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Peri-Operative Considerations in Gout and Hyperuricemia: A Narrative Review

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Abstract

Gout is a chronic metabolic, inflammatory disease causing hyperuricemia with formation and deposition of monosodium urate crystals in joints, tissues and involving multiple systems. It has important anaesthetic and critical care implications. This review article aims to present an overview of gout and the effects of hyperuricemia along with its important peri-operative implications. The literature is sparse regarding its anaesthetic considerations.

Keywords: Tophi; Gouty Arthritis; Hyperuricemia; Excess Purines; Acute Kidney Injury; Cardiovascular disease; Lipidemia; Non-Steroidal Anti-inflammatory Drugs; Colchicine; Chronic renal failure; Anaesthesia.

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Introduction

Gout is a multisystem disorderin which metabolic and excretory abnormalities, often compounded by excessive purine intake, result in hyperuricemia which promotes the formation of monosodium urate crystals. These crystals either induce inflammation (acute gouty arthritis) or deposit in tissues leading to swelling (tophaceous gout), or both. It is the most common inflammatory arthritis in western countries and is characterized by deposition of monosodium urate crystals in joints and tissues resulting in patient having intermittent painful attacks which often require hospital visits and even admissions. These extremely painful and intermittent attacks are followed by long periods of remission.

Egyptians in *2640 BC* were the first to recognize the disorder which was then referred *aspodagra* (acute gout occurring in the first metatarsophalangeal joint) was later recognized by Hippocrates in the fifth century BC, who referred to it as 'the unwalkable disease' due the unbearable pain associated with it. The term Gout has it's etymological in anold French word *goute*, which is derived from the Latin word gutta (or 'drop'). The word refers to the prevailing medieval belief that an excess of one of the four 'humors'--which in equilibrium were thought to maintain health would, under certain circumstances, 'drop' or flow into a joint, causing pain and inflammation. As medicine advanced it was eventually identified

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the actual excess "humor" in gout to be uric acid. Gout has been associated with a protein rich food and excessive alcohol consumption, alifestyle that, at least in the past, could only be afforded by the affluent. It is for this reason gout was at one time referred to as the '*disease of kings*'. However, as the Western diet developed into one that is purine-rich and consumed by all regardless of economic status, there was a rise in the incidence of gout [1,2].

Risk Factors

Sustained hyperuricemia, which can be caused by overproduction or underexcretion of urate is a risk factor of significant importance. Pathological hyperuricemia has been defined as the serum uric acid concentration (408 µmol/L) above which monosodium urate crystals are formed in vitro at physiological pH and temperature. In most cases, it's the underexcretion of uric acid that is the main cause of hyperuricemia but there are other factors associated with the development of gout as well, which include drugs (such as diuretics, cyclosporin, and low dose aspirin), renal impairment and excessive consumption of red meat or seafood in the diet.

When over production or underexcretion of uric acid occurs, the serum urate concentration may exceed the solubility of urate (a concentration of approximately >6.8 mg/dL) which results in supersaturation of urate in the serum and other extracellular spaces. Sustained hyperuricemia increases the risk for urate crystal deposition from the supersaturated fluids into the tissues. Hyperuricemia which is defined as a serum uric acid level of more than 7.0 mg/dL in men or more than 6.0 mg/dL in women, is clearly associated with an increased risk for the development of gout, although most patients with hyperuricemia might not necessarily develop gout.

Epidemiology

About 3.9% population in the United States have gout, compared with about 2.7% in the early 1990s [2]. Hyperuricemia has been reported in 21% of the US population, correlating with a substantial rise in obesity and hypertension [3]. Risk of developing gout could be age-related; the recent epidemiologic data suggest that the typical age group at the time of diagnosis is between 40 and 69 years [3]. Within this age group, more men are affected than women [3]. This sex discrepancy in the incidence of Gout equalizes after women undergo menopause, suggesting that estrogen plays a role in uric acid regulation [4].

