Optimizing Perioperative Management of Sarcoidosis in Noncardiac Surgery: A Focused Review and Practical Framework

Zyad J. Carr, M.D., Jean Paul Higuero Sevilla, M.D.

Sarcoidosis is a multisystem granulomatous disease affecting 8 to 10 per 100,000 individuals (United States), with pulmonary (90%), cardiac (5 to 25%), renal (10%), hepatic (20%), and neurologic (5 to 10%) involvement. Prognosis is strongly associated with ethnicity, genetics, organ involvement, and timing of presentation, with geographic and demographic variation in incidence and prevalence worldwide.[1] In the United States, sarcoidosis incidence in White individuals (8.1 per 100,000) is significantly lower than in Black individuals (17.8 per 100,000).[2] Black patients experience earlier disease onset (mean age, 40 *vs.* 45 yr), more severe multiorgan involvement, and more severe extrapulmonary manifestations.[1] Sarcoidosis has a highly variable course, with spontaneous remission occurring in 10 to 82% of cases (highest in Scadding stage I at 60 to 90%) and 10% developing advanced pulmonary sarcoidosis (lung fibrosis) with a 7% 5-yr mortality and 60% of deaths related to advanced lung disease.[3,4] In the United States, sarcoidosis represents a significant health burden, with analysis of healthcare utilization data revealing considerable morbidity.[2] Despite these clinical implications, evidence-based perioperative management guidance is lacking. Thus, this clinical focus review addresses seven key questions to provide practical, evidence-informed recommendations for anesthesiologists and perioperative physicians caring for sarcoidosis patients undergoing noncardiac surgery.

Pathophysiology: What Is the Pathophysiologic Basis for Perioperative Risk in Patients with Sarcoidosis?

Tissue infiltration with noncaseating granuloma formation forms the pathognomonic basis for sarcoidosis's wide-ranging clinical manifestations (fig. 1).[5] Dysregulated immune responses characterized by tissue infiltration of mononuclear phagocytes and lymphocytes lead to the characteristic noncaseating granuloma formation.[6] The inflammatory cascade is driven by antigen-presenting cells activating $CD4^+$ T-helper cells, which polarize toward a T-helper 1 cell phenotype and secrete proinflammatory cytokines, including interferon-γ, interleukin-2, and tumor necrosis factor-α (TNF-α).[3] TNF-α plays a central role in granuloma formation and maintenance, providing the rationale for anti–TNF-α biologic therapies in the treatment of refractory sarcoidosis. The granulomatous inflammation results in local tissue injury through multiple mechanisms: direct cellular infiltration disrupting normal architecture, cytokine-mediated tissue damage, and fibrotic transformation when inflammation persists.

Pulmonary involvement in sarcoidosis follows a progressive pathway from initial alveolitis to granulomatous inflammation, culminating in irreversible fibrosis in advanced disease (fig. 2).[7] Advanced pulmonary sarcoidosis is defined by significant risk of organ function loss or death and encompasses advanced pulmonary fibrosis, pulmonary hypertension (PH), and chronic respiratory insufficiency.[8] PH develops through multiple mechanisms: parenchymal destruction from fibrosis, direct granulomatous infiltration of pulmonary vessels, extrinsic vascular compression by mediastinal and hilar lymphadenopathy, and left heart dysfunction secondary to cardiac sarcoidosis.[9] Sarcoidosis-related PH has been reported in 5 to 15% of all sarcoidosis patients. In 130 sarcoidosis patients with persistent dyspnea, PH without left ventricular dysfunction confers significantly worse survival compared to patients without PH (hazard ratio [HR], 10.39) and those with PH due to left ventricular dysfunction (HR, 3.14), emphasizing the need for right heart catheterization for proper characterization and management.[10] Patients with advanced pulmonary sarcoidosis who fail medical management may require lung transplantation and face unique challenges including

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Abbreviations: **DLCO**, diffusing capacity of the lung for carbon monoxide; **FVC**, forced vital capacity; **HR**, hazard ratio; **ICD**, implantable cardioverter-defibrillator; **PEEP**, positive end-expiratory pressure; **PFT**, pulmonary function test; **PH**, pulmonary hypertension; **TNF-α**, tumor necrosis factor-α

