" HOW I MANAGE ? "

"EMERGENCY UPPER ARM FRACUTRE REDUCTION IN CHRONIC RENAL FAILURE"

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INTRODUCTION :

Fractures of the upper arm may be simple fracture or compound fractures. Most of the time fractures are due to history of fall or trauma. More common fractures are of upper 1/3rd of humerus, surgical neck of humerus. Sometimes simple dislocation or fracture with dislocation of shoulder also occurs in trauma patients.

These patients are anaesthetized for closed reduction and some times for open reduction with or without internal fixation. Most of these patients are posted for emergency with full stomach. Compound fracture patients may present with hypovolumia due to blood loss. Hpypovolumia should be replaced by fluid and blood in minimum time. After correction these can be subjected for either closed reduction or open reduction with or without internal fixation.

Dislocation are treated immediately due to severe intractable pain inspite of full stomach condition. Some times these patients are kept nil by mouth for 4 to 6 hours, then taken for required procedure. But most of these patients are treated as full stomach with care of acid aspiration prophyalxis.

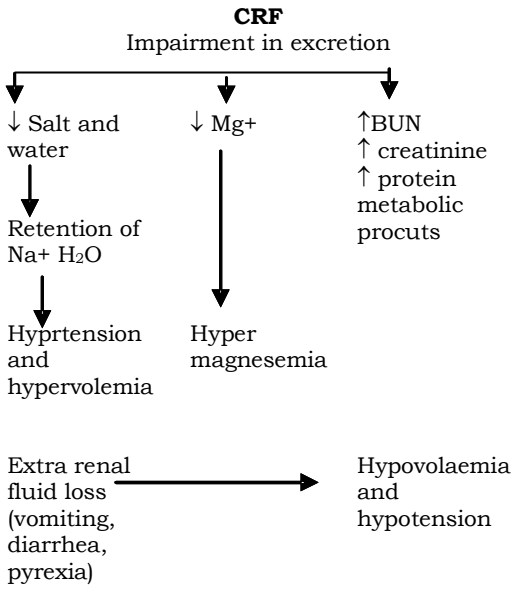
Anaesthesia can be managed by inhalation technique, total intravenous anaesthesia (TIVA) with or without relaxants and various local blocks. If patients are full stomach and associated with medical problems then regional blocks are preferred. Most of the closed reductions are managed as day care procedures by inhalation technique. Sometimes use of succinylcholine can cause violent fasciculation, disturbing some fractures. If it is full stomach patient it is better to secure the airway by intubation even for closed reduction.

But in CRF patients management is different due to various problems like, disturbance in fluid and electrolytes, associated medical diseases like diabetes, hypertension and coronary artery diseases. Therefore we should know what changes occur in CRF patients. CRF patients may be in various stages, these are the changes.

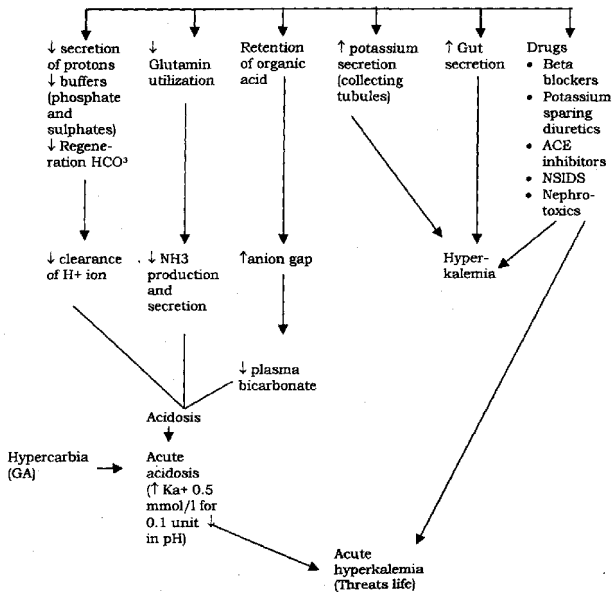
Stages of CRF :

