Renal Angiogram

Background

James Ritchie and Lance Dworkin

Key point: contrast angiography remains the ‘gold standard’ for the investigation of atherosclerotic renovascular disease (ARVD). This can occur in the native or transplant kidney.

Renal angiography is not the first line investigation for ARVD. Because of its invasive nature, with attendant risks, indirect techniques such as CTA, MRA, duplex US are first line diagnostic techniques for ARVD. Rountas (2007) has reviewed the sensitivity and specificity of these techniques.

In the native kidney, less than 50% narrowing does not normally lead to renal hypoperfusion. Even then, renal oxygenation is preserved (Gloviczki, 2010); which is also surprising. It is not known whether the transplant kidney is similarly robust. Because of the importance of maximising transplant function, lower levels of arterial narrowing are usually looked for, and treated.

The conventional angiogram is superior to digital angiography but requires higher doses of contrast medium. Digital subtraction angiography (DSA) requires computer reconstruction to generate images.

It has several significant limitations:

1. Provides only 2D images
2. No functional information. Although conventional angiography can detect the anatomical presence of a renal artery lesion, it does not provide data on its physiological or haemodynamic significance
3. Invasive technique, with significant Complications (discussed later)
4. Contrast nephropathy

Contrast nephropathy is the third leading cause of hospital acquired renal failure and is associated with significant morbidity and mortality.

Dworkin and Cooper published an excellent review article in 2009.

Indications for Angiography

This is an area of considerable debate that will be covered in the chapter on ARVD. Most would agree that the following are indications for angiography:

1. Key point: to confirm the presence of ARVD in the native (or transplant) kidney at the time of a revascularisation procedure. Angiography is normally only performed when there is an established diagnosis of ARVD and a clear indication for revascularisation.
2. Unexplained macroscopic haematuria, thought to be due to arteriovenous malformation.

General Points (should we be looking for ARVD?)

Most of this chapter concerns patients with atherosclerotic RVD. Patients with fibromuscular dysplasia are younger and healthier generally and the complication rate is lower.

Also. There is considerable debate of whether we should be looking for atherosclerotic RVD at all. Based on the current literature, most practitioners, including sensible interventionists, would agree that there is no reason to be looking for ARVD in medically stable patients. These investigations should be reserved for patients that are failing medical therapy.

Although not established as beneficial for any group, it is still rational to consider diagnosing and treating ARVD in patients that have:

1. uncontrolled hypertension despite a maximal medical regimen (emphasis on maximal)
2. declining kidney function (but not those with stable function)
3. (perhaps) severe CHF with recurrent hospitalisations (but not those with stable CHF, or those with CHF but not on a good medical regimen)
4. ‘flash’ pulmonary oedema

Renal Angiogram (showing a proximal RAS)
Normal Anatomy and Variations

The kidneys are paired retroperitoneal structures that are normally located between the transverse processes of T12-L3 vertebrae, with the left kidney superior in position to the right. The upper poles are normally oriented more medially and posteriorly than the lower poles.

The kidneys are bean-shaped structures. In one study of 125 kidney donors (Kang, 2007), after nephrectomy, normal adult kidneys were measured at 11.1 +/- 1.0 cm long, 6.3 +/- 0.7 cm wide, 4.7 +/- 0.7 cm thick, and weighed 196 +/- 41 g. Under 10 cm in length is considered “small”; except in small people, when <9 cm may be the upper limit of normal length.

99.9% of the population have two kidneys. In 70% there is one renal artery supplying each kidney. The renal arteries are usually 4-6 cm in length and 5-6 mm in diameter. They typically arise from the aorta at the level of L1–L2 intervertebral disc space below the origin of the superior mesenteric artery (SMA). Both usually course in a slightly posterior direction due to the anatomic orientation of the kidneys:

Renal Artery and Vein In Situ

The right renal artery orifice is located on the anterolateral wall of the aorta, and is longer and courses downwards toward the right kidney behind the inferior vena cava (IVC). The left renal artery originates from a more posterolateral location, arises a little higher than the right, and has a more horizontal, upward orientation posterior to the left renal vein.

Each renal artery supplies the inferior adrenal artery. The inferior adrenal arteries arise directly from the proximal renal artery in two thirds of people and they may be solitary or multiple.

Each artery divides into 5 segmental end arteries before entering the hilum into renal sinus: superior, inferior, posterior, anterior superior and anterior inferior. The segmental arteries then course through the renal sinus and branch into the lobar arteries giving one branch to each pyramid. Further divisions include the interlobar, arcuate, and
interlobular arteries.

