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Culture-Negative Cystitis With Multiorgan Dysfunction and Septic Shock After Intravesical Bacillus Calmette-Guérin (BCG) Therapy: A Case Report and Review of the Literature

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Abstract

Intravesical Bacillus Calmette-Guérin (BCG) therapy is a live attenuated strain of *Mycobacterium bovis* (*M. bovis*), used in the treatment of non-muscle invasive bladder cancer (NMIBC) after transurethral resection of bladder tumor (TURBT) as adjuvant therapy. Complications of intravesical BCG therapy range from chemical cystitis to disseminated BCG infection. We present a case of an 85-year-old man undergoing intravesical BCG therapy post-TURBT for NMIBC who was initially admitted for complicated chemical cystitis with progression to multiorgan failure and death over a four-week hospitalization. Treatment for systemic BCG infection with antimycobacterial agents and corticosteroids was not initiated until day 12 of hospitalization due to a clinical picture muddled by the patient's comorbidities, the rarity of disseminated BCG, and waxing and waning clinical responses to therapies over the course of the patient's hospitalization. Early recognition and timely treatment are necessary to prevent serious complications or death, as systemic BCG infection can both progress rapidly and respond slowly to treatment. However, the diagnosis of BCG infection/inflammatory response can be difficult as it is often a diagnosis of exclusion and presents with nonspecific diagnostic findings. This case highlights that to optimize patient outcomes, clinicians need a high index of suspicion and should consider early treatment for systemic BCG reaction in patients with multiorgan dysfunction and a history of intravesical BCG treatment, especially in those with negative microbiological data.

Categories: Oncology, Pulmonology, Internal Medicine
Keywords: bcg, bcg immunotherapy, bcg-induced complications, bcgitis, diagnosis of exclusion, disseminated bcg-osis, intravesical bcg therapy, non-muscle invasive bladder cancer, systemic bcg infection

Introduction

For patients with intermediate- to high-grade non-muscle invasive bladder cancer (NMIBC), after transurethral resection of bladder tumor (TURBT), they are treated with Bacillus Calmette-Guérin (BCG) adjuvant therapy, which has been shown to reduce the recurrence and progression of disease [1]. Intravesical BCG instillations are thought to treat NMIBC through granulomatous T-cell-activated inflammation with cytokine cascades that lead to tumor cell death [2,3]. Occurring in over 80% of patients, the most common complication of this therapy is chemical cystitis, which may present with low-grade fever and malaise and typically self-resolves within 48 hours [3,4]. Other complications of intravesical BCG include bacterial cystitis, miliary mycobacterial infection, localized BCG infection, systemic BCG hypersensitivity reaction, and disseminated BCG infection [4,5]. There is still debate over whether systemic reaction to intravesical BCG therapy (BCGitis) is caused by hematogenous spread and disseminated BCG infection versus a type IV hypersensitivity reaction to the BCG therapy. Likely, this is a spectrum of pathologies, primarily with evidence of hypersensitivity with granulomatous inflammation in earlier disease presentation, compared to greater microbiological evidence of active infection in later disease presentation [2,3,6,7].

Though likely underreported, local and systemic BCG infections occur in about 3% of patients treated with intravesical BCG as adjuvant therapy for NMIBC [6]. Risk factors for developing BCGitis include urothelial damage, elderly age, diabetes mellitus, immunosuppression, and genetic susceptibility [2,3,7,8]. Localized infections remain confined to the genitourinary system, though we still do not have standardized diagnostic criteria for systemic BCGitis [1,2]. Typically, criteria include clinically active tuberculosis-like symptoms (fever of 39℃ for more than 48 hours, night sweats, and chills) or infection of a specific organ (e.g., hepatitis and pneumonitis) following at least one intravesical BCG instillation in the absence of other confirmed diagnoses, in addition to a newly positive tuberculin skin test and/or positive PCR or culture for *Mycobacterium tuberculosis* complex and/or histopathological evidence of mycobacterial infection (inflammation with granulomas in biopsy specimens) [1].