Stage	Description	GFR (mL/ min/ 1.73m^2)	Action
1	Kidney damage with normal or ↑ GFR	≥90	Diagnosis and treatment, treatment of progression. Cardiovascular disease risk reduction.
2	Kidney damage with mildly ↓ GFR	60-89	Estimating progression
3	Moderately ↓ GFR	30-59	Evaluating and treating complications
4	Severely ↓ GFR	15-29	Preparation for kidney replacement therapy.
5	Kidney failure	<15 (or dialysis)	Replacement (if uremia is present)

PATHOPHYSIOLOGICAL CHANGES IN CRF:



(1) Impairment in synthesis/ secretion



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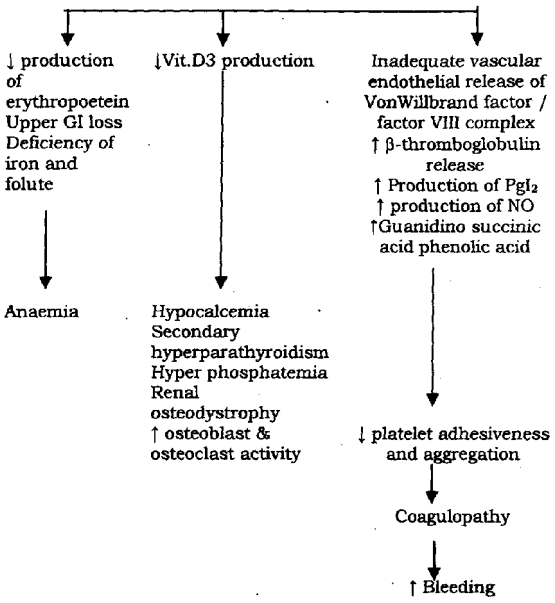

ASSOCIATED SYSTEMIC ABNORMALITIES :

Cardiovascular abnormalities : ***Hypertension*** - Systemic hypertension is most common, with an incidence approaching 80%, although it is often a feature of sodium –wasting nephropathies such as polycystic kidney disease or papillary necrosis. Plasma volume expansion resulting from sodium and water retention is the most frequent cause of hypertension, and may be significantly improved by dialysis. Alteration in the control of rennin and angiotensin secretion may also contribute to hypertension in 30% of patients.

Ischaemic heart disease (IHD) – Is frequent cause of mortality in patients with CRF. The incidence varies with patient subgroup, for example it is present in 85% of diabetics older than 45 years with CRF. Accelerated atherosclerosis results from decreased plasma triglyceride clearance, hypertension and fluid overload causing left ventricular hypertrophy and failure. The elevation in plasma triglyceride levels is caused by a defect in lipoprotein lipase activity and reduced lipolysis.

Valvular heart diseases – The incidence of metastatic calcific valvular heart lesions is increased. Aortic valve calcification occurs in upto 55% of patients, with aortic stenosis being present in 13%. Mitral valve calcification occurs in 40% (stenosis 11%). Elevation in the calcium phosphate product and parathyroid hormone concentrations are the main cause. As a result of these lesions, bacterial endocarditis is much more common in dialysis patients than the normal population.

Pericarditis – Haemorrhagic uraemic pericarditis was often seen prior to the advent of effective dialysis, but is now uncommon and occurs in patients on inadequate dialysis regimen. In untreated, it may progress to pericardial tamponade with hypotension, elevated jugular venous pressure and signs of falling cardiac output. Pericardectomy may be required but should be reserved for those who fail to improve with immediate dialysis. Sudden death from acute cardiac arrhythmias is frequent and is related to both IHD and electrolyte abnormalities.

Pulmonary complications are common in patients with CRF in the postoperative period. Fluid overload, malnutrition, anaemia, impaired humoral and cellular immune function and decreased surfactant production predispose patients to atelectasis and infection.

Immune function

Sepsis is a leading cause of death in patients with CRF. Inhibition of cell mediated immunity and humoral defence mechanisms occurs, with little improvement following the instigation (institution ?) of dialysis. There is an increased production of pro inflammatory cytokines suggesting that activation of monocytes may play a role in uraemic immune dysfunction. Superficial infections are common in fistula and catheter sites, and wound healing is poor.