There is also considerable variation in the number and distribution of the segmental arteries.

Key point: 0.1% of humans are born with unilateral renal agenesis (a single kidney) (Woolf, 2007).

Renal agenesis. IVP showing right renal agenesis (or dysplasia). Right hip dysplasia is also shown.

Renal artery variations

Anatomical variations of the renal arteries are common in general population with different frequencies in different racial groups. These variations are becoming more important in view of the increasing number of interventional radiological procedures, as well as urological and vascular operations, and transplantation.

Accessory renal arteries constitute the most common and clinically important renal arterial variations and can be seen in up to one-third of the normal population. Accessory arteries usually arise from the aorta or iliac arteries at any level between T11 and L4.

Typically, the accessory renal artery courses into the renal hilum to perfuse the upper or lower renal poles. They may also enter the renal parenchyma directly from the renal cortex (called a polar artery). Rarely, they may arise from the lower thoracic aorta, as well as from the lumbar and mesenteric arteries.

Also rarely, renal arteries may originate from the more proximal portion of the abdominal aorta above the origin of the SMA. Furthermore, aberrant renal arteries may originate from the iliac arteries, especially in ectopic kidneys.

In a horseshoe kidney, the main renal arteries develop normally. However, the mesonephric and metanephric arteries often persist to supply the upper and lower poles, respectively. These primitive arteries may arise at different levels in the aorta and iliac arteries.

CT abdomen show fused kidneys, with a parenchymal isthmus at the lower poles. Note the malrotated collecting system of the left kidney, facing anterolaterally.

IVP demonstrates horseshoe kidney. Note the malrotated collecting systems on both sides. The lower pole calyx of the right kidney lies medial to the ureter.

Key point: the number of renal arteries is highly variable; multiple arteries supplying a single kidney are seen in 30% of patients.
Technique

Evaluation of the renal arteries usually begins with an aortic injection of contrast material. A 5F multiple side-hole catheter is placed into the aorta through a femoral or brachial artery approach using a modified Seldinger technique.

Contrast material is injected with an automatic power injector while sequential images are recorded. The rate and volume of contrast injection and the rate of image acquisition are determined and specific for each patient.

A contrast injection of 10 to 20 mL/sec for 2 seconds is usually adequate. Images usually are acquired at a rate of three per second for 3 to 5 seconds, followed by a rate of one to two images per second for the next 5 seconds. These images allow assessment of the arterial, nephrographic, and occasionally the venous phases of flow.

For optimal visualisation of the renal arteries, attention to several technical details is required. The catheter should be positioned with the side holes at the level of the renal arteries, usually between the first and second lumbar vertebrae. Ideally the catheter should be positioned low enough to avoid filling of the superior mesenteric artery and its branches, which may overlap and obscure the renal arteries.

Contrast Material

Intravascular administration of contrast material is an established risk factor for the development of AKI, or AKI on CKD. Factors that appear to increase the risk of renal dysfunction include use of high volumes of contrast material and use of high-osmolar contrast.

The debate over the possible benefits of low-osmolar vs iso-osmolar agents has been 'settled'. Meta-analyses have failed to identify a difference in outcome between the two classes of contrast (Reed, 2009). Though RCTs such as CARE, PREDICT and VALOR suggest the iso-osmolar agents may have a benefit in diabetic patients.

Other radiocontrasts are used by some centres (CO2 for example) to avoid radiocontrast and reduce risk.

Other Techniques

Also, in most centres prior to intervention, after the aortogram, selective injections of all renal arteries is performed. But the aortogram should not be skipped and only selective angiography performed to ensure that all renal arteries are identified. In some centres, pressure gradients across the stenotic lesions are routinely measured. Some believe that this helps to identify patients with haemodynamically significant narrowings.

Complications

General Points

Many authors report complication rates from studies in which not only angiography, but also angioplasty and renal artery stenting are performed. In other words, it is important to realise that the complication rates described are for angiography combined with a percutaneous intervention.

Therefore the discussion below emphasises the negative outcomes, which are real. However, most patients experience transient elevations in serum creatinine that resolve spontaneously.

Furthermore, MRA and CTA, which may be used as screening studies, also have complications. CIN can occur with CTA, during which more contrast is typically needed than in renal angiography. The risk of nephrogenic systemic fibrosis from gadolinium administration for MRA in patients with GFR <40 mls/min should also be noted. This can be a life-threatening complication.

