I (T1–T2) disease there can be perinephric stranding or fascial thickening without perinephric tumour spread [20, 21]. This can be attributable to perinephric oedema, fat necrosis or fibrosis from remote inflammation. Unenhanced vascular collaterals may also simulate perinephric soft tissue nodules [20]. As renal cancers are responsible for approximately 60 % of all cases of spontaneous perinephric haemorrhage, blood in the perinephric space may mask or simulate extracapsular extension [31–33]. Focal thickening of Gerota's fascia contiguous to the tumour tends to be a more reliable indicator of invasion than generalized uniform thickening (Fig. 3). Particularly problematic areas are posteromedially where the kidney abuts the psoas muscle, and anteriorly where the perinephric space and fat tend to be thin. On the right, a particular problem is experienced laterally where the duodenum lies closely related to the lateral margin of the kidney [34]. False-negative results occur with tumours in which the extracapsular spread is only microscopic.

Thus, early perinephric extension of tumour remains extremely difficult to detect and failure to distinguish between Robson's stage I and stage II (T1–T2 vs T3a) accounts for 50 % of all staging errors [35]. The sensitivity of CT in identifying stage II (T3a) disease is only between 44 % and 50 % [20, 30] with a specificity of about 90 % when all the signs are present [20, 30].

Demonstration of *lymph node involvement* identifies disease as Robson stage III or N1–N2 in the TNM sys-

Fig. 1 a, b. Stage I (T1–T2) renal cell carcinoma. **a** CT scan showing a renal cell carcinoma of the left kidney with no evidence of invasion of the perirenal fat *(arrow).* Note the flow artefact within the patent IVC *(arrowheads).* **b** Gradient echo MRI scan (30° flip angle, TR = 9.0, TE = 2.5) showing the same mass lesion without invasion of the perirenal fat. Note the absence of signal within the lesion *(arrows)* due to calcification demonstrated on CT

Fig. 2. Stage II (T3a) renal cell carcinoma. CT scan in a patient with renal cell carcinoma demonstrating definite spread into the perirenal space by showing a discrete 1 cm mass extending into the perirenal space *(arrows)*

Fig. 3. Stage II (T3a) renal cell carcinoma. CT scan through a right upper pole renal cell carcinoma, showing focal thickening of Gerota's fascia *(arrows)* with strands of soft tissue extending into the perirenal fat, just deep to it. Note that the remainder of the perirenal fascia is not thickened

Fig. 4. Stage IIIb (N2) renal cell carcinoma. CT scan demonstrating a left lower pole renal cell carcinoma with ipsilateral para-aortic lymph node enlargement shown on biopsy to be involved *(arrows)*

Fig. 5 a–c. Spiral CT to demonstrate patency of venous structures in renal cell carcinoma. **a** Postcontrast CT scan showing a left-sided renal cell carcinoma. **b** Coronal reconstruction clearly demonstrating the patency of the renal veins. The absence of contrast medium within the inferior vena cava (IVC) is due to the early timing of the scan. **c** Three-dimensional reconstruction of **a**

Fig. 6 a–e. Stage IIIA (T3b) renal cell carcinoma. **a** Postcontrast CT scan showing a large left renal cell carcinoma with thrombus within the left renal vein and IVC. **b** CT scan at a level slightly superior to **a** showing thrombus within the IVC resulting in a filling defect *(arrow).* **c** Postcontrast CT scan in the same patient at the level of the hepatic veins, showing patency of the IVC. **d** Coronal spin-echo MRI scan (TR = 500, TE = 25) in the same patient, showing the left renal tumour with thrombus extending into the left renal vein and into the IVC. Note the patency of the IVC at the level of the hepatic veins *(arrow).* **e** Longitudinal ultrasound scan in the same patient showing thrombus within the IVC which is nevertheless patent *(arrows)* at the level of the hepatic veins