Gastrointestinal abnormalities

Gastrointestinal abnormalities are frequent with ~anorexia, nausea and vomiting contributing to malnutrition. Urea is a mucosal irritant and bleeding may occur from any part of the GI tract. Gastric emptying is delayed, residual volume is increased and pH is lowered. Peptic ulcer disease is common and most patients receive proton pump inhibitors.

Neurological abnormalities

Many patients with CRF have abnormalities in central (CNS) and peripheral nervous system function. CNS changes have a wide spectrum from mild personality alterations to asterixis, myoclonus, encephalopathy and convulsions. Peripheral neuropathy is common in advanced stages of the disease and is initially a distal "glove and stocking" sensory loss progressing later to motor changes. Both dialysis and renal transplantation may improve the neuropathy.

The presence of a peripheral neuropathy should alert the anaesthetist to the presence of an autonomic neuropathy with delayed gastric emptying, postural hypotension and silent myocardial ischaemia.

Two types of neurological disturbances are unique to patients on dialysis. Dialysis dementia with dyspraxia, myoclonus and dementia occurs in patients on dialysis for many years and may be related to aluminium toxicity. The dialysis disequilibrium syndrome is associated with rapid initial reduction in plasma urea levels at the commencement of dialysis.

Endocrine disturbances

Changes in parathyroid function and lipid clearance have been noted above. Glucose tolerance is impaired, but there is a reduced requirement for exogenous insulin in diabetics, probably related to the reduced metabolism of insulin by the failing kidney. Patients with CRF have abnormalities of temperature regulation with reduced basal metabolic rate and a tendency for hypothermia.

Altered drug handling in CRF

A wide range of pharmacokinetic changes occurs in drug handling in patients with CRF. The volume of distribution is usually decreased, but may be increased if there is fluid retention. Hypoalbuminaemia and acidosis increase the free drug availability of highly protein bound drugs. These changes may require an alteration in the loading dose of a drug. The doses of benzodiazepines and thiopentone may need to be reduced by 30% - 50%. Although the pharmacodynamics of propofol are unchanged in CRF and the metabolites lack sedative activity, changes in volume of distribution and mental state mean that a reduction in induction dose may also be appropriate. The elimination of highly ionised, water soluble drugs such as gallamine or atropine are partially or completely dependent on renal excretion and may be markedly reduced. However the duration of action of a single loading dose will be dependent on redistribution rather than excretion. Dialysis can only partially compensate for the loss of excretory ability of the kidney.

Most lipid soluble analgesics are metabolised by the liver to water soluble metabolites for renal excretion. Some of these metabolites may have far greater activity than the parent drug. Morphine is metabolised to morphine-6-glucuronide, a more potent analgesic and respiratory depressant. The interval between doses will need to be increased because of its reduced renal clearance. Metabolism of pethidine produces norpethidine, which can cause seizures. Although fentanyl undergoes hepatic metabolism and is not thought to have active metabolites. its clearance is decreased in severe uraemia. Alfentanil can be used as normal.

The elimination of volatile anaesthetic agents is not dependent on renal function and their activity is unaffected by CRF. The hepatic metabolism of both enflurane and sevoflurane will theoretically produce nephrotoxic fluoride ions and their use should be discouraged for prolonged durations. Metabolism of halothane produces fluoride ions when the liver is hypoxic but has been used safely in patients with renal disease. It has a greater myocardial depressant effect and causes more . arrhythmias than other inhalational agents. and caution should be observed when used in CRF patients with cardiovascular impairment. Isoflurane, although more expensive, may be the agent of choice as it undergoes less metabolism to fluoride ions. Nitrous oxide has little effect on the kidney.

Atracurium and cisatracurium are obvious choices for muscle relaxation. Around 90% is metabolised by ester hydrolysis and Hofmann elimination. Plasma cholinesterase activity is not thought to be affected by CRF and therefore mivacurium and suxamethonium (in the absence of hyperkalaemia) may be used. Limited doses of vecuronium and rocuronium are acceptable alternatives. Acidosis prolongs the action of all muscle relaxants except gallamine. The excretion of anticholinesterases and anticholinergic agents will be prolonged as they are highly ionised and water soluble.