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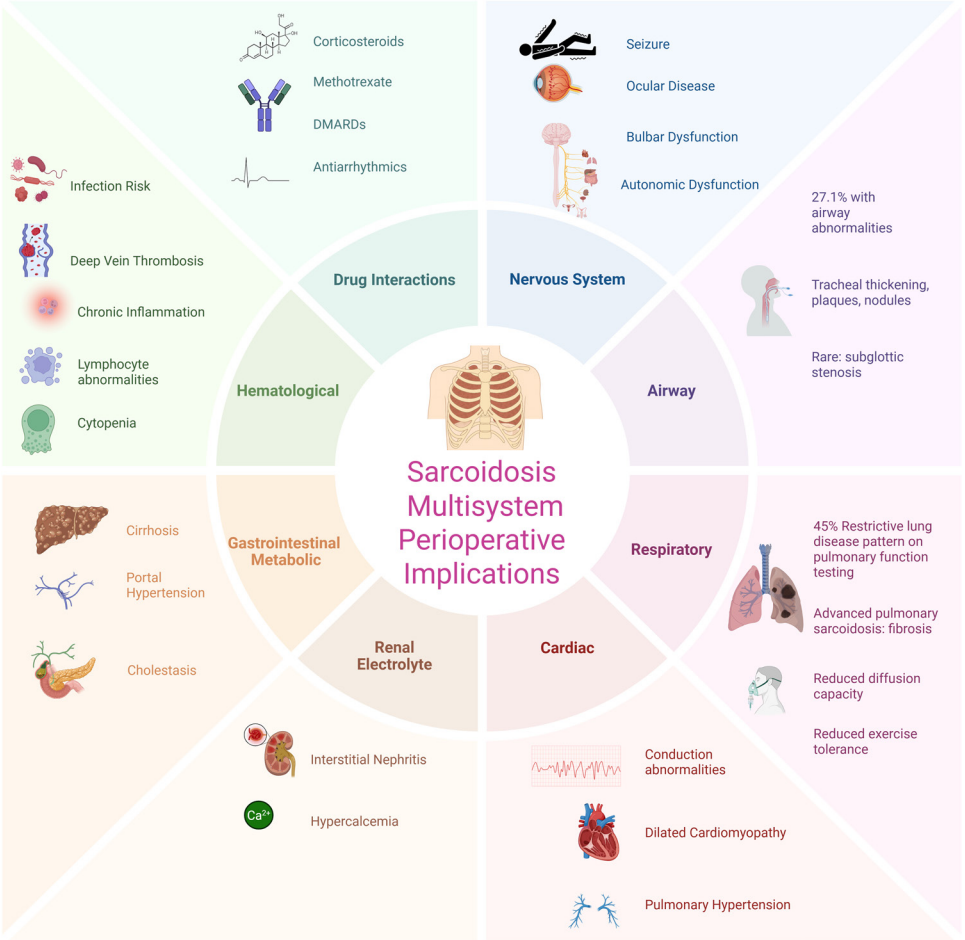


Fig. 1. Illustration of the multiorgan manifestations of sarcoidosis relevant to perioperative management. Drug interactions emphasize immunosuppressive therapies requiring perioperative consideration. Neurologic considerations include seizure and bulbar neuropathies that may increase aspiration risk. Airway involvement may rarely lead to subglottic stenosis, with resultant difficult airway management. Respiratory disease is highlighted by the presence of restrictive lung disease patterns on pulmonary function testing, requiring close attention to mechanical ventilatory support. Conduction abnormalities and their interactions with anesthetic drugs are of critical focus. Electrolyte and liver dysfunction may impact anesthetic drug delivery. Infection and deep vein thrombosis risk in the setting of chronic immune dysfunction and immunosuppression highlight the potential for adverse outcomes in the perioperative time period. Ca^{2+}, calcium; DMARDs, disease-modifying antirheumatic drugs.

extrapulmonary disease manifestations and immunosuppressive therapy complications.[8,11] Overall survival is poor in patients with advanced pulmonary sarcoidosis. In radiographic stage IV sarcoidosis patients, PH was present in 29.7%, aspergilloma in 11.3%, and long-term oxygen therapy in 12%, with 75% of fatalities directly attributable to respiratory causes.[12] The risk of acute exacerbation of interstitial lung disease after noncardiac surgery should be highlighted as an important perioperative concern, with mechanical ventilation and surgical stress triggering acute deterioration in some patients with underlying fibrotic lung disease.[13]

In cardiac sarcoidosis, granulomatous myocardial inflammation disrupts the conduction system and distorts myocardial architecture, resulting in high-grade or atypical conduction disease, ventricular arrhythmias, and biventricular dysfunction that may remain clinically silent until unmasked by perioperative stress.[14] This infiltrative process preferentially affects the basal septum and left ventricular free wall, creating an arrhythmogenic substrate that predisposes to sudden cardiac death.[15,16] Cardiac manifestations, although less common, carrying profound prognostic implications.[15,17] Increasing cardiovascular mortality has been identified in sarcoidosis patients, highlighted by an increased susceptibility in women and Black Americans.[18] Cardiac sarcoidosis poses particular challenges in the perioperative setting, as it can potentially manifest with life-threatening arrhythmias and conduction abnormalities that may be clinically silent until unmasked by surgical stress and anesthetic medication management.[14,16] Nearly 50% of patients referred with unexplained cardiomyopathy and ventricular arrhythmia had positron emission tomography-identified myocardial inflammation and undiagnosed inflammatory or autoimmune cardiomyopathy.[19] Case

