# Acute renal failure in cardiac surgical patients

Donald G. Vidt, M.D.

*Cleveland, Ohio*

The onset of acute renal failure following any surgical procedure has a poor prognosis due not only to the loss of renal function, but also to other major complications including sepsis, gastrointestinal bleeding, and cardiac or pulmonary complications.[1-3]

It is important that the clinician be able to recognize those risk factors that may be related to the development of postoperative renal failure if patient morbidity and mortality are to be minimized. In view of the lethal nature of acute renal failure, the most effective treatment is directed toward careful selection of patients and prevention.

## Definition of acute renal failure

Acute renal failure results when any process causes a rapid reduction in glomerular filtration rate (GFR), often to a level that requires dialysis support. Acute loss of renal function is reflected by a progressive rise in serum creatinine and urea nitrogen concentrations. Acute renal failure may be characterized by a striking reduction in urine volume to levels < 400 ml/dl (oliguria) or occasionally to volumes < 100 ml/dl (anuria). Although acute renal failure is characteristically accompanied by low urine flow rates, nonoliguric renal failure may occasionally be seen with normal or even increased

urine flow. Other common synonyms used for acute renal failure include acute tubular necrosis, vasomotor nephropathy, lower nephron nephrosis, or shock kidney.

## Cause of acute renal failure

The principle causes of acute renal failure are listed in *Table 1.* As will be noted, a large group of clinical problems may be associated with the syndrome of acute renal failure. In approaching the patient with acute urinary suppression, historical features, clinical observations, and a number of simple laboratory studies may be of considerable assistance in the differential diagnosis.[4, 5] Immediate efforts should be directed toward detecting those causes of acute renal failure that can be rapidly corrected with subsequent return of renal function. For this reason, it is helpful to classify the differential diagnosis of acute renal failure into several categories determined by the anatomic location of the lesion

**Table 1.** Causes of acute renal failure

| |
|---|
| Prerenal—decrease in effective renal blood flow |
| Volume depletion (hemorrhage, vomiting, diarrhea, excessive diuretics) |
| Relative volume reduction (congestive heart failure, sepsis, shock) |
| Postrenal—obstruction |
| Bilateral ureteral (stones, tumor, urates) |
| Bladder outlet (stricture, BPH) |
| Intrinsic renal disease |
| Acute renal failure |
| Bilateral cortical necrosis |
| Acute glomerulonephritis |
| Acute vasculitis |
| Papillary necrosis |
| Interstitial nephritis |
| Malignant hypertension |
| Hepatorenal syndrome |
| Renovascular—bilateral vascular occlusion |
| Arterial (thrombotic, embolic) |
| Venous (thrombotic) |

BPH = benign prostatic hypertrophy.

responsible for the acute renal failure. Prerenal failure relates to a decrease in effective renal blood flow and may be due to absolute or relative volume depletion. The diagnosis is suggested by a history of vomiting, diarrhea, or the use of diuretics and clinical evidence of weight loss, decreased skin turgor, tachycardia, and postural hypotension. The urinalysis will usually show little to no protein, is highly concentrated and virtually free of sodium, reflecting intact renal tubular function. The urine sediment usually reveals little to nothing in the way of cells or other formed elements.

Postrenal disorders relate to obstruction to urine flow. Symptoms suggestive of prostatism and the clinical finding of a distended bladder provide valuable clues to diagnosis. Renal colic or gross hematuria may occur in patients with a renal calculus. The finding of anuria is strongly suggestive of urinary tract obstruction. Patients with partial urinary tract obstruction may have intermittent oliguria or may have polyuria, since elevations of intrarenal pressure impair tubular reabsorption and renal concentration.

A variety of intrinsic renal diseases may be associated with acute reduction in renal function. Patients with primary glomerular diseases often have a history of hematuria and clinical findings of hypertension and edema. Heavy proteinuria and the finding of red blood cell casts on urinary sediment examination are characteristics of glomerular inflammation. Acute renal failure resulting from renal hypoperfusion associated with hemorrhage, trauma, and major surgical procedures represents a major cause of acute renal failure in clinical practice. Together with a variety of nephrotoxic insults, e.g., antibi-

otics, heme pigments, and contrast media, they make up the majority of episodes of acute tubular necrosis as a cause of acute renal failure. Although it is most unusual to see simultaneous acute vascular occlusion of arterial or venous blood supply to both kidneys, acute renal failure may occur following a vascular accident in a patient with a solitary kidney.