Local anaesthetics are valuable agents for peri-operative pain control in CRF, but their duration of action is reduced secondary to acidosis. Maximum doses

of local anaesthetics should also be reduced by 25% because of reduced protein binding and a lower CNS seizure threshold.

Non-steroidal anti-inflammatory drugs [NSAIDs) should be avoided in patients with CRF. NSAIDs inhibit the production of renal prostaglandins PGE, and PGI,, which are responsible for maintaining renal blood flow during hypovolumia and in the presence of vasoconstrictors, could precipitate acute renal failure.

HISTORY AND PHYSICAL EXAMINATION :

Conduct a thorough history and physical examination because these are essential in the evaluation of patients with CRF prior to surgery. Obtain information on the following during the history and physical examination :

- Blood pressure and blood sugar trends
- Presence of anemia
- Radio-contrast exposure
- Prior surgical experiences
- Bleeding tendencies
- Allergies
- Use of potentially nephrotoxic drugs
- Nutritional and volume status
- Significant cardiac history
- Presence of comorbid disease
- Functional capacity
- Drug history

Other important history :

Obtain the patient's history related to the following conditions because this information is important in the perioperative treatment of patients with CRF.

- Stable or unstable angina
- Arrhythmias (atrial fibrillation)
- Comorbid disease (e.g., pulmonary disease, history of stroke, transient ischemic attacks).

Obtain the patient's functional capacity by using simplified questions of usual daily activities (eg, climbing flights of stairs, playing tennis, shoveling snow in the winter). Strenuous activities such as swimming, tennis, or basketball have estimated energy requirements of at least 10 metabolic equivalents (METs).

Also enquire about the patient's history of previous surgeries, which helps to determine the effects of general anesthesia and the presence of allergies to medications.

Perform a thorough physical examination, particularly to obtain evidence of volume overload and cardiovascular abnormalities (eg, murmurs, carotid bruits; pericardial effusion, abnormal peripheral pulses). Note the presence or absence of hair on the lower extremities because this information may herald undiagnosed PAD. Record all extremity blood pressures.

Abnormal calcium metabolism is observed in secondary and tertiary hyperparathyroidism, which is prevalent in patients with ESRD. In one retrospective study, the annual incidence of severe valvular heart disease was estimated as 1519 cases per 10,000 patients who were on dialysis. Of these patients, the most common etiology was calcific valvular disease in 69% and endocarditis in 19%. Calcific valvular disease manifested primarily as aortic stenosis endocarditis and mitral regurgitation, which could be due primarily to calcific valvular disease or secondary to endocarditis; therefore, a history of syncope, heart failure, or chest pain should imply not only ischemic heart disease but also the possibility of significant aortic valvular disease.

LABORATORY INVESTIGATIONS :

Investigations	Information
Hematocrit	Anaemia
Complete blood count	Nature of anaemia, leukocytosis (infection)
Urinalysis	Blood, protein, infection
Electrolytes	Na, K, Mg, calcium, phosphate, total CO2
BUN, creatinine	Recent change is most important
Coagulation study	PT, PTT, TT, platelet count, bleeding time
ECG	Myocardial ischemia, LVH, arrhytmias, K status
Chest radiograph	Pleural, pericardial effusion, LVH, pneumonia
Transthoracic echocardiogram	LVH, myocardial contractility, pericardial effusion.

PREOPERATIVE DECISION MAKING AND MANAGEMENT :

Cardiovascular risk

Clinical predictors of preoperative cardiovascular risk (eg, MI, CHF, death) can be describe intermediate or minor risk factors.

- Major predictors
    - Unstable coronary syndromes – Recent MI with evidence of important ischemic clinical symptoms or the results of noninvasive testing or unstable or severe angina (Canadian Heart Association class III or IV).
    - Decompensated CHF
    - Significant arrhythmias – High grade atrioventricualr block, symptomatic ventricular arrhythmias in the presence of underlying heart disease, supraventricular arrhythmias uncontrolled ventricular rate.
    - Severe valvular disease
- Intermediate predictors
    - Mild angina pectoris (Canadian Heart Association class I or II)
    - Prior MI based on history findings or the presence of pathological Q waves.
    - Compensated or prior CHF.
    - Diabetes mellitus
- Minor predictors
    - Advanced age
    - Abnormal ECG findings (eg, left ventricular hypertrophy, left bundle-branch block abnormalities).
    - Rhythm other than sinus (eg, atrial fibrillation)
    - Low functional capacity (eg, inability to climb one flight of stairs).
    - History of stroke.
    - Uncontrolled hypertension.