Genetics

Several DNA sequence variants that increase a patient's relative risk of developing hyperuricemia and gout have been indentified in genome-wide association studies [5-7]. The SLC2A9, SLC22A11, and SLC22A12 genes, which correspond with the Glut-9, OAT1, and URAT1 transporters, respectively, have been found to be highly associated with hyperuricemia [5,6]. Glut-9 which plays a role in urate reabsorption at the proximal tubules of the kidney may account for up to 3.7% of a patient's serum uric acid variance [6].

Pathophysiology

Sudden fluctuations in serum uric acid lead to acute flaring up of gout. The monosodium urate crystals begin to form and deposit into bursas, joints and tendons at serum uric acidlevels greater than 6.8 mg/dL [8].

On a cellular level, synovial cells phagocytize the monosodium uratecrystals, which then form an inflammasome. The inflammasome releases interleukin-1 beta (IL-1b) which in turn, leads to release of chemokines and inflammatory mediators to attract neutrophils. A very potent inflammatorystate in the joint or synovial tissues results from this cascade [10]. Tophi formation results from repetitive accumulation of monosodium urate crystals, and is referred to as tophaceous gout. Factors such as temperature,mechanical trauma, previous disease, and underlyingosteoarthritis make a joint susceptible to monosodiumurate crystal deposition and tophi formation [8,9].

Role of diet

A diet consisting of alcohol and excess proteins such as certain meats, seafood, and vegetables primarily associated with the disease. The data available indicates that intake of purinerichmeats and seafood are associated with increases in serum uric acid; whereas, intake of purine-rich vegetables was surprisingly not associated with increased risk of gout [10,11]. Alcohol consumption increases risk of gout and the risk depends on type of alcohol consumed, Beer intake is associated with a high risk of developinggout; whereas, liquor and wine consumption are associated with moderate and low

risks [10,11,12]. Certain food items such as cherries and dairy products have been linked to lower serum uric acid levels and their consumption could lower the incidence of gout [10,11,13]. The data available currently supports thatthe use of low-fat dairy items could be beneficial in reducing serum uric acid.

Associated Comorbidities

There is high association between Gout and hypertension, kidney disease, and cardiovascular disease [14,15,16,17,18]. Rennin angiotensin system stimulation by uric acid leads to vascular constriction, and renal inflammation and injury at microscopic level [16]. The use of uric acidlowering agents such as allopurinol in patients with both hyperuricemia and hypertension has shown a mild blood pressure lowering effect; this hemodynamic effect was reversed when allopurinol was discontinued [17]. Lowering serum uric acid also benefitted the patients by slowing the progression of renal disease [15,16]. As observed in the Multiple Risk Factor Investigative Trial (MRFIT), which was a study of primary prevention of coronary heart disease, gout could be an independent risk factor for myocardial infarction [18]. Antihypertensive such as losartan and antilipemic agents like fenofibrate, have modest uric acid–lowering effects and may be considered along with lifestyle modification specially in hypertensive patients with gout and hypercholesterolemia [19].

Presentation

Presentation of gout consist of spectrum of clinical and pathologic features built on a foundation of an excess burden of uric acid in the body, manifested in part by hyperuricemia, which is defined as a serum urate level greater than either 6.8 or 7.0 mg/dl. Most of the clinical features of gout are due to deposition of monosodium urate monohydrate crystals in supersaturated extracellular fluids of the joint, and certain other sites. Typically, the initial clinical presentation is acute episodic arthritis but patients can also present with chronic arthritis of one or more joints [20].