A carefully performed urinalysis again may provide important clues to a differential diagnosis. With acute tubular interstitial damage following ischemia or nephrotoxic injury, moderate to heavy proteinuria may be noted and the sediment typically contains many brownish, coarse granular casts. The urine sodium concentration is usually greater than 20 mEq/L and urine specific gravity or osmolality is characteristically low. The typical urine findings in acute renal failure are summarized in *Table 2.*

## Clinical setting for acute renal failure

The usual preparation of patients for major surgical procedures may in fact predispose the individual to the development of acute renal failure in association with or following the surgical event. Typically, the patient is kept NPO for 8 to 12 hours or longer prior to surgery, serving as a significant antidiuretic stimulus to the patient with normal kidneys and resulting in significant decreases in urine output. To this period of fluid restriction is superimposed preoperative narcotics or sedatives and followed by the induction of anesthesia, all of which may serve as antidiuretic stimuli. The patient may be further predisposed to acute renal failure by intraoperative insults such as prolonged hypotension, massive hemorrhage, transfusion reactions, or manipulation of major vessels and viscera during abdominal procedures. The administration of diagnostic contrast media or other nephrotoxic drugs in the immediate preoperative or postoperative periods may further predispose the patient to an acute renal insult.

## Acute renal failure after cardiac operations

Acute renal failure following cardiac surgery, although an infrequent event, is a highly lethal complication.[1-3, 6, 7] Therapy for this postoperative complication, once it is confirmed, is far better directed toward its prevention rather

**Table 2.** Urine findings in acute renal failure

| Urine | Prerenal | Renal | Postrenal |
|---|---|---|---|
| Specific gravity | High (>1.015) | Approx. 1.010 | Approx. 1.010 |
| Osmolarity (mOsm/L) | High (>450) Uosm/Posm >1.5 | Uosm/Posm approx. 1.0 | Uosm/Posm approx. 1.0 |
| Proteinuria | Absent or minimal | Moderate to heavy | Absent or minimal |
| Sediment | Not remarkable | Dirty brown casts in ATN | Not remarkable |
| Uncreat/Pcreat | >40 | <40 | <40 |
| Sodium excretion | <10 | >20 | >20 |

ATN = acute tubular necrosis.

than to treatment. *Table 3* lists some of the preoperative indicators that may assist the physician in predicting those patients preoperatively who have a higher risk of acute renal failure following cardiac operation. The cardiac patient with presenting features of one or more of these indicators warrants careful preoperative evaluation of renal function, including a more quantitative assessment such as that provided by 24-hour endogenous creatinine clearance or possibly a GFR determination. Patients with preoperative impairment in renal function are at a greater risk for the development of acute renal failure following cardiac operations.[6, 8] In view of the extremely high mortality rate as a function of renal failure following cardiac surgery, careful preoperative assessment of renal function would indeed appear imperative in assessing individual patients as surgical risks.

A careful urinalysis with sediment examination should be included, also a quantitative urine protein determination when indicated. The need for special studies such as intravenous urography or tomograms, determination of residual urine, cystoscopy, or even renal angiography is determined by the history, clinical findings, and screening tests of renal function.

**Table 3.** Preoperative indications for ARF after cardiac operation

| |
|---|
| Older patients |
| Poor preoperative hemodynamics |
| Bonafide cardiovascular emergency |
| History of previous cardiac operation |
| Preoperative renal dysfunction |
| Duration of cardiopulmonary bypass (total time of operation, time of aortic crossclamping) |
| Blood pressure at termination of operation |
| Type of operation (valve, congenital, coronary artery bypass) |
| Preoperative diuretic requirements |

ARF = acute renal failure.

## Prevention of acute renal failure

It is far more desirable to take steps to prevent acute renal failure rather than to attempt the management of established cases. Several simple guidelines may be followed. Every attempt should be made to prevent preoperative volume depletion or dehydration. A normal effective circulating blood volume and a high urine flow rate at the time of surgery may in fact help minimize the risk of acute renal ischemia and subsequent acute renal failure. Rapid correction of volume deficits or hypotension or both during surgery should be accomplished. As noted earlier, careful preoperative evaluation of renal function is desirable to identify the higher risk patient. Every attempt should be made to avoid or minimize the use of potential nephrotoxic drugs before or immediately following surgery. Early, aggressive treatment of infection is mandatory because this surgical complication represents the leading cause of death in patients in whom acute renal failure develops following surgical procedures.