Complications in Previous Eras

In the Cooperative Study of Renovascular Hypertension, 2,719 renal angiograms were performed (Reiss, 1972). Initial studies were done in 2,374 patients, and 345 studies were done on a follow-up basis. The transfemoral catheter technique was used in 77% of the cases. In this study, the mortality was low (0.11%), with a non-fatal major complication rate of 1.2%. Most of the major complications involved haemorrhage, thrombosis, and renal injury.

Serious Complications in the Modern Era

Now, given that direct angiography is rarely used as a diagnostic tool for RVD and is mainly used during revascularisation, these figures may under-represent patient risk. In ASTRAL (2009) serious adverse events were reported in 6.8% of patients following revascularisation, and in STAR (Bax, 2009), 3 of the 46 revascularization procedures
Key point: if associated with an intervention, angiography has a serious major complication rate of approximately 10%, and a mortality of 1% (0.1% without an intervention).

The risks of renal angiography are significant and may include complications related to the arterial puncture (e.g., haematoma, pseudoaneurysm), contrast-induced nephropathy (CIN), and cholesterol embolisation. Janes (2010) has pointed out that AKI following coronary angiography is associated with a sustained loss of renal function, and a future decline in function.

There are strong associations between coronary artery/portal vascular disease and asymptomatic ARVD (30-40% respectively). As such, many ARVD patients are represented in reports of coronary angiography (whether they or the operator knows it). Also, large RCTs such as ASTRAL (2009) (and to a lesser extent, STAR, 2009) give an indication of the complication rates which can be expected following angiography in a CKD population with high burden of vascular disease (e.g., 11% groin haemorrhage/haematoma with 1 to 30 days of procedure in ASTRAL).

The complications of angiography are often insidious and difficult to assess accurately, especially retrospectively. Probably the two most important complications of renal angiography are contrast-induced nephropathy (CIN) and cholesterol embolisation, covered in the next section.

Contrast Nephropathy and Cholesterol Embolisation

Contrast Nephropathy

Definition

The most commonly used clinical definition of renal impairment in CIN is a relative rise in serum creatinine of >25% from the baseline value (or an absolute increase 50 mcmol/l), within 48 h after the administration of CM. Renal failure, which is typically non-oliguric, usually peaks within 3 to 5 days. In patients without risk factors the incidence is 2%, and acute CIN is usually transient.

Epidemiology

But the incidence of CIN reported in the literature varies markedly depending on the definition of CIN used, the type of procedure performed, the volume and type of contrast agent, patient risk factors (most notably pre-existing renal impairment and diabetes) and the length of follow-up. McCullough (1997), in a study of 1826 consecutive patients, reported an overall incidence of 14.5% in patients undergoing coronary interventions.

Such studies predate the use of low-osmolar contrast agents and formal pre/post-angiography hydration. Data since would suggest rates are somewhat lower e.g., 11% incidence of CIN with both low and iso-osmolar agents in meta-analysis. 10% of patients within ASTRAL (2009), who were higher risk, having an increase in sCr following angioplasty and stenting.

In patients with mild-to-moderate renal impairment and diabetes, the incidence of CIN is reported in the range of 10-50% and in patients with diabetic nephropathy, CIN can occur in 50-90% of patients (Lamiere, 2006). This figure of ’50-90%’ of CIN in diabetic CKD patients is based both on varying definitions and data preceding 1980. In a fairly recent review, Richard Solomon (who publishes a great deal in this area), hedged his bets, and said “the incidence of CIN in diabetics is generally 50-100% higher than in non-diabetics” (Solomon, 2006).

In patients with advanced renal failure, particularly diabetic patients with nephropathy, renal function may not recover following CIN and chronic dialysis may be required. The mechanism of CIN is uncertain but includes: altered rheological properties, perturbation of renal haemodynamics, regional hypoxia, auto- and paracrine factors (adenosine, endothelin, reactive oxygen species), and direct cytotoxic effects (Persson, 2006).

Sadeghi et al. (2003) found that patients with CIN had a significantly greater 1-year mortality rate (23.3%) compared with those who did not develop CIN (3.2%) and the relative risk of 1-year mortality after CIN was 7.4.