PREOPERATIVE PREPARATION :

Correction of anaemia :

Blood transfusion is indicated to treat recent acute blood loss or for patients with associated cardio-pulmonary disease undergoing major fracture surgeries, who have heamatocrit less than 25%.

Treatment of hyperkalemia :

Methods of treating a high serum potassium in an emergency include:

1. Administration of 0.5 ml/kg of 10% calcium gluconate (max 20 ml). This has an immediate but transient stabilizing effect on the myocardial cells.
2. 50ml of 50% glucose as an intravenous bolus or infusion. Glucose and insulin will produce an immediate migration of potassium into the cells thus reducing the serum level. Blood glucose levels should be closely monitored but unless the patient is diabetic, endogenous insulin will be secreted and maintain normal glycaemia. Alternatively 5-10 units of soluble insulin may be added to the infusion. Apart from the risk of errors which may occur, the patient may also become hypoglycaemic as secretion of endogenous insulin is also stimulated.
3. Administration of 1-2 mmol/kg sodium bicarbonate intravenously over 5-10 minutes. This provides a large sodium and fluid load which may not be desirable.
4. Nebulised salbutamol 2.5 – 5 mg will assist in moving K+ into the cells.

Total body potassium levels can then be reduced :

1. By dialysis
2. With calcium resonium (0.5g/kg) 8 hourly either rectally or orally. This takes approximately 12 hours to produce an effect.
3. By the introduction of a low potassium diet.

Treatment of hypertension :

Proceeding with emergency surgery may be acceptable in the presence of chronic moderate hypertension (eg. 170/100); however severe or labile hypertension especially if symptomatic (eg. Headache,

nausea, visual deterioration(disturbance ?)) must be controlled in the preoperative period.

BP control may be very difficult in CRF, and patients are likely to be on multiple antihypertensive medications.

Clonidine – Alpha 2-agonist is an effective centrally acting antihypertensive agent but is associated with severe rebound hypertension with abrupt withdrawal. If oral therapy is not able to be restarted immediately after surgery, a clonidine transdermal patch (0.1 – 0.3 mg/day) provides parentral therapy for 1 week.

Other antihypertensive – The ACE inhibitors (eg., captopril, enalapril, lisinopril) can improve renal function when used in the treatment of severe hypertension, but may induce renal decompensation in the presence of hypotension or hypovolemia. Their use should be avoided in patients with renal dysfunction.

Control of blood sugar :

It is better to control with soluble insulin according to blood sugar levels.

Indications for emergency preoperative dialysis include :

- Hyperkalemia (K^+ >6.0 mmol/L)
- Fluid overload and pulmonary oedema
- Metabolic acidosis
- "Uraemic" toxicity and coma.

ANAESTHETIC MANAGEMENT :

Choice of anaesthesia depends upon –

- Type of fracture and procedure either closed or open reduction.
- Associated medical diseases.
- Presence of coagulopathy
- Level of potassium.
- Full stomach due to delayed gastric emptying.

Pre-medication :

CRF patients are susceptible to excessive sedation and respiratory depression hence pre-medication should be –

- Kept to minimum
- Used only if really indicated
- Omitted in the presence of uraemic encephalopathy.

Small dose of short acting anxiolytic (eg. Midazolam) is appropriate for alert, oriented and anxious patient. Glycopyrolate is preferred to atropine and scopolamine, to minimize anticholinergic CNS effects. To decrease the risk of nausea, vomiting, and aspiration an H2-blocker plus metoclopramide may be helpful in reducing gastric volume and acidity.