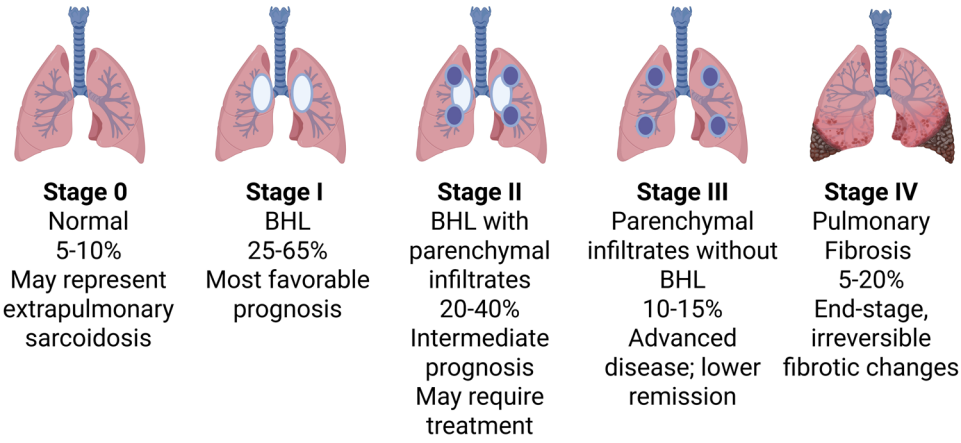


Fig. 2. The Scadding scale classifies pulmonary sarcoidosis into five stages (0 to IV) based on chest radiographic findings. Each depicts characteristic radiographic features with corresponding prevalence at presentation. Stages demonstrate inverse correlation between disease severity and likelihood of spontaneous remission although progression through stages is not necessarily sequential, and patients may present at any stage. BHL, bilateral hilar lymphadenopathy.

reports have documented perioperative heart block in young patients as the initial presentation of cardiac sarcoidosis.[20,21]

Neurosarcoidosis is characterized by granulomatous infiltration of the central and peripheral nervous system, affecting 5 to 10% of patients with systemic sarcoidosis. It commonly involves the basal meninges (leptomeningeal disease in 40% of cases), hypothalamic–pituitary axis (diabetes insipidus and hypopituitarism), or cranial nerves (VII or II most commonly). Clinical presentations include seizure, altered mental status, cranial nerve palsies, autonomic dysfunction (rare), or diabetes insipidus.[22] Ocular sarcoidosis commonly manifests with granulomatous anterior uveitis and inflammatory involvement of the retina and optic nerve, and occurs in roughly 25% of patients with systemic sarcoidosis.[23] Vision loss occurs through multiple mechanisms: (1) direct inflammatory damage, (2) cataract formation (chronic inflammation or corticosteroid therapy), (3) secondary glaucoma from trabecular meshwork obstruction, (4) cystoid macular edema, or (5) direct retinal vascular occlusion (granulomatous vasculitis).

Renal disease is characterized by granulomatous interstitial nephritis, nephrocalcinosis, electrolyte disturbances, or hypercalcemia from dysregulated vitamin D metabolism associated with granulomatous disease. Dysregulated 1-α-hydroxylase activity in granulomas produces excessive calcitriol, causing hypercalcemia in 10 to 20% and hypercalciuria in 40 to 60% of patients.[3] Hypercalcemia induces anorexia, nausea, vomiting, or constipation, all reducing oral intake and nutritional status. Hepatic disease related to granulomatous infiltration may cause cholestasis, portal venule obstruction, cholestatic disease ultimately developing into cirrhosis, and portal hypertension. Portal hypertension develops through presinusoidal obstruction from portal vein granulomas or postsinusoidal obstruction from hepatic vein involvement (Budd–Chiari—like syndrome). Although frequently present in subclinical form (80%), clinically significant hepatic disease is present in fewer than 20% of patients.[24] Cholestatic injuries manifest as elevated alkaline phosphatase (30 to 50%) with variable elevation in aminotransferases and bilirubin. Gastrointestinal sarcoidosis is rare (less than 1%) and frequently presents with submucosal/mucosal granulomatous infiltration, gastroparesis from myenteric plexus involvement, or gastric outlet obstruction from antral/pyloric masses.[25]

Hypoalbuminemia (less than 3.5 g/dl) occurs in 20 to 40% of hospitalized sarcoidosis patients, impairing drug binding, and altering pharmacokinetics of highly protein-bound drugs. Reduced oncotic pressure enhances the development of peripheral edema, ascites, and pulmonary edema, complicating fluid management. This is further exacerbated by the development of sarcopenia related to chronic inflammation, corticosteroid therapy, and physical deconditioning. Sarcopenia is frequently associated with sarcoidosis and impairs respiratory muscle strength, likely predisposing to postoperative respiratory insufficiency and prolonged mechanical ventilation.[26,27] Sarcoid arthropathy presents as acute arthritis (Lofgren syndrome) or chronic arthritis affecting knees, ankles, wrists, and small joints of the hand. Osseus infiltrations typically affect the phalanges of the hands and feet, weakening bone structure and increasing the risk of pathologic fractures.

Preoperative Assessment: Which Preoperative Assessments Are Essential to Identify High-risk Sarcoidosis Patients?