Suddenly, often in the middle of the night, an affected individual will develop severe, unbearable pain, most commonly in a toe, ankle, or knee. The gouty joint will exhibit a swollen, bluish red appearance and be very warm to the touch compared with the non-inflamed, contralateral joint or even the unaffected skin nearby. If patient presents with pain in single joint (acute monoarticular arthritis) then one should first rule out infection in the joint. The pathognomonic feature of gout is a swelling termed Tophi which is mainly found in periarticular, bursal, bone, auricular, and cutaneous tissues, these swellings are detectable by physical examination and/or by imaging and pathology examination. Renal manifestations of gout include urolithiasis, typically occurring with an acidic urine pH. An uncommon clinical manifestation of gout is, chronic interstitial nephropathy which occurs due to monosodium urate monohydrate crystal deposition in the renal medulla occurring mostly in severe disease [20].

The conditions that promote hyperuricemia, including hypertension, obesity, metabolic syndrome, type 2 diabetes mellitus and chronic kidney disease (CKD) are included in factors responsible for rising prevalence of gout and could be main presenting problems in patients of gout [20].

Diagnosis

Diagnosis of gout requires a detailed history regarding the onset, timeline, location, previous joint trauma or injury, other arthralgias, dietary intake, alcohol consumption, and certain medications. A thorough review of medications in case of acute gout is required since it can be caused byrecently implemented or chronic use of loop diuretics (and to a lesser degree, thiazide diuretics), niacin, or low-dose aspirin which are frequently prescribed these days in patients with cardiovascular disorders [22,23]. Cyclosporine and tacrolimus prescribed in patients who have undergone transplant surgeries have been strongly associated with precipitating gout [24].

The Gold standard for diagnosis of gout is joint aspiration and fluid analysis under microscope during episode of acute gout, in which we observe needle-shaped, negatively green birefringent crystals under polarized light microscope which are characteristic of gout [25,26,27]. Aspiration during the inter-critical or asymptomatic period may only show positive microscopy in about 70% of patients with diagnosed gout therefore the optimal time to make the definitive diagnosis by aspiration is during the acute stage [25,26,27]. Since simultaneous septic arthritis may occur in 4% of patients so to rule out septic arthritis, in addition to crystal examination, aspirated fluid should also be sent for gram stain and culture [27]. The usefulness of serum uric acid level in diagnosing gout remains poor since many patients with hyperuricemia remain asymptomatic and never develop gout. Hyperuricemia (serum

uric acid level greater than 6 mg/dL) indicates only an elevated serum uric acid level and is not diagnostic [26,27,28,29].

Imaging studies during acute gout episodes are useful only for ruling out trauma as no abnormality other than nonspecific soft tissue swelling occur in acute gout. Punched-out erosions and interosseous tophi are the hallmark signs seen in chronic gout. CT, MRI, and ultrasound have proven clinically useful in evaluating joints for tophi in cases with chronic gout. Advanced imaging methods are more typically used to monitor the success of disease treatment rather than for diagnosis.

Treatment

When patients with hyperuricemia experience multiple, frequent, disabling attacks, develop nodular disease that causes bone destruction, or have an illness involving injury to a major internal organ (such as the kidney), then the treatment with Allopurinol may be indicated to lower the level of serum uric acid, which in turn reduces the risk of tissue damage from further crystal deposition. Uric acid lowering therapy [21] which include, xanthine oxidase inhibitor (XOI) namely allopurinol or febuxostat, and uricosuric agents (probenecid, fenofibrate and losartan) are the main line of treatment in patients of gout. An important, but sometimes difficult step in the treatment is to stop theurate-elevating medications such as thiazide and loop diuretics, niacin, and calcineurin inhibitors, since these medications arebeing taken for other conditions such as cardiovascular diseases. An elevated uric acid level can occur even with low-dose acetylsalicylic acid (aspirin 325 mg daily), but it might not possible to recommend discontinuation of this modality as cardiovascular disease prophylaxis in gout patients. In such patients it becomes necessary to have a cardiologist opinion regarding alternative therapies for cardiac disorders. It is imperative to evaluate and manage the associated conditions and risk factors such as obesity, dietary factors, excessive alcohol intake, metabolic syndrome, type 2 diabetes mellitus, hypertension, hyperlipidemia, serum urate–elevating medications, history of urolithiasis chronic kidney, glomerular, or interstitial renal disease (e.g., analgesic nephropathy, polycystic kidney disease). After ruling out common causes, one must search for potential genetic or acquired cause of uric acid overproduction (e.g., inborn error of purine metabolism or psoriasis, myeloproliferative, or lymphoproliferative disease, respectively) and lead intoxication in rare cases [20].