Systemic BCGitis can present either early (days to weeks) or late (months to years) after the last intravesical

BCG instillation. Early presentation is associated with a cluster of granulomatous inflammation of the liver, lungs, and bone marrow. Late presentation is associated with granulomatous inflammation of the muscles, bones, and vasculature. Diagnosis is often difficult as it relies heavily on clinical suspicion, often with nonspecific imaging findings indistinguishable from other inflammatory pathologies and microbiological data with low sensitivity [3]. Patients can present with systemic illness and signs of end-organ dysfunction. Diagnosis can sometimes be confirmed through biopsies of affected organs with positive microbiological data present in roughly half of samples [6].

We present a case of systemic BCGitis where the diagnosis was one of exclusion and, despite the start of appropriate treatment, the patient succumbed to his illness. Diagnosis was subsequently confirmed microbiologically on autopsy. This report emphasizes the importance of the early recognition of potential BCGitis pathology in high-risk patients and encourages the consideration of early treatment initiation for BCGitis when it remains a plausible differential diagnosis.

Case Presentation

History

An 85-year-old man with a past medical history of Lewy body dementia with parkinsonism complicated by autonomic dysfunction (typically oriented to person, place, and date), paroxysmal atrial fibrillation, coronary artery disease, idiopathic interstitial lung disease (ILD) not on home oxygen, and bladder cancer status post-TURBT procedure seven months prior, now undergoing intravesical BCG treatment, presents with fever, altered mental status, and gross hematuria. The patient received his last BCG treatment instillation, session 3 of cycle 2, the day prior to presentation, with reported mucosal trauma. In the 24 hours since instillation, the patient developed passage of large blood clots and dysuria with clean intermittent catheterization (CIC), worsening confusion and balance, and low-grade fevers to 37.9℃. He was noted to have had worsening bladder irritation with subsequent BCG treatments.

Initial physical examination

On presentation, the patient was afebrile with oral temperature of 37.3℃, heart rate of 86 beats/minute, respiratory rate of 16 breaths/minute, blood pressure of 81/39 mmHg, with oxygen saturation of 93% on 4 L nasal cannula. He was ill-appearing, flushed, spontaneously opening his eyes but minimally interactive, and with rigors. His chest was clear to auscultation bilaterally, without respiratory distress, and his abdomen was nontender, without costovertebral angle tenderness. Initial diagnostics are detailed in Table *1*.

Laboratory Parameters	Result	Normal Range
White blood cell (WBC)	17.4 × 1000/uL with 90.8% polymorphonuclear leukocytes	3.9-10.6 × 1000/uL
Hemoglobin	10.5 g/dL	13.3-17.7 g/dL
Platelets	169 × 1000/uL	150-400 × 1000/uL
Lactate	2.5 mmol/L	0.5-2 mmol/L
Sodium	134 mmol/L	136-145 mmol/L
Bicarbonate (CO_2)	16 mmol/L	23-31 mmol/L
Blood urea nitrogen (BUN)	38 mg/dL	8-26 mg/dL
Creatinine (Cr)	2.09 mg/dL	0.6-1.3 mg/dL
Aspartate aminotransferase (AST)*	96 U/L	11-34 U/L
Alanine aminotransferase (ALT)*	74 U/L	0-45 U/L
Alkaline phosphatase (ALP)*	96 U/L	40-150 U/L
Total bilirubin*	0.8 mg/dL	0.2-1.0 mg/dL
Urinalysis	Hazy, 3+ leukocyte esterase, 100 mg/dL protein, 2+ blood, >100 WBC/HPF, and rare bacteria	Clear, zero leukocyte esterase, 0 mg/dL protein, zero blood, zero WBC/HPF, and no bacteria
Urine culture	No growth	No growth
Blood cultures including acid-fast bacilli (AFB)	No growth for six weeks	No growth

TABLE 1: Initial laboratory values after admission.

Laboratory values obtained on day 1 of hospitalization, except as noted.