Risk of Dialysis

Cochran (1983) published a review of 216 patients who had a renal angiogram. Forty-five (16.9%) had an significant increase in serum level of creatinine, six developed oliguria or anuria, and one required permanent dialysis. Age, proteinuria, abnormal baseline serum creatinine, use of a particular contrast medium, and pre-existing renal disease were independent risk factors for worsening of renal function. For example, patients with underlying renal disease were 6.6 times more likely to develop a transient increase in creatinine than those with no renal disease.

Finn (2005) has reviewed the renal and clinical consequences of CIN. Mehran (2004), in a study of 8357 patients, found that the risk of dialysis after coronary angiography, was very variable according to the patients’ risk factors: from 0.04% in low risk patients to 12.6% in high risk patients. Patients who experience CIN also have reduced medium and long-term survival.

McCullough also assessed the in-hospital mortality rates for patients who developed CIN requiring dialysis, of which there were 14. The in-hospital mortality rate for these patients was 35.7%.

Risk Factors and Predicting Probability of CIN

Mehran (2004) has also developed a clinical prediction rule to estimate probability of nephropathy (= increase ≥ 25% in serum creatinine at 48 hours):

Risk Factors

- Systolic blood pressure < 80 mm Hg - 5 points
- Intra arterial balloon pump - 5 points
- Congestive heart failure (Class III-IV or history of pulmonary oedema) - 5 points
- Age > 75 y - 4 points
- Haematocrit level (< 39% for men and < 35% for women) - 3 points
- Diabetes - 3 points
- Contrast media volume - 1 point for each 100 mL
- Serum creatinine level > 150 mcmol/L - 4 points

Serum creatinine level >150 mcmol/L - 4 points

Leertouwer, 2000

ASTRAL, 2009

STAR, 2009
Cholesterol emboli often present as AKI. There may or may not be signs in hands and feet ('trash feet', blue toe syndrome, digital gangrene), as above.

Management

There is no cure for CIN and treatment options are limited to supportive care. Because the risk factors for CIN are common and the consequences of this disorder can be serious, it is important for physicians using angiography to incorporate preventive strategies into clinical practice.

Prevention

Choice of contrast agent

The osmolality of the contrast agent was previously believed to be of paramount importance in contrast-induced nephropathy (CIN). It is now increasingly clear that other physico-chemical properties play a greater role, such as viscosity. Attention should be paid to use contrast agents of low viscosity. Moreover, sufficient fluids should be supplied to limit fluid viscosity of urine.

Early studies suggested that iso-osmolar, non-ionic contrast media may be superior to others. However, a subsequent large RCT (Rudnick, 1995) failed to confirm this observation. Solomon (2005) has published a review of the literature. Nonetheless, modern iodinated contrast agents are non-ionic; the older ionic types caused more adverse effects and are now rarely used.

Hydration with or without bicarbonate

Hoste (2010) has reviewed the evidence for sodium bicarbonate, compared to normal saline, and found marginal benefits. Even if hydration with sodium bicarbonate decreases the incidence of CIN, it has not been shown to reduce the need for renal replacement therapy or in-hospital mortality. Additional confirmatory trials with sodium bicarbonate are needed because the largest trial to date showed no benefit of sodium bicarbonate over normal saline. The renoprotective effects of bicarbonate are thought to be due to urinary alkalisation, which creates an environment less amenable to the formation of harmful free radicals.

Weisbord (2008) has reviewed the use of volume expansion.

Key point: At present, it is recommended that either isotonic sodium chloride or sodium bicarbonate is given at a rate of 1 ml/kg per h for 12 h before and 12 h after radiocontrast administration in high-risk hospitalised patients and outpatients who are undergoing non-emergency procedures, including renal angiography - independent of type of radiocontrast or route of administration.

As well as volume expansion, other recommendations include: the use of the smallest possible dose of low- or iso-osmolar contrast media, stopping nephrotoxic drugs, and avoiding repeat contrast injections within 48 hours.

Methylxanthines

Adenosine antagonists, such as the methylxanthines theophylline and aminophylline, may help - although studies have conflicting results. The best studied dose is 200 mg of theophylline given 30 minutes before contrast administration.

N-acetylcysteine (NAC)

This is one of the most studied preventative agents. It is not without risk. In one study 15% of patients receiving NAC intravenously had allergic reactions. Its use is controversial.