Drugs used in Treatment of Gout

Nonsteroidal anti-inflammatory drugs (NSAIDs) such as naproxen, indomethacin, and sulindac are indicated for initial management in acute gout [30]. Some patients might show intolerance to traditional NSAIDs in such patients selective COX-2 inhibitors can be considered. The risks and benefits of NSAIDs should be carefully considered in each patient—adverse reactions to these drugs include an increased risk of bleeding, gastrointestinal (GI) distress, fluid retention, and hypertension which are become common with increased use of these drugs [30,31,32].

Physicians must show caution or avoid NSAIDs in patients with significant renal impairment, poorly controlled congestive heart failure, history of or active peptic ulcer disease, anticoagulation therapy, or hepatic dysfunction. There are many patients in whom NSAIDs and Colchicine are contraindicated or avoided, in such cases Corticosteroid injection forman effective alternative first-line therapy. Intra-articular approach with triamcinolone acetonidecan be considered in Gout involving fewer than three joints [27,30], otherwise intramuscular injection with triamcinolone acetonide can be given [30,32].

Colchicine is anotheranti-inflammatory drug used for managing acute gout, and should be started within the first 24 to 36 hours from the onset of symptoms [30,32,33]. The therapy might be discontinued due to the gastro-intestinal adverse reactions associated with higher dosages of the drug. Initiating colchicine before starting allopurinol therapy will prevent acute inflammatory episodes as the uric acid load in the body is only gradually eliminated by allopurinol [34]. Colchicine at high doses, is bone marrow–suppressant and in patients with renal insufficiency or patients taking cyclosporine or statins, it can cause neuromyopathy. It has a small benefit-to-toxicity ratio and should only be considered in patients if there is no alternative therapy. Lately subcutaneous injections of Canakinumab, which is a human monoclonal antiinterleukin-1 beta (IL-1b) antibody [31] are being considered in patients who are refractory to the first line drugs used in initial management and in patients whose treatment options are limited because of underlying conditions.

Causes of Hyperurecemia

Uric acid is the catabolic end product of purine nucleotides degradation and itsoverproduction

or under-excretion are leading causes of its accumulation in the body. Diet rich in purines such as organ meat (liver and spleen), red meat, and excessive consumption of alcohol and low alcoholic drinks such as beer is one of the reasons for hyperuricemia condition.

Over production may be caused due to genetic defect such as *Leschnyan syndrome* and health conditions such Multiple Myeloma, where there is high production of cellular nucleotides. Patients should be informed about the factors contributing to their hyperuricemia, such as obesity, a high-purine diet, regular alcohol consumption, and diuretic therapy, which may all be correctable.

Concerns Associated with Hyperurecemia

Cardiovascular Disease and Lipidemia

Hyperuricemia is becoming an important cause for metabolic diseases and CVD [35]. There are reports on hyperuricemia condition that it is associated with metabolic syndrome such as obesity, dyslipidemia and hypertension [36]. Lipid disorders like hypertriglyceridemia have frequently been observed to be linked with hyperuricemia [37]. Various studies have shown that elevated serum uric acid is also related to increased incidence of cardiovascular diseases [38]. Sedentary lifestyle and a diet rich in proteins and alcohol which are now very common in urban population makes them prone to hyperuricemia coupled with dyslipidemia which becomes a main CVD risk factor. Thus, it is of paramount importance that these patients restrict their high fat diet and bring lifestyle modifications to avoid the wave of CVD risk.