Preoperative assessment targets multiorgan involvement with focus on sarcoidosis symptoms, exercise tolerance, arrhythmia screening, or worsening pulmonary function tests (PFTs; table 1) balanced by procedural complexity. Sarcoidosis patients with no symptoms or normal PFTs likely require standard preoperative investigation. Sarcoidosis is associated

with reduced exercise tolerance and respiratory muscle weakness, frequently independent of body mass index.[28,29]

Pulmonary assessment includes review of respiratory symptoms, pulmonary function testing (spirometry and diffusion capacity), and chest imaging (ideally within less than 12 months). Typical pulmonary function testing in sarcoidosis most often identifies a restrictive pattern (approximately 45%), is typically correlated with radiographic lung staging (Scadding staging), and usually presents with reduced forced vital capacity (FVC) and total lung capacity.[30,31] Black Americans are far more likely to have a restrictive pattern (41% *vs*. 9%), while White patients typically present with obstructive patterns (17% *vs*. 9%).[32] Diffusing capacity of the lung for carbon monoxide (DLCO) may be disproportionately reduced relative to restriction. Although not validated, FVC less than 70%, DLCO less than 70%, absolute FVC decline of 10% or greater, or DLCO greater than 15% for longer than 6 months are suggestive of clinically meaningful thresholds that may trigger initiation or escalation of therapy.[33] Optimization strategies may include corticosteroid supplementation, bronchodilator trial (if obstructive pattern is present), or pulmonary rehabilitation. Review of recent high-resolution computed tomography imaging may aid identification of fibrosis and PH features that may significantly impact perioperative care.[3] If PH is suspected, echocardiography is the initial screening step, followed by right heart catheterization for confirmation.[10] Baseline screening for obstructive sleep apnea should be performed, as it has been identified with high frequency in sarcoidosis (88.2%), theorized to be related to the increased susceptibility to upper airway collapse associated with restrictive lung disease or, possibly, sarcoid neuropathy.[34]

Cardiac sarcoidosis (clinically apparent: 5%, autopsy: 20 to 30%), mandates preoperative electrocardiogram given the risks of conduction abnormalities, ventricular arrhythmias, and cardiomyopathy, particularly where granulomatous inflammation has caused scarring.[16] Cardiac sarcoidosis leads to various atypical electrocardiogram abnormalities, notably high-grade atrioventricular block and right bundle branch block, occurring in 15 to 25% of cases due to preferential involvement of the basal interventricular septum.[35] Conduction abnormalities are present in 65% of cardiac sarcoidosis patients at presentation, with complete heart block the most common (48%), followed by first-degree atrioventricular block (44%) and second-degree atrioventricular block (34%). Bundle branch blocks are also frequent, including right bundle branch block (37%) and left bundle branch block (21%). Complete heart block serves as a presenting feature in nearly half of cardiac sarcoidosis, emphasizing the need for early screening and monitoring for progression.[36] Cardiac magnetic resonance imaging and/or fluorodeoxyglucose positron emission

Table 1. Clinical "Red Flag" Preoperative Symptoms in Sarcoidosis Patients for Noncardiac Surgery

Organ System	Red Flag Symptoms
Cardiac	Syncope or presyncope
	Palpitations or documented arrhythmias
	Known/high-grade atrioventricular block or bundle branch block
	Abnormal electrocardiogram suggestive of conduction disease or ventricular arrhythmia risk
	New or worsening heart failure symptoms (orthopnea, edema)
	Unexplained reduced LVEF or cardiomyopathy
	Previous cardiac sarcoidosis diagnosis without recent assessment
	ICD/PPM issues or recent shocks
Pulmonary	Resting hypoxemia or significant exertional desaturation
	Rapid decline in exercise tolerance
	Recent decline in PFTs (FVC, DLCO)
	New/worsening dyspnea, rales, or cough suggestive of progression
	HRCT or CXR suggesting extensive parenchymal disease/fibrosis
	Clinical or echo signs of pulmonary hypertension
	Active respiratory infection or exacerbation
Airway/ENT	Hoarseness, stridor, or dysphonia suggestive of laryngeal/subglottic involvement
	History of difficult intubation or airway stenosis
	Symptoms of upper airway obstruction
Neurologic	Active neurosarcoidosis symptoms (recent focal deficits, seizures, meningitis symptoms)
	Autonomic dysfunction impacting hemodynamic stability
	Raised intracranial pressure concerns
Ocular	Active uveitis or severe ocular inflammation impacting perioperative steroid needs or positioning
Renal/metabolic	Hypercalcemia (especially symptomatic or severe)
	Worsening kidney function (creatinine/eGFR decline) attributable to sarcoidosis
Hepatic	Significant hepatic enzyme elevation or cholestasis impacting drug handling
Systemic/ trajectory	Rapid clinical progression in the previous weeks to months
	Poorly controlled systemic inflammation requiring escalation of immunosuppression
	Recent initiation or changes in biologics without infection risk assessment