*Values obtained on day 3 of hospitalization; no admission values available for reference.

HPF: high-power field

Hospital course

The patient was admitted for hypotension and sepsis secondary to suspected acute complicated cystitis in the setting of traumatic intravesical BCG therapy and was treated with fluid resuscitation and antibiotics for a total of five days for suspected non-BCG, bacterial urinary tract infection with empiric coverage for possible community-acquired pneumonia. Urine cultures remained negative, and given the high incidence of chemical cystitis in those treated with intravesical BCG, the working diagnosis shifted to BCG hypersensitivity reaction with chemical cystitis and sepsis-like reaction, and antibiotics were discontinued on day 5 due to concern for drug rash. The patient's mentation, respiratory status, and balance improved; however, he developed intermittent spiking fevers to 39.2℃ and had worsening hypoxic respiratory failure requiring escalation to bilevel positive airway pressure (BiPAP) support and transfer to the medical intensive care unit (ICU) on day 6 of hospitalization. Imaging obtained (Figure *1*) was consistent with multifocal pneumonia, and the patient was started on empiric broad-spectrum antibiotics with new vasopressor requirements on day 7 of hospitalization, which were weaned off by day 8.

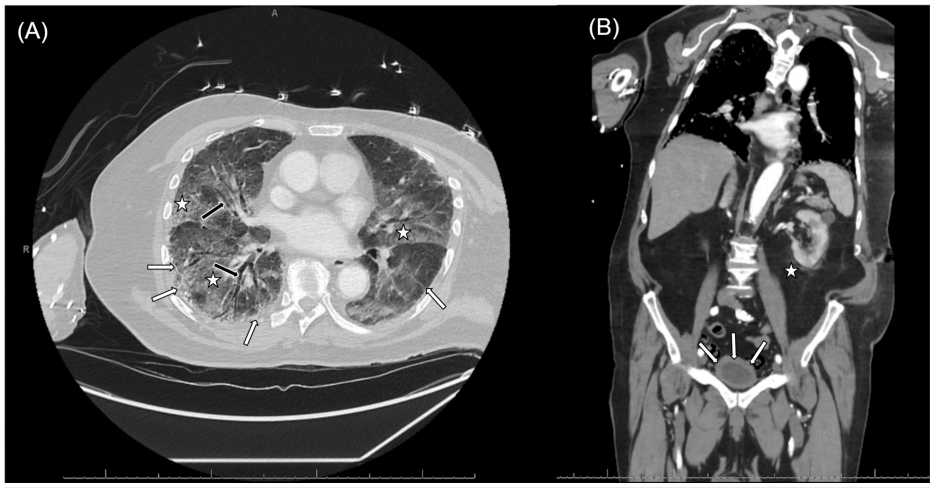


FIGURE 1: Non-contrast enhanced CT scan of the chest, abdomen, and pelvis from day 6 of hospitalization.

(A) Thoracic axial image with (white stars) nonspecific ground-glass opacities and (white arrows) subpleural reticular changes and (black arrows) lobar bronchiectasis, findings consistent with the patient's known ILD and probable usual interstitial pneumonia (UIP) but without typical honeycombing; superimposed pneumonia not excluded. (B) Abdominal coronal image with (white stars) nonspecific perinephric edema and (white arrows) concentric bladder wall thickening concerning for cystitis. Of note, there is no identified liver or gallbladder pathology despite transaminitis noted on day 3 of hospitalization.

ILD: interstitial lung disease

Cardiac evaluation with transthoracic echocardiogram revealed concentric left ventricular hypertrophy with preserved ejection fraction and reduced right ventricular systolic function and elevated pro-B-type natriuretic peptide (BNP). Vasopressors and respiratory support were initially weaned after starting diuresis and antibiotics. Despite achieving apparent euvolemia, by day 12 of hospitalization, the patient redeveloped hypoxic respiratory failure and rising transaminitis; aspartate aminotransferase (AST), 177 U/L; alanine aminotransferase (ALT), 194 U/L; alkaline phosphatase (ALP), 312 U/L; and total bilirubin, 1.7 mg/dL. Liver ultrasound was significant for slightly increased echogenicity secondary to hepatic steatosis or other cause of hepatitis.