Association of Hyperuricemia with hypertension and Renal Injury

Uric acid has many effects on vasculature and renal tissue whichcan cause hypertension. Uric acid directly stimulates the renin-angiotensin system in the kidney [39-41]; inhibits the synthesis of vascular nitric oxide (NO), is a potent vasodilator [39,42]; stimulates the proliferation of smooth muscle cells to promote vascular narrowing and constriction [42-44]; and induce renal abnormalities like renal interstitial inflammation and tubular injury that can indirectly lead to hypertension [43,45]. Treatment with allopurinol blocks uric acid generation by inhibiting enzyme xanthine oxidase and has been found to be useful in correcting both the serum urate levels and the increase in blood pressure [43]. Allopurinol also results in a number of other effects, such as alterations of overall serum antioxidant levels [47-48], which could very well be the mechanism of action as opposed to urate lowering per se.

Hyperuricemia, Gout, Insulin Resistance, and Obesity

Studies done in past have shown that Metabolic syndrome is associated with higher incidence of gout, and individuals with hyperuricemia may also have an increased incidence of insulin resistance. One such study was by Yoo and colleagues [49], who observed that the incidence of insulin resistance in gout patients may be increased by as much as 35% over individuals without gout.

The oxidative stress caused by uric acid in adipocytes is a causative factor in insulin resistance as well as in cardiovascular disease. Sautin and coworkers [50] reported on the formation of NADPH (nicotinamide-adenine dinucleotide-phosphate) oxidase-dependent reactive oxygen species by uric acid. Stimulation with uric acid resulted in activation of MAP (mitogen-activated protein) kinases p38 and ERK1/2, a decrease in NO bioavailability, and an increase in protein nitrosylation and lipid oxidation in cultured mouse adipocytes. Study done by Choi and colleagues [51] observed that a diagnosis of gout conveyed a 35% to 65% increase in risk for future incidence of Type II diabetes.

Animal studies have observed that there is a possibility that urate-lowering may also have indirect benefits in reducing the expansion of the adipose compartment and treatment with allopurinol could reduce the rate and level of obesity [52].

Hyperurecemia and Renal Failure

There is a close association between hyperuricemia and chronic kidney disease (CKD) and elevated uric acid level isalso a risk factor for renal insufficiency in general populations. It is a poor prognostic factor of renal function in patients who also have IgA nephropathy. The treatment of hyperuricemia with allopurinol in patients suffering from CKD resulted in a fall in blood pressure and inhibition of the progression of renal damage. Conversely, the cessation of allopurinol treatment in CKD was followed by a rise in blood pressure and the development of renal damage but this was seen only in patients not receiving angiotensin converting enzyme inhibitor (ACEI) or angiotensin receptor blocker (ARB). The

protective effect of these drugs suggests that the renin angiotensin (RA) system plays an important role in the development of hypertension and renal damage from hyperuricemia.

Hyperurecemia in Tumor Lysis Syndrome and Kidney Injury

Tumor lysis syndrome refers to the metabolic disturbances that occurs when large numbers of neoplastic cells are destroyed rapidly during or more commonly 48-72 hours after chemotherapy, leading to the release of intracellular contents including various ions and metabolic byproducts into the systemic circulation. This release can inundate renal elimination and cellular buffering mechanisms, leading to numerous metabolic derangements. The cell breakdown during the chemotherapy releases nucleic acid purines, which are ultimately metabolized to their end product uric acid by hepatic enzyme xanthine oxidase. The excess conversion occurring in presence of high purine load following chemotherapy leads to hyperuricemia. Uric acid is a weak acid with a pKa of approximately 5.4. It is soluble in plasma and is freely filtered at the renal glomeruli, however it precipitates in renal tubular and collecting duct fluid due to the acidic pH of the media there. This decreased solubility at the level of tubules and collecting duct increases the formation of renal calculi. Since elimination of uric acid primarily occurs from the kidney and a preexisting volume depletion or any other renal dysfunction predisposes patients to worsening metabolic derangements and acute kidney injury (AKI).