CXR, chest radiograph; DLCO, diffusing capacity of the lung for carbon monoxide; echo, echocardiographic; eGFR, estimated glomerular filtration rate; ENT, ear–nose–throat; FVC, forced vital capacity; HRCT, high-resolution computed tomography; ICD/PPM, implantable cardioverter-defibrillator/permanent pacemaker; LVEF, left ventricular ejection fraction; PFT, pulmonary function test.

tomography is indicated if electrocardiogram abnormalities or high-grade features (reduced ejection fraction, high-grade atrioventricular block, sustained ventricular tachycardia, syncope) are present in the absence of previous diagnosis of cardiac sarcoidosis. Holter monitoring may identify subclinical arrhythmias and should be considered in all patients with palpitations.[14] Cardiac biomarkers play an important but nonspecific role in cardiac sarcoidosis evaluation. Elevated troponin may indicate active myocardial inflammation or injury, while natriuretic peptide (B-type natriuretic peptide greater than 100 pg/ml or N-terminal pro-brain natriuretic peptide greater than 300 pg/ml) may suggest ventricular dysfunction or overt heart failure.[37] Frequently used antiarrhythmic therapies in cardiac sarcoidosis include implantable cardioverter-defibrillators (ICDs), amiodarone, sotalol, β-blockers, and mexiletine for refractory ventricular tachycardia. ICD placement is indicated for secondary prevention in patients with previous cardiac arrest or sustained ventricular tachycardia, and primary prevention in those with left ventricular ejection fraction 35% or less despite optimal medical therapy or select patients with extensive myocardial scarring on cardiac magnetic resonance imaging greater than 20% of LV mass.[38]

A serum metabolic panel is recommended to screen for abnormal renal function, liver involvement, and hypercalcemia, a sequela of excessive granulomatous vitamin D production in a subset of sarcoid patients (10%).[39] Hypercalcemia impacts cardiac conduction (shortened QT, prolonged PR, QRS) and coagulation (increased thrombosis) and may cause preoperative volume depletion. Uncontrolled hypercalcemia necessitates referral for consideration of therapy initiation.

Neurosarcoidosis (5 to 15%) should be carefully evaluated due to seizure risk (20%). Severe cases may have bulbar and autonomic dysfunction, requiring both aspiration risk assessment and postoperative ventilation planning. Ocular sarcoidosis may require medication adjustment to achieve a quiescent eye examination at least 3 months before surgical intervention. Identified cutaneous lesions (lupus pernio, papular sarcoidosis) require dermatologic assessment as they may indicate systemic disease activity.

Muscle atrophy and sarcopenia is present in 25% of sarcoidosis patients and is associated with more severe pulmonary disease.[40] Prehabilitative assessment should include (1) unintentional weight loss history (greater than 10% in 6 months may indicate malnutrition), (2) serum albumin and prealbumin (prealbumin less than 15 mg/dl indicates acute malnutrition), (3) complete blood count assessing for anemia and lymphopenia, (4) comprehensive metabolic panel including calcium and phosphate, (5) vitamin D (25-hydroxyvitamin D) and vitamin B12 levels, and (6) functional assessment including grip strength and 6-min walk distance. Respiratory muscle weakness may benefit from preoperative inspiratory muscle training.[41]

We recommend deferring elective procedures with acute or subacute deterioration in pulmonary status (for example, a recent 10% or greater decline in FVC or DLCO 15% or greater, new or increased oxygen requirement, or radiographic evidence of progression), unstable cardiac sarcoidosis (high-grade atrioventricular block, sustained ventricular arrhythmias, recent decompensated heart failure, or recent implantable cardioverter-defibrillator placement), clinically significant hypercalcemia requiring urgent treatment, or uncontrolled neurosarcoidosis with seizures, bulbar dysfunction, or autonomic instability. When clinically feasible, elective surgery should be delayed until symptoms and objective parameters of disease activity have stabilized or improved with therapy. A multidisciplinary preoperative assessment, particularly involving pulmonology and cardiology, is strongly recommended. For urgent or time-sensitive procedures, shared decision-making is essential and may favor proceeding with enhanced perioperative surveillance rather than surgery deferral.

Medication Management: How Should Immunosuppressive and Cardiovascular Medications Be Managed Perioperatively?

Chronic immunosuppressive therapy required for sarcoidosis disease control creates competing risks of inadequate stress response and infection during the perioperative time period. Patients on chronic corticosteroids (longer than 3 weeks, more than 5 mg/day prednisone) require procedural risk-adjusted stress-dose supplementation to avoid adrenal crisis. Methotrexate, recently proven to be noninferior to prednisone as first-line therapy,[42] is usually considered safe for perioperative continuation, while azathioprine and TNF-α inhibitors (infliximab, adalimumab) are typically held for 1 to 2 weeks and 1 to 2 half-lives, respectively, but should be made with multidisciplinary consultation.[43]

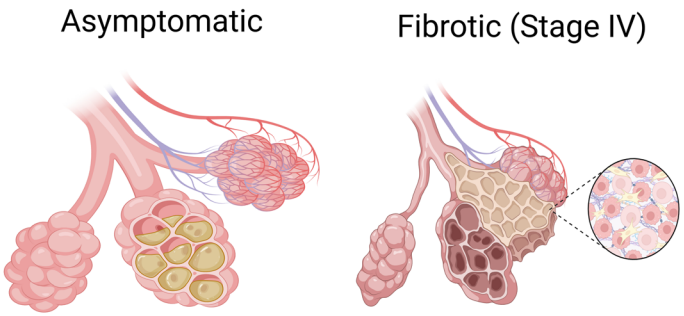
Intraoperative Management: What Intraoperative Monitoring and Anesthetic Strategies Optimize Patient Safety?

