Optimising contrast enhancement in abdominal CT

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Dr Peng Hui Lee
Consultant Radiologist
Mid Essex Hospitals
Most abdominal CT scans are carried out with intravenous contrast and, in order to get the best out of fast multislice scanners, it is important to understand the general principles of contrast enhancement so that contrast protocols can be optimised. This article will review some of these principles as applied to the abdomen and describe how intravenous contrast enhancement can be optimised.

It is useful to think of contrast enhancement in the abdomen in terms of vascular enhancement that takes place in blood vessels and in well-perfused organs such as the pancreas, and parenchymal organ enhancement that occurs, for example, during the portal venous phase in the liver. These two types of enhancement are governed by different kinetics. Vascular enhancement is proportional to the rate of iodine delivery, which depends on injection rate and contrast strength. Arterial enhancement is improved with high injection rates (4 to 5 ml/s) and high strength contrast (350 – 400 mg/ml of iodine). It increases cumulatively with increased duration of injection, so that a longer duration of injection results in better enhancement. Arterial enhancement is also inversely related to cardiac output. There are wide variations in the time it takes for the contrast bolus to arrive in the aorta, ranging from as little as eight seconds to as much as 40 seconds in patients with cardiac disease, so the use of bolus tracking is essential to ensure that imaging is carried out at the right time. Parenchymal enhancement, as typified by enhancement of the liver in the portal venous phase, increases with the amount of iodine administered and is inversely related to patient weight. It is relatively independent of injection rate.

**Contrast enhancement in the liver**

The most important phases in CT of the liver are the late arterial phase, which occurs 10-15 seconds after arrival of contrast in the aorta, during which hypervascular lesions are most conspicuous, and the portal venous phase, about 50 seconds after contrast arrival in the liver when there is maximal enhancement of the liver parenchyma. The portal venous phase will be discussed first.

Opacification of the liver parenchyma occurs mainly as a result of distribution of contrast medium into the extracellular interstitial space, rather than from opacification of blood vessels. The liver has a dual blood supply, with about 80% of hepatic blood flow coming from the portal system and the remainder from the systemic arteries. This results in broadening of the contrast bolus and a delay in the arrival of contrast to the liver. Peak enhancement of normal liver parenchyma occurs during the portal venous phase, 40 to 50 seconds after arterial enhancement, or about 70 seconds after initiating the contrast injection, and is relatively prolonged. Contrast between normal liver parenchyma and most solid liver lesions, which are generally hypodense, is generally adequate as long as contrast medium has not diffused into the interstitial spaces of these lesions, making them potentially isodense. Because of this broadening of arterial contrast enhancement, timing in the portal venous phase is less critical than with arterial enhancement. Although bolus tracking can be used, this is not essential and a fixed delay of about 70 seconds generally works well.

Contrast enhancement of normal liver parenchyma is independent of injection rate, is directly proportional to the total amount of iodine delivered and is related to the size of the patient. 500-600 mg of iodine per kilogram of body weight is generally regarded as being necessary to provide an adequate level of hepatic parenchymal enhancement. Anecdotally, injection protocols using 100 ml of 300 mg/ml contrast are fairly common in the UK, which equates to 500 mg/kg for a patient weighing 60 kg (9 stone 6.3 pounds). Some studies suggest that this injection protocol might not produce adequate hepatic enhancement in larger patients.

Several studies have shown more consistent liver opacification when contrast is administered according to body weight compared with when a fixed dose of contrast is given to all patients. Larger contrast volumes are necessary to provide adequate hepatic enhancement in larger patients. Conversely, in smaller patients less contrast is necessary to achieve an acceptable level of enhancement, and reducing contrast volumes may reduce the risk of nephrotoxicity which is dose dependent. Ho et al found that calculating the contrast dose on the basis of lean body weight (rather than total body weight) resulted in better uniformity of hepatic enhancement between patients, and Kondo et al found that the iodine dose was optimised when calculated according to body fat percentage. Evaluating body fat percentage might be impractical in a busy department, but enthusiasts should note that domestic weighing scales which automatically compute percentage body fat using bioelectric impedance analysis are available for as little as £50.