## Management of acute renal failure

**Fluid balance.** Fluid intake should be restricted to match insensible losses, renal losses, and other extrarenal losses of fluid. Although careful daily weights should be obtained, the best guide for determining fluid restriction is a daily weight loss of approximately 0.5 kg. This degree of weight loss must be encouraged during the oliguric phase of acute renal failure if an increase in total body water is to be avoided.

**Electrolytes.** Sodium and potassium intake should be restricted to approximate calculated losses. Hyperkalemia represents an early complication in the postsurgical patient with acute renal

failure and may be lethal if not recognized and treated early. Electrocardiograms and careful monitoring of serum potassium concentrations are indicated. The administration of hypertonic glucose intravenously or the ion exchange resin, sodium polystyrene sulfonate (Kayexalate) may be administered orally or by retention enema. Hyperkalemia represents an early indication for dialysis.

**Acid base balance.** The patient must be monitored carefully for the development of symptomatic metabolic acidosis.

**Nutrition.** Nutrition is provided by administering at least 100 g of glucose per day to minimize protein catabolism, and protein intake is restricted. Calories are preferably given by mouth if the patient's condition allows oral intake. Hypertonic glucose (10% to 15%) can be given intravenously if the patient is unable to eat. Recent experience with intravenous hyperalimentation utilizing essential amino acid and hypertonic glucose has suggested that mortality may be reduced in postoperative patients with acute renal failure by the administration of these solutions.[9] Hyperalimentation is particularly appropriate for the patient who is in a poor state of nutrition preoperatively or if the patient is unable to take nourishment orally for periods exceeding 5 to 7 days.

**Hypercalcemia and hyperphosphatemia.** These disorders do not usually require specific therapy. In an occasional patient in whom neuromuscular irritability develops, intravenous calcium supplements may be administered as a therapeutic trial.

**Medications.** The dosage of medications excreted by the kidney will require careful adjustment. If acute renal failure develops while the patient is in the hospital, the record should be screened carefully for evidence of current administration of potentially nephrotoxic drugs, which should be promptly discontinued.

**Prevention of infection.** Infection is the leading cause of death in acute renal failure. Exquisite care must be given to urinary catheters, endotracheal tubes, intravenous lines, and other equipment to minimize the risk of infection and potential sepsis.

**Dialysis.** Dialysis is indicated to control uremia, electrolyte disorders or severe acidosis, or volume overload in acute renal failure. Earlier, prophylactic dialysis is indicated in postoperative patients with acute renal failure, since hypercatabolism increases the risk of early hyperkalemia and other electrolyte disturbances, and is associated with rapid increments in urea nitrogen and creatinine concentrations. Depending upon the surgical procedure preceding the acute renal failure, indications for hemodialysis versus peritoneal dialysis may be considered.

## References

1. Holper K, Struck E, Sebening F. The diagnosis of acute renal failure (ARF) following cardiac surgery with cardio-pulmonary bypass. Thorac Cardiovasc Surg (Stuttgart) 1979; **27:** 231–7.
2. Abel RM, Buckley MJ, Austen WG, Barnett GO, Beck CH Jr, Fischer JE. Acute postoperative renal failure in cardiac surgical patients. J Surg Res 1976; **20:** 341–8.
3. Abbott WM, Abel RM, Beck CH Jr, Fischer JE. Renal failure after ruptured aneurysm. Arch Surg 1975; **110:** 1110–2.
4. Lemann J Jr. Acute renal failure. Am Fam Physician 1978; **18:** 146–56.
5. Levinsky NG. Pathophysiology of acute renal failure. N Engl J Med 1977; **296:** 1453–8.
6. Abel RM, Buckley MJ, Austen WG, Barnett GO, Beck CH Jr, Fischer JE. Etiology, incidence, and prognosis of renal failure following cardiac operations; results of a prospective analysis of 500 consecutive patients. J Thorac Cardiovasc Surg 1976; **71:** 323–33.

7. Hilberman M, Myers BD, Carrie BJ, Derby G, Jamison RL, Stinson EB. Acute renal failure following cardiac surgery. J Thorac Cardiovasc Surg 1979; **77:** 880–8.
8. Yeboah ED, Petrie A, Pead JL. Acute renal failure and open heart surgery. Br Med J 1972; **1:** 415–8.
9. Abel RM, Beck CH Jr, Abbott WM, Ryan JA Jr, Barnett GO, Fischer JE. Improved survival from acute renal failure after treatment with intravenous essential L-amino acids and glucose; results of a prospective, double-blind study. N Engl J Med 1973; **288:** 695–9.