In the large ACT (Acetylcysteine for Contrast-induced nephropathy Trial) in 2011, no protective effect of NAC was seen. In this study, 2308 patients undergoing an intravascular angiographic procedure with at least 1 risk factor for contrast-induced acute kidney injury (age >70 years, renal failure, diabetes mellitus, heart failure, or hypotension) were randomised to acetylcysteine 1200 mg or placebo. The incidence of contrast-induced acute kidney injury (primary endpoint) was 12.7% in the acetylcysteine group and 12.7% in the control group (p=0.97). A combined endpoint of mortality or need for dialysis at 30 days was also similar in both groups (2.2% and 2.3% respectively; p=0.92).

In a previous smaller but landmark study in 2000, Tepel and coworkers evaluated N-acetyl cysteine in a randomised, prospective, placebo controlled study comprising 83 patients with chronic statin pretreatment. Two percent of patients in the treatment arm developed a significant increase in serum creatinine versus 21% of the control patients. Several subsequent studies have validated the efficacy of N-acetyl cysteine. The mechanism of N-acetyl cysteine’s protective effect is poorly understood but may be related to increased nitric oxide production and decreased oxidative stress.

Prophylactic haemodialysis

Zwang (2011) has reviewed the literature. Among 6 cohort studies, 4 showed chronic statin pretreatment had a preventive effect against CIN. This was not confirmed by a meta-analysis of 6 RCTs, incorporating 1,194 patients. So current data are not conclusive as to whether statins are protective for CIN due to the inherent limitations of the included studies.

Other interventions

Other pharmacological agents, such as furosemide, mannitol, dopamine, and atrial natriuretic peptide have been tried; but have either not had beneficial effects, or had detrimental effects. Of course, limiting the total contrast volume reduces the incidence of contrast nephropathy. Metformin should be stopped in patients with diabetes and renal impairment who undergo renal angiography (Thomsen, 1999). Stopping ACEi or ARBs before the procedure does not affect the incidence of CIN (Rosenstock, 2008).

Kelly (2008) has reviewed drugs used in the prevention of CIN.

Cholesterol Embolisation

Cholesterol emboli often present as AKI. There may or may not be signs in hands and feet ('trash feet', blue toe syndrome, digital gangrene), as above.
Renal biopsy appearance are characteristic. This biopsy shows cholesterol clefts in a branch of a renal artery.

Background

It is sometimes called Cholesterol Embolisation Syndrome (CES). It is a multisystem atheroembolism involving multiple organs, caused by distal showering of cholesterol crystals from aortic atheromatous plaques; involving the kidneys in 75% of cases. If AKI is present, classical biopsy findings are found in 90-100% of cases. Other organs involved include spleen (55%), pancreas (50%), GI tract (30%), liver (20%), brain (15%), eyes and extremities.

Like contrast nephropathy, patients with widespread atheroma and pre-existing renal impairment are the patients who are most susceptible to atheroembolism. It can also occur spontaneously, especially in arteriopathies.

History

Cholesterol emboli were first recognised by the Danish pathologist Peter Ludvig Panum in 1862. Further evidence that eroded atheroma was the source of emboli came from American pathologist Curtis M. Flory, who in 1944 reported the phenomenon in 3.4% of a large autopsy series of older individuals with severe atherosclerosis of the aorta.

Since the 1960s, various investigators have reported cases of CES such as blue toe syndrome (see picture above) or AKI as a complication of angiography, major vessel surgery, or thrombolytic therapy. Ipsilateral RAS has been reported in up to 80% of patients with renal CES. In 1987, Fine et al described recognisable skin findings in patients with cholesterol embolism (eg livedo reticularis), thereby linking cholesterol embolism to conclusive, conspicuous signs and symptoms.

Definition

There is no accepted definition of CES. Fukomoto suggested one in 2003, based on a combination of classical clinical findings and presence/not of AKI.

Epidemiology

It is an increasingly recognised complication of renal angiography. Typically patients are 50-85 years, with known atheroma.

It tends to be underdiagnosed because it is an insidious disorder that is difficult to identify with certainty without performing a biopsy. Only the most fulminant cases are recognised clinically. Although clinical studies generally have reported a low incidence (1.4% in one large series; Fukomoto, 2003), autopsy studies found evidence of atheroemboli in 25% to 30% of patients who died within 6 months after cardiac catheterisation or aortography. For these reasons, the actual incidence of this syndrome remains uncertain.

CES is iatrogenic in approximately 75% majority of cases (Belenfant, 1999). The most common triggering events are angioplasty, vascular surgery (50 and 15% respectively) and long-term anticoagulant therapy. Fibrinolytic therapy is another reported aetiology. The atherosclerotic patient may also suffer spontaneous detachment of a plaque, or low grade, clinically silent migration of crystals from the aortic wall.