Simple fractures :

Closed reduction for simple fractures will be carried out by TIVA with or without succinylcholine (no hyperkalaemia). Due to full stomach it is preferred to intubate and maintain with nitrous oxide, oxygen and halothane or isoflurane. For TIVA pentothal sodium or profofol is preferred. If hyperkalemia is present then rocuronium, atracurium or cisatracurium can be preferred in reduced doses. Rocuronium will provide good intubating condition in shorter time than atracurium or cisatracurium. Patient can be reversed with neostigmin and glycopyrolate combination. Stage III and IV patient can be done with interscalene brachial plexus block.

Monitoring :

ECG, Pulse oxymeter, capnography.

Compound fractures :

Compound fractures are some times associated with hypovolumia due to blood loss. Hypovolumia should be corrected by blood and fluid replacement. Here duration of the surgery is also important. Persistant bleeding may be associated with coagulopathy. DDAVP (8-amino-d-arginine vasopressin) in dose of 0.3 microgram / kg IV, normalize the bleeding time and improve the platelet function within about 1-2 hours, and action persists for 6-12 hours. One should be careful in patients already in hypovolumic state. Tourniquet should be avoided in these patients due to cardiac diseases like CAD, hypertension and Valvular Heart Diseases and complication of diabetes like peripheral neuropathy, acidosis.

Type of anaesthesia in compound fracture reduction depends on –

- Duration of the procedure
- Associated medical diseases
- Stage of renal failure
- Full stomach condition
- Coagulopathy
- Presence of hyperkalaemia.

Here due to full stomach and associated diseases regional blocks like interscalene brachial plexus block is preferred. But associated coagulopathy limits its choice. But pretreatment with DDAVP brings the bleeding time

to normal & then regional block can be performed. Premedication with small dose of short acting anxiolytic midazolam is preferred. Due to prolonged duration, of bupivacaine (0.3%) without adrenaline 30-40ml for interscalene approach is used. Ropivacine is an alternative. Procedure less than 60 minutes lidocaine / lignocaine without adrenaline can be used. Positioning of the limb is very important due A-V fistula sites. Good sedation and oxygen supplementation during the procedure is a must.

General anaesthesia :

In presence of coagulopathy general anaesthesia with controlled ventilation is preferred. Whatever the duration of NPO, rapid sequence induction is the choice. One prior DDAVP can be administered to reduce the bleeding.

Pre-medication : Short acting midazolam and fentanyl in reduced doses are preferred for premedication.

Pre-oxygenation : Due to anaemia these patient needs pre-oxygenation for 3-4 minutes.

Intubation : Pentothal sodium or Propofol are preferred IV induction agents. Intubation can be facilitated by succinylcholine (in absence of hyperkalaemia) or rocuronium is preferred. Rapid sequence intubation is the choice due to delayed gastric emptying and full stomach. For maintenance atracurium or cisatracurium or rocuronium should be used in reduced doses. Atracurium and cisatracurium are eliminated by ester hydrolysis or Hoffman elimination.

Maintenance : Anaesthesia should be maintained with nitrous oxide, oxygen, atracurium, cisatracurium or rocuronium.

Fentanyl or sufentanyl or remifentanyl can be used as analgesic.

Volatile anaesthetics like isoflurane or halothane are preferred due to less fluoride ions. One must be careful with halothane due to its potent myocardial depressant action. Care should be taken regarding positioning.

Reversal : Patient can be reversed with neostigmin and glycopyrolate combination in reduced doses due to their prolonged effects.

Monitoring :

- ECG
- Pulse oximeter
- Perpheral nerve stimulator
- Capnography
- Temperature
- CVP - Fluid shift expected and blood loss
  - Stage III and IV CRF.
- PAC is indicated in patients with associated CAD or VHD for monitoring and fluid management.
- Urine output monitoring is a must .

Intraoperative problems like hypertension, arrhythmias and hyperkalaemia should be recognized and proper treatment should instituted. Sometimes pulmonary oedema is also a problem.

Postoperative care :

1. Volume status should be assessed and corrected.
2. Residual neuromuscular block should be assessed.
3. Depression due to opioid should be observed.
4. Postoperative analgesia with systemic opioids.
5. Postoperative ventilation in patients like large fluid shift, fractures with fixation and stage III or IV CRF patients.
6. Intraoperative treated patients.
7. Supplementation of oxygen is must.
8. Fluid should be given as per CVP guidelines.

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