Mechanical obstruction by uric acid crystals in the renal tubules leads to obstructive nephropathy which is the major cause of AKI. A high acidity and high concentration in the renal tubular fluid are factors leading to increaseduric acid precipitation. Renal medullary hemoconcentration and a decreased tubular flow rate also contribute to crystallization [54].

Significant Side Effects of Drugs used in Gout

Gout Medication	Side Effects
NSAIDS	indigestion, stomach pain, passing black tarry bowel motions, rash, mouth ulcers, swollen lips, difficulty breathing
Corticosteroids	indigestion, stomach pain, passing black tarry bowel motions, infections, mood changes, sleep problems, weight gain
Colchicine	nausea, vomiting, diarrhea, abdominal pain, blood in the urine
Allopurinol	rash, mouth ulcers, swollen lips, difficulty breathing, kidney stones (severe pain in your back or side), blood in the urine
Probenecid	rash, mouth ulcers, swollen lips, difficulty breathing, kidney stones (severe pain in your back or side), blood in the urine
Febuxostat	diarrhea, nausea, headache, rash, mouth ulcers, swollen lips, difficulty breathing

There is anincreased risk of bleeding, gastrointestinal (GI) distress, fluid retention, and increased BPin patients who are on anti-inflammatory drugs such as NSAIDs for a long period of time. They could also lead to analgesic nephropathy in these patients. NSAIDs should be used with caution or not at all in patients with any of the following: significant renal impairment, poorly controlled congestive heart failure, history of or active peptic ulcer disease, anticoagulation therapy, or hepatic dysfunction. In such scenarios it would be better to take the patients on corticosteroids and avoid NSAIDS.

Anaesthetic Considerations

Preoperative Considerations

As highlighted before gout is associated with many chronic medical conditions which require a thorough preoperative history and physical examination when such patient presents to us before surgery. The changing lifestyles and increased consumption of alcohol and excess proteins have made elevated uric acid a common finding. Detailed history including diet and substance abuse form an important part of preoperative questions and also guide the peri-operative physician to take complete history and order relevant investigations. All Co-morbidities and metabolic changes associated with gout such as disorders in cardiovascular disorders, gastrointestinal and endocrine diseases, obesity, renal dysfunction, renal stones and musculoskeletal disorders should be evaluated, and optimized preoperatively. These disorders have important implications for anesthetic management.Therefore, adequate information about each of the affected systems and ongoing treatment for preexisting problems is the key for giving safe anaesthesia. Excessive consumption of red meat or seafood and drugs (such as diuretics, ciclosporin, and low dose aspirin), renal impairment are some of the important risk factors associated with the development of gout and questions regarding them must be asked from these patients at the time of pre anesthetic check up of the patients. Since lowering serum uric acid also is associated with a slower

progression of renal disease and lowering of blood pressure therefore the patients should be asked during the preoperative visit to continue the uric acid lowering drugs before the surgeries. Special attention and care are needed in these patients regarding cardiovascular status. Assessing cardio pulmonary functional status in these patients could be challenging since they are often unable to perform any physical exercise as a consequence of pain or disability related to arthritisdue to severe pain. Dobutamine stress echocardiogram may be ordered in cases in which we are suspecting cardiac abnormality.

Evaluation of the co-morbidities such as hypertension, ischemic heart disease, pre-existing renal damage, signs of gastro intestinal bleed becomes important part of preoperative examination of these patients. Patients could present in the symptom free inter critical period of the disease so it is advisable to get uric acid levels done for patient with history of joint pain. The patients can be advised to restrict consumption of purine-rich meats and alcohol so as to prevent acute flare ups. A rheumatology work-up could be done in patients with such symptoms so as to start medication for acute flare ups or for review of ongoing medications. A gout patient requires a multidisciplinary approach in the perioperative period. However, there seems to be is no consensus on how to modify its treatment in the perioperative setting.