Due to his continued decline, the patient was initiated on therapy for presumed BCGitis with isoniazid, rifampin, ethambutol, and stress-dose corticosteroids on day 12 of hospitalization. Due to his respiratory failure and tenuous clinical status, it was unsafe to obtain bronchoscopy with biopsy specimens or liver biopsy for microscopic analysis. Repeat CT of the chest was performed and revealed worsening patchy ground-glass opacities and subpleural reticular changes suggestive of underlying chronic interstitial lung disease with worsened areas of consolidation concerning for superimposed pneumonia or inflammatory changes (Figure *2*).



FIGURE 2: Non-contrast enhanced chest CT scan from day 18 of hospitalization.

Findings include (white stars) nonspecific ground-glass opacities (white arrows), redemonstrated subpleural reticular changes (black stars), and consolidative opacities in the bilateral lung parenchyma with visible air bronchograms. These CT findings are largely nonspecific and can be associated with infectious or inflammatory lung pathologies superimposed on ILD, contributing to the diagnostic challenge of this case.

ILD: interstitial lung disease

Due to persistent oxygen requirements, treatment was escalated to pulse-dose methylprednisolone on day 22 with the intention to treat ILD flare, as well as BCGitis. Unfortunately, due to the difficult hospital course and clinical deterioration, the patient ultimately transitioned to hospice care and subsequently expired on hospital day 29 (Figure *3*). Given that the diagnosis was not definitive and the patient experienced clinical deterioration despite antimycobacterial medication and corticosteroids, autopsy was performed. Nonnecrotizing granulomatous inflammation was identified within the lungs, liver, and prostate gland, with additional areas of necrotizing granulomatous inflammation in the prostate. Acid-fast bacillus (AFB)-positive organisms were identified within the area of necrotizing granulomatous inflammation in the prostate gland, findings consistent with a unifying diagnosis of systemic BCGitis.

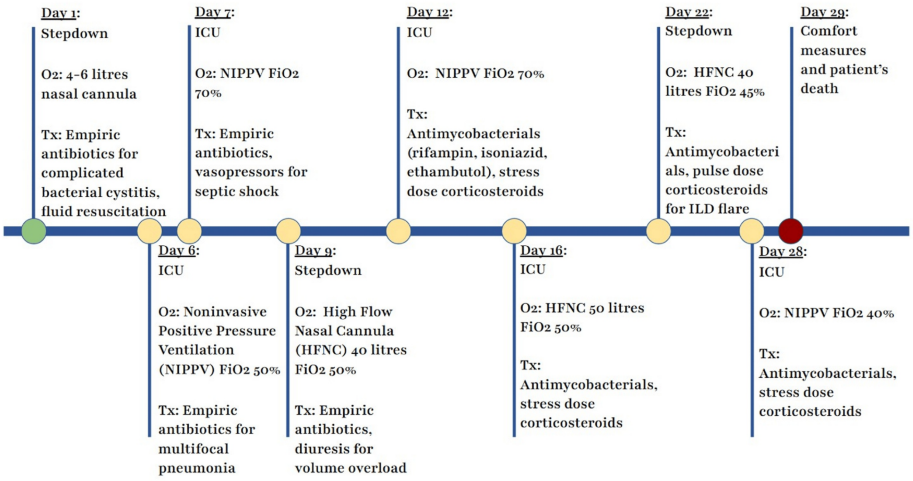


FIGURE 3: Hospital course timeline.

Hospital course including patient location, oxygen requirements (O_2), and treatments (Tx).