While the majority of solid liver lesions are hypovascular and are most conspicuous in the portal venous phase, a smaller proportion of liver lesions are hypervascular. These lesions are supplied via the systemic arterial system rather than by the portal system and their enhancement kinetics are of the vascular type. Their enhancement is improved by high flow rates and high strength contrast, and bolus tracking is essential. Examples of such lesions include many hepatocellular carcinomas and some metastases in patients with breast cancer, renal cell carcinoma, endocrine tumours, carcinoids and melanoma. They enhance maximally in the late arterial phase, 10-15 seconds after contrast arrival in the aorta. During this time the portal veins are slightly opacified and this phase is therefore sometimes called the portal venous inflow phase.

There is no consensus as to whether late arterial phase imaging should be carried out in the staging or follow-up of patients with malignancies where hypervascular liver metastases occur. Some authors suggest that dual phase hepatic imaging is only routinely necessary at initial staging, while the guidelines of the Royal College of Radiologists advise that dual phase imaging is not routinely necessary in patients with renal cell carcinoma, breast cancer, melanomas or sarcomas, as it is unlikely to influence outcomes. Delayed imaging in the liver is useful in some circumstances. Images acquired around three minutes post-injection may aid the detection of well differentiated hepatocellular carcinomas and demonstrate retention of contrast in haemangiomatas, and images acquired at 10-15 minutes post-injection may demonstrate retention of contrast in cholangiocarcinomas.

**Pancreas**

Enhancement of the pancreas follows arterial dynamics. In order to achieve maximal pancreatic enhancement and to maximise the conspicuity of hypovascular lesions, such as most pancreatic adenocarcinomas, imaging should be carried out during the pancreatic phase, about 20-25 seconds after arrival of contrast in the aorta, using a high injection rate, high strength contrast, and bolus tracking. While hypervascular lesions are known or suspected (eg endocrine tumours), a delay of 10-15 seconds after aortic opacification is recommended. Staging of pancreatic tumours includes pancreatic phase imaging for assessment of pancreatic tumour, pancreatic duct and arterial encasement, and portal phase imaging for assessment of venous involvement and hypovascular liver lesions.

**Kidneys**

Four phases of contrast enhancement are described in the kidney. The arterial phase occurs about 15-25 seconds after...
contrast injection when there is marked enhancement of the renal arteries. The corticomedullary phase occurs around 30-40 seconds after injection and is characterised by marked cortical enhancement and relatively less enhancement of the medulla. During this phase there is maximal enhancement of the renal veins. The nephrographic phase occurs around 80-120 seconds after injection and is characterised by uniform parenchymal opacification, and it is therefore the best phase for detection of subtle parenchymal masses. The excretory phase begins around 180 seconds after injection when the pelvicalyceal systems and ureters are opacified. Non-contrast images are also important in imaging the kidney in order to assess calcification and as a baseline for measuring enhancement when characterising lesions.

Renal masses are usually detected by ultrasound and most solid masses are regarded as suspicious. CT is generally carried out for staging and characterisation. Most solid masses are renal cell carcinomas that enhance brightly, so a biphasic examination with images acquired in the corticomedullary and nephrographic phases is optimal for characterisation and staging. The corticomedullary phase will demonstrate the enhancing mass, facilitate evaluation of the renal vein and also help detect hypervascular liver lesions, while the nephrographic phase is most sensitive for detecting renal masses and for detecting most liver metastases.

Where there is uncertainty about the nature of the renal mass, excretory phase imaging may be helpful in identifying upper tract transitional cell tumours. When a small hyper-echoic lesion is detected on ultrasound with a high prior probability of it being an angiomyolipoma, non-contrast CT may suffice for characterisation by confirming the presence of fat in the lesion. Early arterial phase images are sometimes used for arterial mapping in patients undergoing laparoscopic nephrectomy, but the arteries are usually well visualised in the corticomedullary phase.

**Bladder**

Using 4-slice CT, Kim et al found that bladder cancers enhanced best 60 to 80 seconds after contrast injection, with peak enhancement occurring at 60 seconds. Enhancement at 40 seconds and 100 seconds was significantly poorer.

Imaging the full bladder during portal phase imaging of the abdomen and pelvis should provide good quality images for local staging. The addition of an excretory phase allows evaluation of the upper tracts for synchronous transitional cell tumours.