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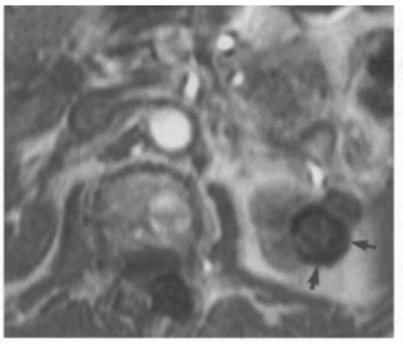
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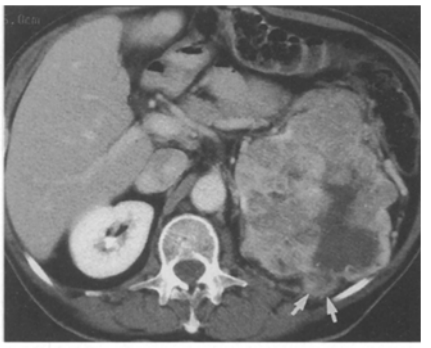
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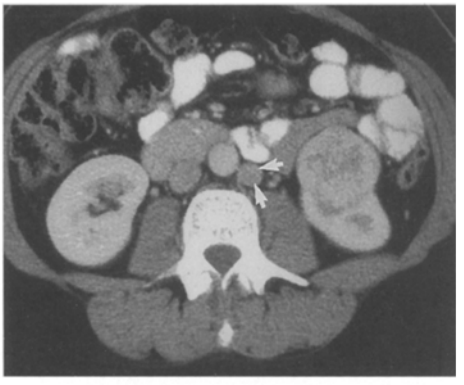
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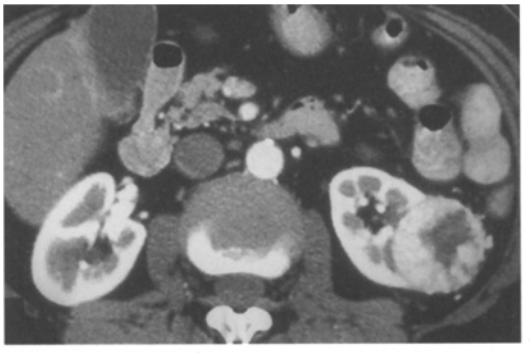
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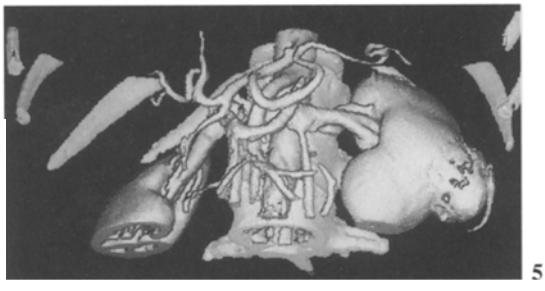
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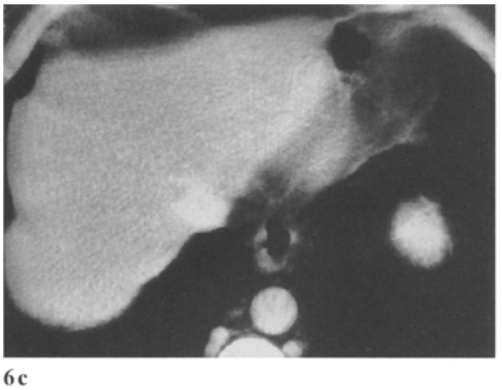
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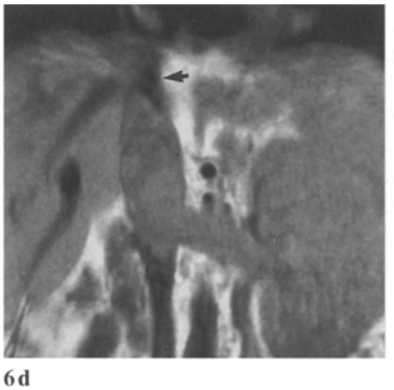
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tem. The lymphatic drainage of the kidney is highly variable. Usually, the collecting lymphatic vessels form an intrarenal plexus with four or five trunks which follow the renal veins and end in the ipsilateral para-aortic lymph nodes. Efferents from the lateral aortic lymph nodes pass to the contralateral side to form the lumbar trunk, and these lumbar trunks terminate in the cysterna chyli. However, direct connections to the thoracic duct and mediastinum do exist, accounting for the uncommon finding of mediastinal or hilar lymph node involvement, particularly on the right at the time of presentation.

As elsewhere in the body, and as with all cross-sectional imaging modalities, the detection of lymph node involvement on CT relies on detecting an increase in the size of infiltrated lymph nodes, and the limitation of using size criteria in identifying lymphatic metastases in renal cell carcinoma is well recognized [30, 35, 36] (Fig. 4). False-positive rates as high as 43 % have been reported due to reactive hyperplasia or other benign conditions when using 1.00 cm as the upper limit of normal [37]. The presence of primary tumour necrosis, or tumour thrombus, appears to be associated with a higher rate of reactive lymphadenopathy with a resultant increase in false-positive results on imaging [38]. False-negative results due to microscopic invasion of lymph nodes occur less frequently, being reported in only about 4–5 % of patients [37]. The overall accuracy of lymph node staging in renal cell carcinoma is reported as varying between 83 % and 89 % [29, 30, 35].