Airway Considerations

Airway abnormalities were identified in 27.1% of sarcoidosis patients (thickening, 21.4%; nodules, 11.9%; plaques, 7.2%), with affected patients more likely to have chronic cough (67.1% *vs.* 51.5%) and more extensive pulmonary infiltration on review of recent high-resolution computed tomography.[44] Although rare, sarcoidosis can cause subglottic/tracheobronchial stenosis and difficult intubation, necessitating detailed preoperative airway assessment and availability of fiberoptic intubation equipment.[45]

Ventilation Management

Careful respiratory management remains a focus of intraoperative care (fig. 3). Lung-protective ventilation (6 to 8 ml/kg), plateau pressures greater than 30 cm H_2O, and optimizing positive end-expiratory pressure (PEEP) based on compliance is essential. Ventilation strategies should minimize barotrauma risk, particularly in those with evidence of pulmonary fibrosis (Scadding stage IV). Avoid excessively high inspired oxygen concentrations to reduce the risk of absorptive atelectasis.



Tidal Volume	6-8 mL/kg IBW	6 mL/kg IBW
Plateau Pressure	<30 cmH_2O	<30 cmH_2O
PEEP	Optimize by compliance	Lower (5-8 cmH_2O)
FiO_2	Lowest for SpO_2 >92%	Lowest for SpO_2 >92%
Respiratory Rate	Adjust for pH 7.35-7.45	Adjust for pH >7.25
Permissive Hypercapnia	Not typically needed	May be necessary
Recruitment Maneuvers	Cautious (PNX risk)	Avoid

Fig. 3. Comparative ventilation parameters for patients with asymptomatic sarcoidosis *vs.* advanced fibrotic disease (Scadding stage IV). *Left*, Lung morphology and recommended ventilation goals for asymptomatic sarcoidosis with preserved lung architecture. *Right*, Advanced fibrotic disease with honeycomb pattern and reduced compliance, requiring more conservative ventilation parameters. Aim for the lowest plateau pressure compatible with adequate ventilation; values greater than 25 cm H_2O may be unavoidable in severe fibrosis. Obtain periodic arterial blood gas analysis to confirm acceptable $Paco_2$ and detect life-threatening respiratory acidosis. Fio_2, fraction of inspired oxygen; IBW, ideal body weight; PEEP, positive end-expiratory pressure; PNX, pneumothorax; Spo_2, oxygen saturation measured by pulse oximetry.

Lung recruitment maneuvers should be used sparingly and with caution, given the increased risk of barotrauma and pneumothorax in patients with bullous lung disease, which occurs in approximately 1% of individuals with sarcoidosis.[4] Although a restrictive pattern is most common, a substantial subset of sarcoidosis patients will present with obstructive physiology or mixed patterns, posing different intraoperative ventilatory challenges. In obstructive disease, dynamic hyperinflation and intrinsic PEEP can develop with insufficient expiratory time, leading to hypotension and ventilator-associated lung injury. Vigilant preoperative identification of spirometric evidence of airflow limitation, coupled with history of wheezing, cough, or bronchodilator responsiveness, should be highlighted. Intraoperatively, plateau pressure and auto-PEEP measurements, expiratory flow waveforms, and rising airway pressures suggest dynamic hyperinflation. Management includes reducing respiratory rate, lengthening expiratory time (inspiratory to expiratory [I:E] ratio, 1:3 or greater), permissive hypercapnia, and utilization of the lowest effective external PEEP, while monitoring hemodynamics. Bronchodilators and meticulous airway suctioning further reduce air trapping. Ventilator settings should be individualized, not only to the degree of restriction but also to the presence of obstructive physiology. When general anesthesia is necessary, early extubation should be prioritized in patients with adequate pulmonary reserve, while those with severe restrictive disease (FVC less than 50% predicted) or PH may benefit from careful postoperative ventilation planning. For advanced pulmonary sarcoidosis, a perioperative oxygen saturation target of 92 to 96% is recommended.

Advanced Hemodynamic Monitoring

In patients with cardiac sarcoidosis or associated PH, invasive hemodynamic monitoring (arterial catheterization, pulmonary artery catheter, transesophageal echocardiography) is warranted for major surgery. In patients with severe PH or right ventricular dysfunction, intraoperative hemodynamic goals include using vasopressors that preserve systemic blood pressure without excessive tachycardia, supplemented by selective pulmonary vasodilators, when necessary.[46] Perioperatively, patients with known or suspected cardiac sarcoidosis require continuous telemetry monitoring, availability of temporary pacing, and close coordination with electrophysiology services, as perioperative stress and hemodynamic changes can precipitate life-threatening arrhythmias or conduction disturbances. Goal-directed fluid therapy avoids pulmonary edema risk, while active warming prevents hypothermia-induced arrhythmias.