There is a high degree of association between hyperuricemia, hypertension, kidney disease, and cardiovascular diseasehence asking relevant history, examining the patient and getting ECG and KFT done to rule out associated problems becomes an important part of patient care in preoperative period. An X-ray KUB could be ordered to look for presence of urolithiasis.

According to the Multiple Risk Factor Investigative Trial (MRFIT) gout is an independent risk factor for myocardial infarction. Losartan and fenofibrate have modest uric acid–lowering effects hence they can be useful in patients having gout specially those who also have hypertension and hypercholesterolemia. Caution or complete avoidance should be observed with use of NSAIDS in patients with any of the following: significant renal impairment, poorly controlled congestive heart failure, history of or active peptic ulcer disease, anticoagulation therapy, or hepatic dysfunction. Corticosteroids are better than NSAIDS in such scenarios to prevent life threatening adverse drug effects.

Intraoperative considerations

Proper patient positioning is crucial for ease of surgery and preventing complication due to wrong position is one of the many responsibilities of anaesthesiogist. Positioning can be difficult in any case of arthritis due to pain, hence adequate analgesia and sedation are of extreme importance in these patients. All the standard ASA monitors are applied once the patient is inside Operation theatre (OT), including non-invasive blood pressure, temperature probe, pulse oximetry, ECG, and end-tidal capnography. Since many of these patients are already on long term steroids, a stress-dose corticosteroid should be considered in these patientsto prevent hemodynamic instability due to adrenal insufficiency. As corticosteroid use is a major risk factor for perioperative infection in these patients, therefore one must balance risks of adrenal insufficiency with infection before giving steroids [55]. As even low-dose corticosteroids can lead to disruption of the hypothalamic-pituitary axis, therefore no single cut-off dose can be used to determine which patients may be at risk of adrenal insufficiency [56]. Stress-dose corticosteroids required in the perioperative timeframe depends on the type of surgery and usually 50-75 mg of hydrocortisone or 10-15 mg methylprednisolone can be given intravenously on the day of the procedure. The dose then has to be tapered to the routine corticosteroid dose that patient was taking before the surgery over a period of 1-2 days postoperatively [57].

Gout can result in wide ranging involvement of the larynx including cricoarytenoid arthritis and tophi of laryngeal tissue, but such reports are exceedingly rare. A previously undiagnosed laryngeal arthritis can exacerbate with the use of laryngeal mask airway (LMA) or trauma during intubation, and this possibility must be kept in the list of differential diagnosis in a setting of acute upper airway obstruction, particularly following extubation.

Postoperative period

The important concerns included in the post operative period are adequate pain control, wound care, prevention of deep vein thrombosis, and maintenance of renal function fluid management. Extreme pain on movement of affected joints in these patients require use of multimodal pain control regimen. The multimodal approach may include acetaminophen, NSAIDs, intravenous opioids, local anesthetics injected

to wound or port site, tramadol and regional anaesthesia. A good pain control results in early mobilization and prevention of complications related to immobility such as thrombosis. Fluid management should be considered according to specific requirements of the patient and presence of associated comorbidities specially cardiac and renal dysfunction. A careful recording of input and output status must be done. In addition to all these factors a high quality nursing care is of extreme importance to ensure reduction in complications and faster recovery.

Conclusions

Gout and hyperuricemia form important risk factors for surgery and anaesthesia. It can lead to diseases of the musculo-skeletal, cardiovascular and renal systems. The adverse effects of drugs used to treat gout and hyperuricemia should also be considered in the peri-operative period. Maintenance of end organ perfusion, evaluation of renal function tests, assessment of cardio-pulmonary exercise testing and padding of pressure points and joints is of paramount importance. Hyper-uricemic emergencies like tumor lysis syndrome may require admission to the intensive care unit and renal replacement therapy.

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