Evaluation of the *venous system* is critical both in the detection of venous thrombosis and in the determination of the level of thrombus formation. Optimal delineation of tumour thrombus requires meticulous technique. Helical (spiral) CT scanning, with or without reconstruction, is likely to improve the delineation of the venous structures [39, 40] (Fig. 5). A precontrast scan is still required. Our technique is to inject 100–150 ml of contrast medium intravenously at 2–3 ml/s. Spiral CT at 5 mm collimation is then performed through the kidneys and liver from bottom to top. The scan sequence is then immediately repeated from top to bottom. The purpose of this technique is to detect renal vein thrombi during peak intravascular opacification of the vein on the early scans, and to detect vena cava thrombosis and hepatic lesions during the second phase of the post-contrast examination. Other techniques have been described [20, 41].

Intrarenal venous invasion is beyond the resolving power of conventional CT [42]. The most important sign of venous tumour infiltration is a persistent filling defect following intravenous contrast administration, within the renal vein or IVC (Fig. 6 a–c). No false-positives are reported for this sign in the diagnosis of thrombus in association with renal cell carcinoma [30, 43]. A secondary sign of venous thrombosis is venous enlargement. This sign alone is of limited value and is associated with a 65 % false-positive rate and a 90 % false-negative rate for diagnosing thrombosis, partly because 78 % of renal cell carcinomas are hypervascular, generating increased flow enlarging the renal vein [43]. Conversely, tumour thrombus does not necessarily cause enlargement of the venous structures.

The sensitivity of CT for detecting venous invasion has been reported as 78–79 %, rising to 89 % for the detection of vena caval involvement [29, 30, 35, 43]. Data on the accuracy of spiral CT in assessing venous invasion are not yet available.

There are few difficulties in the diagnosis of venous thrombosis on CT. On the right side, where the renal vein is short and straight, the diagnosis may be more difficult than on the left side where the vein is longer and the collateral circulation is better developed. The cardiac status of the patient may preclude the use of a large bolus of contrast medium. Streaming of contrast medium (laminar flow layering) may produce false-positive diagnoses (Figs. 1, 5) and failure to identify more than one renal vein draining a kidney can result in false-negative findings [21]. Improved longitudinal reconstructions from axially acquired spiral CT images have largely reduced the previous difficulty in visualizing the upper limit of thrombus on the IVC. The cephalic extent of the thrombus related to the level of the hepatic veins, crucial in planning surgery, can now be made with greater certainty.

The distinction between direct venous extension of malignancy and bland blood clot is usually extremely difficult. The only reliable sign is neovascularity or enhancing tumour vessels within the thrombus itself, but this is often difficult to recognize. A less reliable distinction between the two can be made when bland thrombus is seen within the IVC separate from a patent entry of the renal vein, and malignant thrombus is inferred from direct continuity of the filling defect. Diagnosis of direct invasion of the wall of the IVC by malignant invasion is also unreliable.

When considered overall, and taking into account detection of lymph node infiltration and venous invasion, the sensitivity of CT in distinguishing between Robson's stage I and III is 88–95 % and the specificity is 99–100 % [29, 30, 35].

The diagnosis of *stage IV disease* (T4a or b; M1) can be achieved accurately with CT. Direct spread into adjacent muscles, and detection of disease in the contralateral kidney or adrenal are well demonstrated. Adrenal metastases may be the only evidence of stage IV disease and occasionally confusion can arise between incidental benign adrenal cortical adenomas and deposits. Metastases are usually larger than 3 cm in diameter, of soft tissue density, inhomogeneous in texture and enhance patchily after intravenous injection of contrast medium (Fig. 7). Adrenal adenomas tend to be less than 3 cm in diameter, of low density, approaching that of fluid, and enhance only slightly after intravenous injection of contrast medium. The contralateral kidney is involved with a synchronous tumour in about 2 % of cases. CT is also a sensitive technique for the detection of haematogenous spread to the liver and lung. Liver metastases tend to be vascular and enhance after intravenous contrast medium injection. Invasion of the IVC can result in the appearance of a Budd-Chiari syndrome which can cause confusion in detecting metastases (Fig. 8). Ac-