Device and Drug Interactions

ICDs require careful management with consideration for reprogramming to asynchronous pacing modes or magnet application during monopolar cautery use. Antiarrhythmic medications present specific anesthetic challenges: Sotalol increases bradycardia and QT prolongation, while amiodarone may increase defibrillation threshold and interacts with common anesthetic adjuncts including ondansetron (QT prolongation). Immediate availability of antiarrhythmic medications, temporary pacing capability,

and defibrillation equipment is essential. Anesthetic selection should minimize myocardial depression in cardiac sarcoidosis, and careful attention should be paid to serum potassium and magnesium correction. Hypoalbuminemia, common in advanced disease, alters pharmacokinetics of protein-bound drugs, increasing free drug fraction and potentiating effects of propofol, benzodiazepines, and neuromuscular blocking agents, necessitating dose reduction and careful titration. Glucose monitoring is essential given steroid administration.

Anesthetic Technique

Regional anesthesia offers advantages when feasible, reducing systemic effects and exposure to mechanical ventilation, with careful consideration in patients on anticoagulation or with thrombocytopenia related to hypersplenism.

Postoperative Care: Which Postoperative Monitoring Strategies and Care Pathways Reduce Complications?

Optimal care emphasizes early mobilization, cardiac monitoring, infection control, and pulmonary hygiene (fig. 4). Intensive care unit or telemetry admission for 48 to 72 h is recommended for cardiac sarcoidosis or identified perioperative arrhythmias. Postoperative telemetry is appropriate for those with abnormal electrocardiogram, evidence of heart failure, or high-risk surgery. Infection surveillance is necessary as sarcoidosis is associated with higher rates of hospitalized infections (HR, 2.0) when compared to sex- and age-matched controls. Oral glucocorticoids were a significant predictor (HR, 3.03, less than 10 mg/day *vs.* HR, 4.48, greater than 10 mg/day).[47] Of note, untreated sarcoidosis patients also demonstrated a significantly higher rate of hospitalized infection. Higher incidence of postoperative respiratory insufficiency and failure were identified when compared to matched controls (10.5% *vs.* 5.1%), suggesting that biomarker surveillance (serum natriuretic peptide or high-sensitivity troponin) may be helpful to detect cardiovascular sequelae of perioperative volume overload.[48] Incentive spirometry and chest physiotherapy improve secretion mobilization and optimize known respiratory muscle weakness. Pain in sarcoidosis is multidimensional and is frequently compounded by chronic headache, osseous pain, and small-fiber neuropathy (neuropathic pain with allodynia), all of which can significantly complicate postoperative pain assessment and management.[49] Prioritize multimodal analgesia and regional techniques when feasible, particularly for thoracic or upper abdominal procedures, to minimize opioid-related respiratory compromise and improve quality of recovery.

Early mobilization remains paramount despite exercise limitations from cardiopulmonary involvement. Sarcoidosis is associated with an increased lifetime venous thromboembolism risk when compared to age- and sex-matched controls (HR, 2.73; risk factors: thrombophilia, Black race, steroid use).[50] Perioperative thromboprophylaxis with mechanical and pharmacologic methods is mandatory given the inflammatory state and immobility risk. Sarcoidosis-focused early postoperative complications include surgical stress–related disease exacerbation, frequently manifesting as worsening respiratory symptoms, radiographic infiltrates, or new/worsening extrapulmonary involvement. Focus should remain on clinical monitoring with early subspecialty consultation. Procalcitonin and other biomarkers are generally viewed as supportive within a broader diagnostic framework that integrates imaging, microbiology, and clinical course, rather than a stand-alone discriminator between infectious and noninfectious respiratory complications. Steroid augmentation (usually prednisone 20 mg/day) may be necessary for pulmonary exacerbation management and has been found noninferior to higher doses.[51] Neurosarcoidosis and cardiac sarcoidosis exacerbations may require much higher steroid and early initiation of steroid-sparing therapies.

Subspecialty Coordination: When Should Multidisciplinary Consultation and Long-term Follow-up Occur?

Multidisciplinary consultation should be framed by surgical risk (cardiac, thoracic, major vascular) and occur sufficiently in advance (4 to 6 weeks) to permit diagnostic workup, treatment optimization, and ICD placement if indicated. Cardiology consultation is recommended for patients with known or suspected cardiac sarcoidosis, given that advanced imaging is more effective in identifying subclinical disease than clinical assessment alone.[35] Pulmonary medicine consultation should occur for patients with FVC less than 60% predicted, DLCO less than 60%, features of advanced pulmonary fibrosis, or supplemental oxygen requirement.[8,30]

Optimal long-term postoperative recovery may require subspecialty coordination. Surgical stress may unmask subclinical organ involvement. A low threshold for repeat PFT and/or cardiac assessment at 4 to 6 weeks should be considered, particularly for patients with evidence of extrapulmonary involvement, new cardiorespiratory symptoms, or an increase in baseline oxygen requirements is observed. Repeat cardiac assessment should be considered if new arrhythmias, conduction abnormalities, heart failure symptoms, or unexplained hemodynamic instability occur postoperatively. Postoperative surveillance is focused on clinical evaluation. While biomarkers (angiotensin-converting enzyme, interleukin-2) have limited diagnostic utility, they may be useful for monitoring disease activity in individual patients trended over time. Patients with advanced pulmonary sarcoidosis with fibrosis, bronchiectasis, or PH should receive particular attention.