Mortality, if AKI is present, is high (70% to 90%).

Clinical Features

Clinical consequences of CES vary considerably, from being completely asymptomatic to presenting with acute multiorgan failure - including AKI or AKI/CKD (many patients have pre-existing CKD) - or cutaneous involvement, or both. 80% of patients have dermatological abnormalities, including 'blue-toe syndrome' ('trash feet'), digital gangrene and livedo reticularis. Fever, often low-grade, is characteristic.

Non-renal involvement can lead to a variety of clinical features. These include abdominal pain, nausea, vomiting, ileus, GI bleeding (from ischaemic bowel), hepatitis, angina and neurological deficits. When retinal CE occurs, refractile yellow deposits known as Hollenhorst plaques, may be seen at the bifurcation of retinal vessels on fundoscopic examination. The phenomenon is named after the American ophthalmologist, Robert Hollenhorst (1913–2008), who first described their significance in 1961 (Hollenhorst, 1961).

Diagnosis and Investigation

It is a clinical diagnosis in cases without AKI. Measuring the serum eosinophil count is useful, as this increases during the active phase of CES. A myriad of laboratory abnormalities indicative of tissue injury occur, including a raised ESR/CRP (95%), raised amylase (60%), leucocytosis (60%), hypocomplementaemia (25-70%) and elevated liver and muscle enzymes (60%).

TOE may be required to look for a mobile ulcerative plaque in the aorta.

Urinary findings are non-diagnostic, but may include mild proteinuria, microhaematuria, pyuria and eosinophiluria. In cases with AKI, renal biopsy usually confirms the diagnosis.
Differential Diagnosis

This includes contrast nephropathy and vasculitis. The frequent presence of eosinophilia and eosinophiluria, rash, fever and AKI, may lead to the misdiagnosis of acute interstitial nephritis.

Management

There is no known cure. Supportive management (eg dialysis) may be necessary. ESRF may ensue. Further endovascular procedures should be avoided. ACEi may be effective in managing labile hypertension. Corticosteroids have been used successfully to treat patients with CES and associated inflammatory symptoms. HMG CoA reductase inhibitors may also have a role.

CES has also been reported to occur in patients following anticoagulation. Whilst direct causality between the two has not been proven, the proposed mechanism is that anticoagulants prevent thrombus organisation in an ulcerative plaque. Therefore, anticoagulation is contra-indicated in the setting of CES unless a life-saving indication for anticoagulation exists. When dialysis is indicated, some physicians would favour peritoneal dialysis or heparin-free haemodialysis.

Prevention is the most effective management strategy. And the risk of CES (and CIN) should be part of the risk:benefit analysis when angiography (of any type) is being considered in a patient with known (or suspected) atheroma.

Summary

Top Tips: To prevent CIN in high-risk patients, give isotonic sodium chloride or sodium bicarbonate. Look out for Cholesterol Emboli

1. Duplex ultrasonography, CTA or MRA are preferred first line investigations for ARVD. Duplex US is the least invasive technique and very reliable when performed by experienced operators
2. Contrast angiography remains the ‘gold standard’ for investigation of atherosclerotic renovascular disease (ARVD) but carries some risk
3. The principle indication for angiography is to confirm the presence of ARVD in the native (or transplant) kidney at the time of a revascularisation procedure
4. 0.1% of humans are born with unilateral renal agenesis (a single kidney)
5. There is more than one renal artery in 30% of humans
6. Non-fatal serious major complication rate for angiography (plus angioplasty or stent) is approximately 10%, with a mortality of 1%. Risks of renal angiography without a procedure are significantly lower (mortality 0.1%)
7. Contrast-induced nephropathy (CIN) occurs in 5-10% of patients, with a very variable dialysis risk (0.5-10%)
8. To prevent CIN in high-risk hospitalised patients and outpatients, give isotonic sodium chloride or sodium bicarbonate at a rate of 1 ml/kg per h for 12 h before and 12 h after radiocontrast administration

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Richard Solomon is an authority on CIN


Guidelines


Websites

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CORAL trial: www.coralclinicaltrial.org


Fibromuscular Disease Society (recently begun to develop a registry service): www.fmdsa.org

UK NFK RAS: www.kidney.org.uk/Medial-info/kidney-disease/Ras.html
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