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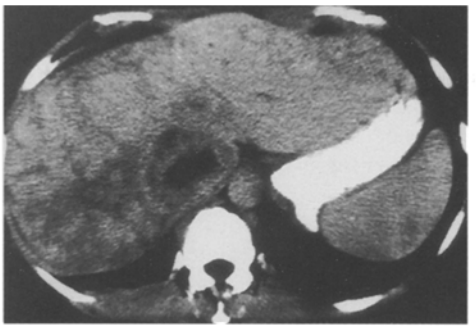

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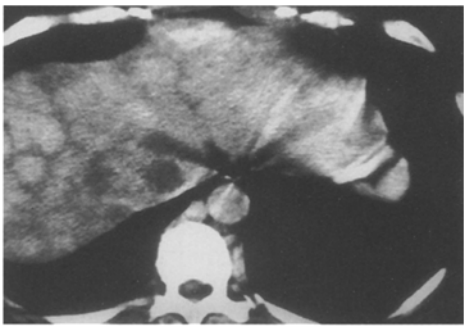

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Fig. 7. Stage IV renal cell carcinoma. Postcontrast CT scan showing a right-sided renal cell carcinoma extending directly into the IVC. There is a left-sided adrenal metastasis *(arrow)*

Fig. 8 a, b. Budd-Chiari syndrome resulting from invasion of IVC by tumour from right renal carcinoma. **a** Postcontrast CT scan with tumour thrombus within the IVC *(arrow)*. There is an inhomogeneous enhancement pattern of the liver with areas of marked enhancement and areas without enhancement. Note the relative sparing of the left lobe. **b** Postcontrast CT scan, at a level slightly superior to **a**, showing similar enhancement pattern and thrombus within the IVC extending above the level of the hepatic veins

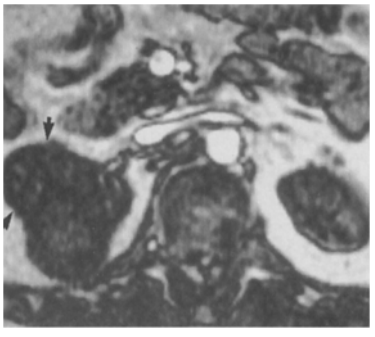

Fig. 9. Low flip angle gradient-recalled echo (30° flip angle, TR = 9.0, TE = 2.5) MRI showing the patency of the IVC in a patient with a right upper pole renal carcinoma *(arrows)*

curate staging of lung disease is of great importance as complete surgical resection of a solitary metastasis can increase the 5-year survival rate from 32% to 58% [21]. Thus, if the chest radiograph is clear or shows a solitary metastasis at the time of presentation, a staging investigation should include a CT scan.

The sensitivity and specificity for CT in the overall staging of stage IV renal cell carcinoma are 98% and 99%, respectively [29, 30, 35].

Magnetic resonance imaging

Advantages of MRI in staging renal cell cancer include direct imaging in sagittal and coronal planes (Fig. 6d), particularly in allowing the extent of IVC thrombus to be clearly demonstrated. Another advantage is that intravascular contrast material is not required for the evaluation of vascular structures as soft tissue masses within the venous system are easily distinguished from flowing blood [44]. This is of particular value for patients at risk of reactions to iodinated contrast medium. A further relative advantage of MRI over CT is its improved contrast. MRI does, however, have some disadvantages when compared with other imaging techniques: it is more expensive, the examination time is usually longer than that for CT, there is a lack of a universally accepted bowel contrast agent, and in many countries it is less readily available.

The technique for scanning patients will vary widely. Most institutions will perform standard T1- and T2-weighted sequences, with the application of presaturation pulses above and below the volume of interest to reduce flow-related artefact, and respiratory compensation. Fat suppression techniques are increasingly being used on T2-weighted sequences, particularly to increase the conspicuity by reducing chemical shift artefact and by reducing the dynamic range of abdominal signal intensities. Fast spin-echo T2-weighted sequences should beused as they improve spatial resolution and also decrease motion-induced artefacts. Gradient-recalled echo (GRE) sequences which produce a high signal in areas of flow are useful in evaluating venous structures (Fig. 9). Axial images are standard; sagittal and coronal images are useful in evaluating the venous system. In addition, cardiac gating can be used for assessing tumour thrombus extension into the upper IVC and the heart [45].