**Small bowel**

CT enterography is a technique for evaluating the small bowel. The small bowel is distended with a large volume (1-1.5 litres) of water or other neutral oral contrast agent ingested prior to contrast enhanced CT. With CT enteroclysis, the low attenuation small bowel contrast is administered via jejunal intubation. The contrast enhanced bowel stands out against the low density lumen, enabling detection of inflammatory changes and other pathology. There is no consensus regarding the optimal timing for CT enterography. Schindera et al found that peak mural enhancement occurred at 50 seconds after injection. However, Vandenbroucke et al found no difference in the detection of Crohn’s Disease between patients scanned at 40 seconds and those scanned at 70 seconds after injection.

**Conclusion**

Optimising contrast enhancement is one of the key factors in getting the best out of your CT scanner. In some areas of the abdomen, enhancement kinetics are well understood. Enhancement of the pancreas and hypervascular liver lesions follows arterial kinetics and is optimised with high flow rates, high strength contrast and bolus tracking. Enhancement of the liver in the portal venous phase is relatively independent of injection rate, and bolus tracking is not essential, but an adequate amount of iodine is required and this should be adjusted according to the size of the patient. In other areas of the abdomen, such as the bowel and bladder, contrast injection strategies have been investigated empirically and documented. For general abdominal scanning, imaging in the portal venous phase will provide good enhancement for most structures, but where there are specific areas that are of interest, the delivery of contrast should be tailored accordingly. The use of appropriate contrast protocols will help to ensure consistently good image quality.

<table>
<thead>
<tr>
<th>Organ</th>
<th>Enhancement phase</th>
<th>Timing (A + time of arrival of contrast in aorta)</th>
<th>Indication/role</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Liver</strong></td>
<td>Early arterial</td>
<td>A + 8s</td>
<td>Arterial mapping</td>
</tr>
<tr>
<td></td>
<td>LAP</td>
<td>A + 16s</td>
<td>Assessment of hypervascular lesions</td>
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<td></td>
<td>PVP</td>
<td>A + 50s, or 70s fixed delay</td>
<td>Assessment of hypovascular lesions</td>
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<tr>
<td></td>
<td>Delayed equilibrium</td>
<td>3 min</td>
<td>Detection of well differentiated HCC. Contrast retention in haemangiomas</td>
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<td></td>
<td>Delayed post equilibrium</td>
<td>10-15 min</td>
<td>Delayed contrast retention in cholangiocarcinoma</td>
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<tr>
<td><strong>Pancreas</strong></td>
<td>Non-contrast</td>
<td>-</td>
<td>Localise pancreas, detect calcification</td>
</tr>
<tr>
<td></td>
<td>Arterial</td>
<td>A +8s</td>
<td>For suspected hypervascular lesions, eg neuroendocrine tumours</td>
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<td></td>
<td>Pancreatic</td>
<td>A + 25s</td>
<td>Assessment of pancreatic tumour, pancreatic duct and arterial encasement</td>
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<td></td>
<td>Portal venous</td>
<td>A + 50s</td>
<td>Assessment of venous involvement and hypovascular liver lesions</td>
</tr>
<tr>
<td><strong>Kidney</strong></td>
<td>Non-contrast</td>
<td>-</td>
<td>Detect calcification, baseline to measure lesion enhancement, identify fat in lesion (angiomyolipoma)</td>
</tr>
<tr>
<td></td>
<td>Arterial</td>
<td>15-25s + A+8s</td>
<td>Arterial mapping</td>
</tr>
<tr>
<td></td>
<td>Nephrographic</td>
<td>80-100s</td>
<td>Detection of focal renal lesions. Evaluation of IVC. Detection of hypovascular liver metastases</td>
</tr>
<tr>
<td></td>
<td>Excretory</td>
<td>180s or more</td>
<td>Demonstration of collecting systems and urothelial lesions</td>
</tr>
<tr>
<td><strong>Bladder</strong></td>
<td>60-80s</td>
<td>-</td>
<td>Peak contrast enhancement of tumour</td>
</tr>
<tr>
<td><strong>Small bowel</strong></td>
<td>40-70 s</td>
<td>-</td>
<td>Bowel wall enhancement; detection of inflammatory disease</td>
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</table>
References