ILD, interstitial lung disease; FiO_2, fraction of inspired oxygen; ICU, intensive care unit

Discussion

Systemic BCGitis is a rare but serious complication of intravesical BCG therapy that can present nonspecifically and mimic a patient's comorbidities and sequelae of critical illness. This case is unique due to the diagnostic uncertainty that plagued this patient's hospital course, as well as his initial presentation with sepsis, with sepsis only reported in 0.4% of patients treated with intravesical BCG [9]. The most common complication of intravesical BCG is bladder irritation, affecting up to 90% of patients [4,9]. Initially, our patient presented with expected bladder irritation, low-grade fevers, and sterile urine, most consistent with complicated chemical cystitis, which does not typically require targeted treatment [10]. However, his clinical picture gradually evolved to disseminated organ involvement of the lungs and liver with septic shock.

In one systematic review, around 72% of patients who developed BCGitis were found to have single organ system involvement, with about 28% having two or more affected organs [6]. Most commonly, the sites affected by systemic BCGitis are the lungs, vasculature, liver, bones, and joints, with other sites including musculature and bone marrow (Table *2*) [6,11,12]. Localized genitourinary BCGitis can involve the testis, kidney, penis, and prostate [11]. The top three organ systems affected by BCGitis are the lungs, vasculature, and liver, with 4% mortality for patients with lung and/or liver involvement and up to 17% mortality for those with vascular involvement [6,12]. Cutaneous rashes are uncommon for systemic BCGitis, but there have been case reports of skin involvement presenting as palpable purpura and violaceous patches found to have noncaseating granulomas on biopsy [13].

	Organ	Rate of Involvement (%)	Early Versus Late Presenting	Rate of Microbiological Detection of BCG (%)	Death Due to BCGitis Complications (%)
Localized BCGitis	Kidney	21	Early	30	0
	Penis	22	Early	29	0
	Prostate	20	Early	61	0
	Testis	24	Late	58	0
Systemic BCGitis	Bone marrow	24	Early	48	0
	Liver	56	Early	32	4
	Lungs	73	Early	35	4
	Muscular	21	Late	100	5
	Osteoarticular	41	Late	92	0
	Vascular	65	Late	84	15-17

TABLE 2: Features of patients with BCG infection based on organ involvement.

Localized BCGitis remains confined to the genitourinary system, while systemic BCGitis involves one or more non-genitourinary organs. Each affected organ has varying rates of involvement, time of presentation, positive microbiological testing, and associated mortality. Early presenters typically develop symptoms one to six weeks after the last BCG instillation, while late presenters typically develop symptoms in one to two years.

BCG: Bacillus Calmette-Guérin

The rate of disability after BCGitis has been reported in 7% of patients, most commonly including the loss of visual acuity, chronic arthralgias, and end-stage renal disease [13]. Mortality risk is higher in those older than 65 years and, expectedly, in those with systemic BCGitis compared to localized [12]. However, BCGitis is rare, and there is likely underreporting of cases, making it difficult to truly estimate morbidity and mortality, with multiple single-institution studies suggesting that overall mortality may be as high as 10%-17%, with vascular involvement carrying the highest mortality risk, as high as 17.5% in one Finnish national register study [11,12].

In this patient's case, his original presentation was within 24 hours of his last BCG instillation, and during his four-week hospitalization, he developed inflammatory sequelae in both his lungs and liver, consistent with the early-presenting systemic BCGitis. Diagnosis remained difficult due to patient instability preventing biopsy. When biopsies of affected organs can be obtained, pathology reveals granulomas with or without caseous necrosis in upward of 60% of samples [6]. Acid-fast stain, tissue culture, and mycobacterial PCR can also confirm diagnosis; however, mycobacteria are only found in about 50% of samples and are more likely to be negative the more recent the last BCG instillation was, making the diagnosis of BCGitis often one of exclusion [4,6,9]. PCR appears to be the most sensitive microbiological test, reported to be positive in about 41% of samples from patients diagnosed with BCGitis in a single-institution review [11]. Microbiological diagnoses in patients with BCGitis also vary depending on the organ biopsied, with the lung and liver being some of the lower-yield sites with only about one in three samples with microbiological positivity, compared to samples from vascular or osteoarticular involvement with 80%-90% positivity (Table *2*) [6]. Overall, those that present with the late symptomatic cluster of vascular and musculoskeletal involvement are more likely to yield a positive microbiological diagnosis when biopsied [6].