What Are the Critical Evidence Gaps and Future Directions?

Rare diseases present unique perioperative challenges for perioperative physicians, highlighted by heterogeneous disease presentation and limited outcome data.[48] Sarcoidosis research priorities include multicenter prospective trials and registries to develop validated risk prediction models and

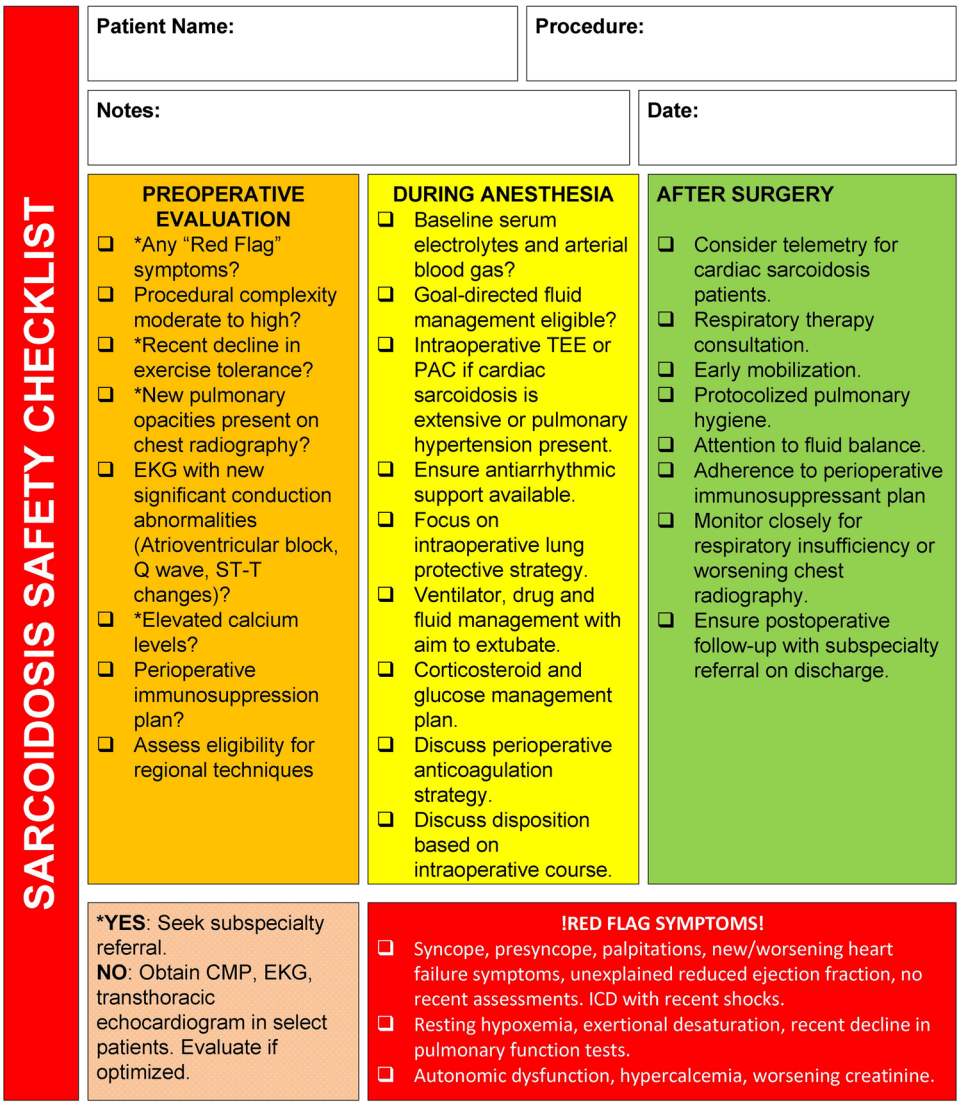


Fig. 4. Perioperative management framework for patients with sarcoidosis. These recommendations emphasize comprehensive preoperative assessment, focused intraoperative management, and enhanced postoperative care with modifications for organ-specific involvement. CMP, comprehensive metabolic panel; EKG, electrocardiogram; ICD, implantable cardioverter-defibrillator; PAC, pulmonary artery catheter; TEE, transesophageal echocardiography.

improved biomarker data to enhance postoperative surveillance. Implementation strategies should leverage electronic health record alert systems to identify high-risk patients and incoporate targeted educational initiatives targeting perioperative teams.

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