MRI is faced with the same limitations as CT in identifying early *extension into the perinephric space,* and although it appears slightly more sensitive it is equally non-specific in distinguishing between stage I and stage II disease (T1–T2 vs T3a). Thus, tumour extension into the perinephric space is recognized as strands of low signal intensity and intermediate signal intensity on T1-weighted and T2-weighted images, respectively. Detection of perinephric invasion can be improved by using fat-suppressed contrast-enhanced images, where enhancement of previously low signal intensity areas in perinephric tissue is indicative of intrarenal tumour extension [45–47]. As with CT, an inability to detect microscopic extension into the perinephric space reduces the sensitivity to 60–70% [29, 35, 48, 49]. The specificity is substantially higher, at 94% [27, 35, 48, 49].

As with CT or US, the only criterion for the diagnosis of *lymph node infiltration* on MRI is an increase in size. The sensitivity and specificity of MRI for N-staging in renal cell carcinoma therefore do not exceed those of CT [45, 50, 51]. MRI does have the disadvantage when compared with CT of the unavailability of a universally

acceptable bowel contrast agent which, in thin patients particularly, can cause bowel to be confused with lymph nodes. On the other hand, MRI can be useful in distinguishing between large collateral vessels and lymph nodes when the CT findings are uncertain. The signal void of flowing blood on spin-echo sequences will usually make the distinction accurately [45].

The most significant role of MRI in staging renal cell carcinoma, however, is its accuracy in assessing *venous involvement.* Spin-echo sequences have shown very high accuracies in making this assessment. However, use of limited flip angle, GRE techniques facilitates the imaging of vascular structures [52] (Fig. 9) and their application in renal cell carcinoma has allowed accuracies of 100 % in assessing vena caval invasion, 88 % for renal vein thrombosis detection and 80 % for atrial infiltration [52, 53]. Other potential advantages of MRI over CT include the ability to discriminate between malignant (tumour) thrombus and bland thrombus [20]. This, together with a reported ability to detect invasion of the IVC wall with MRI [54], has not been confirmed by other larger series, but would be of great value in managing patients. MR venography can be used to study venous thrombosis within the abdomen, including the renal veins and IVC, in renal cell carcinoma [55].

Whatever the technique used, MRI appears to be the best modality for detecting venous involvement. The accuracy is extremely high and negative predictive values for vascular invasion can be as high as 98 % or 99 % [27, 52, 53].

The accuracy for *visceral invasion* has been reported to be between 97 % and 98 % [29, 48, 53], as the excellent contrast resolution of MRI allows assessment of the spread of stage IV tumours to neighbouring organs within the abdomen. MRI, by using chemical shift imaging, is of particular value in distinguishing between incidentally detected benign cortical adenomata and deposits of the contralateral adrenal gland [56, 57].

Ultrasonography

Although US is extensively used in the detection of renal cell carcinoma, it is less accurate than CT or MRI in the overall staging of tumours [58, 59]. It cannot usually distinguish accurately between Robson's stage I and II (T1–T2 vs T3a) tumours as it cannot accurately distinguish the planes between the perinephric fat and the renal fascia [25].

In 50 % of patients the retroperitoneal region may not be adequately visualized and so the lymph node infiltration, renal vessels and infrahepatic vena caval invasion will not be detected [24, 25]. However, US is extremely accurate in identifying tumour extension into the intrahepatic vena cava and right atrium (Fig. 6 e). Thus, although the sensitivity of US for detecting thrombus in the entire IVC (including the infrahepatic portion) or renal vein may be as low as 54 %, its sensitivity for detecting the clinically important tumour thrombus that extends into the intrahepatic vena cava can be as high as 100 % [59–61].

US is also unable to detect muscle invasion reliably, so that stage IV tumours may be incorrectly assessed [21].

For all of these reasons, the overall accuracy of US in staging renal cell carcinoma is as low as 50–70 % [24, 29], and it is therefore seldom used as the sole staging technique. Nevertheless, if local surgical practice requires only a knowledge of venous invasion, and a complete and technically satisfactory US examination of the venous structures is obtained, then no further examination is required.

Conclusion

The survival rate of renal cell carcinoma has changed little over the years. Detailed epidemiological studies are still being conducted into those factors that most influence its prognosis and also into the most appropriate management. Modern non-invasive imaging has become extremely important in determining the form of surgery undertaken. The difficult problem of distinguishing between stage I (T1) and stage II (T3a) disease remains unresolved. This may become increasingly important in the management of a specific group of patients in whom partial nephrectomy is planned. At present MRI and spiral contrast-enhanced CT have similar accuracies in detecting lymph node and venous invasion. The choice between these techniques will depend on several factors apart from accuracy, such as availability and cost. Currently CT is the most widely used technique, with MRI kept in reserve to solve particular problems.

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