There are limited studies to guide the optimal management of systemic BCGitis, though recommended treatment includes a combination of antimycobacterial medications and corticosteroids, thought to treat both components of infection and hypersensitivity reaction [4,6]. For localized BCG infections within the urinary tract, corticosteroids are not necessary, and patients can often be managed with antimycobacterial medications alone. However, for systemic BCGitis, corticosteroids are recommended and should be gradually tapered to prevent recurrence and granuloma formation, though exact duration, agents, and dosages are not defined [2,9,11]. The literature reports the use of varying antibiotic regimens including single, double, or triple therapy combinations of isoniazid, rifampin, and ethambutol, with varying courses from three to 13 months [3,6,8,11,14,15]. Reportedly successful treatment regimens have included three months of isoniazid for localized BCGitis versus isoniazid plus rifampin for six months with two induction months including ethambutol or triple therapy for the full six months of treatment for systemic BCGitis [11,14]. For BCG sepsis, the European Association of Urology (EAU) recommends the most commonly utilized regimen of isoniazid, rifampin, and ethambutol for six months [4,10,15]. Additionally, the Connaught BCG strain is

susceptible to fluoroquinolones, with case reports of the successful treatment of BCGitis of this strain with ofloxacin, levofloxacin, ciprofloxacin, or moxifloxacin [11]. Neither pyrazinamide nor cycloserine should be used for antimycobacterial treatment as Mycobacterium bovis (*M. bovis*) has known resistance to both medications [6,11]. Ultimately, therapy should be individualized to the patient based on organ involvement, severity, and the persistence of symptoms during therapy [10,14]. Particularly for systemic BCGitis, treatment should not be delayed while awaiting diagnostics due to the severity of disease, risk of morbidity and mortality, and poor microbiological testing sensitivity.

In this case, alternative diagnoses were initially considered due to the rarity of BCGitis, apparent response to therapies, and clinical presentation muddled by the patient's known ILD. BCGitis was considered later in this patient's course when he continued to deteriorate despite appropriate empiric management. It is difficult to determine if the earlier initiation of treatment would have resulted in a different outcome given his difficult hospital course, deconditioning, and other comorbidities. Generally, the early-phase inflammatory response to intravesical BCG occurs within hours to days to weeks, which raises suspicion that appropriate early treatment may be beneficial for long-term outcomes, consistent with the EAU's recommendation for early corticosteroids for BCG sepsis [10].

Whether or not developing BCGitis is a contraindication to resuming BCG intravesical therapy is debated, with some suggesting that for local or mild BCGitis, instillations can be resumed upon treatment completion. Still, most researchers support the discontinuation of BCG therapy for those who develop systemic BCGitis, but further studies documenting long-term outcomes in these patients are needed [2,10,11].

Conclusions

This case underscores the critical need for a high index of suspicion for BCGitis in patients who present with multiorgan failure with a history of intravesical BCG treatment, particularly with culture-negative diagnoses, given the poor reliability of microbiological data in early BCGitis. It also highlights the importance of clinician familiarity with both early and late symptomatic clusters of BCGitis in order to differentiate from inflammatory etiologies or chronic condition exacerbations. Additionally, despite appropriate therapy for over two weeks, the patient succumbed to his illness after just one month from symptom onset. To prevent delayed diagnosis, systemic BCGitis and involved organ biopsy should be considered early in patients with unexplained multiorgan dysfunction following intravesical BCG therapy.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Layla Annous

Acquisition, analysis, or interpretation of data: Layla Annous, Victoria Lui, Andrew Deitchman

Drafting of the manuscript: Layla Annous, Victoria Lui, Andrew Deitchman

Critical review of the manuscript for important intellectual content: Layla Annous, Victoria Lui, Andrew Deitchman

Supervision: Layla Annous

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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