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Your Next Celebration at US National Parks: A Case Emphasizing Lyme Neuroborreliosis (Bannwarth Syndrome)

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Your Next Celebration at US National Parks: A Case Emphasizing Lyme Neuroborreliosis (Bannwarth Syndrome)

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Abstract

Introduction:

Bannwarth syndrome is synonymous with Lyme neuroborreliosis. The neurological component consists of neuropathy, radicular pain, and lymphocytic pleocytosis. This is due to inflammation of the nerve fibers by *Borrelia Burgdorferi*. *Borrelia Borgdorferi* is a spirochete bacteria transmitted hematogenously from the Ixodes tick vector to the human host. The commonly known Lyme disease is diagnosed with symptoms of arthritis, heart block, and integumentary signs. Lyme disease can present as early disseminated or late manifestations of disease. Bannwarth syndrome takes it a step further and emphasizes the neurologic sub set of symptoms. The Centers for Disease Control and Prevention (CDC) estimates about 300,000 new cases annually. It is the triad of neuropathy, radicular pain, and lymphocytic pleocytosis caused by immune inflammation of nerve fibers.

Case Presentation:

A 73 year old gentleman with a past medical history notable for stage 2 chronic kidney disease and adenocarcinoma of the prostate status post prostatectomy presenting to the emergency department with radicular left sided chest discomfort. It was waxing and waning, unilateral, and radiating to his left upper back for the past one month. For the last 2 weeks leading to this presentation, he had accompanying numbness and tingling in his hands. Upon discussion, he resides in the countryside and spends a great deal of time outdoors during the fall season. His favorite hobbies include collecting tractors and walking his pets.

The 2nd case involves a 24 year old gentleman without significant past medical history presented to the hospital emergency room complaining of progressive upper torso pain and extremity paresthesia for the past month. Developed extremity paresthesias in the forearms along with blurry vision in the right eye. The diagnosis of Disseminated Lyme Infection was made with positive IgM and IgG tests for Lyme Bands. 8 weeks of intravenous ceftriaxone with continued infusion in the outpatient setting was completed. The erythematous migratory rashes disappeared over the following week, and the neuropathies and radicular pain steadily decreased until they disappeared. His EKG reverted back to a normal sinus rhythm without further complications.

Results:

A positive IgM is reported when any 2 or more are present: 23, 39, or 41 kDa. This positive antibody test warranted Lyme Western Blot test, which was positive for *Borrelia Burgdorferi*. He was treated with a 14 day course of doxycycline. After the full course of doxycycline, the chest discomfort resolved. His subsequent clinical course did not show any further complications.

Conclusion:

The purpose of this case report is to spotlight the neurologic findings in Bannwarth syndrome. The neurological symptoms that warrant thorough history for Bannwarth Syndrome include radicular pain, neuropathies, and facial/ocular palsy. It is important to differentiate lyme disease from Bannwarth syndrome because the neurologic symptoms dictate duration of treatment. Compared to the common 5

to 7 day course treatment of Lyme's disease, Bannwarth Syndrome requires at least 3 weeks of treatment. The neurologic physical exam should be included during lyme disease for suspicion of Bannwarth and to broaden the differential. As physicians, we should interrogate these neurologic manifestations and begin early intervention to prevent further complications.

Keywords

Lyme, Lyme Neuroborreliosis, National Park

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Conflict of Interest Statement

The authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest

CASE REPORT

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Introduction: Bannwarth syndrome is synonymous with Lyme neuroborreliosis. The neurological component consists of neuropathy, radicular pain, and lymphocytic pleocytosis. This is due to inflammation of the nerve fibers by *Borrelia burgdorferi*. *Borrelia burgdorferi* is a spirochete bacteria transmitted hematogenously from the Ixodes tick vector to the human host. The commonly known Lyme disease is diagnosed with symptoms of arthritis, heart block, and integumentary signs. Lyme disease can present as early disseminated or late manifestations of disease. Bannwarth syndrome takes it a step further and emphasizes the neurologic sub set of symptoms. The Centers for Disease Control and Prevention (CDC) estimates about 300,000 new cases annually. It is the triad of neuropathy, radicular pain, and lymphocytic pleocytosis caused by immune inflammation of nerve fibers.

Case presentation: A 73 year old gentleman with a past medical history notable for stage 2 chronic kidney disease and adenocarcinoma of the prostate status post prostatectomy presenting to the emergency department with radicular left sided chest discomfort. It was waxing and waning, unilateral, and radiating to his left upper back for the past one month. For the last 2 weeks leading to this presentation, he had accompanying numbness and tingling in his hands. Upon discussion, he resides in the countryside and spends a great deal of time outdoors during the fall season. His favorite hobbies include collecting tractors and walking his pets.

The 2nd case involves a 24 year old gentleman without significant past medical history presented to the hospital emergency room complaining of progressive upper torso pain and extremity paresthesia for the past month. Developed extremity paresthesias in the forearms along with blurry vision in the right eye. The diagnosis of Disseminated Lyme Infection was made with positive IgM and IgG tests for Lyme Bands. 8 weeks of intravenous ceftriaxone with continued infusion in the outpatient setting was completed. The erythematous migratory rashes disappeared over the following week, and the neuropathies and radicular pain steadily decreased until they disappeared. His EKG reverted back to a normal sinus rhythm without further complications.

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Conclusion: The purpose of this case report is to spotlight the neurologic findings in Bannwarth syndrome. The neurological symptoms that warrant thorough history for Bannwarth Syndrome include radicular pain, neuropathies, and facial/ocular palsy. It is important to differentiate lyme disease from Bannwarth syndrome because the neurologic symptoms dictate duration of treatment. Compared to the common 5 to 7 day course treatment of Lyme's disease, Bannwarth Syndrome requires at least 3 weeks of treatment. The neurologic physical exam should be included during lyme disease for suspicion of Bannwarth and to broaden the differential. As physicians, we should interrogate these neurologic manifestations and begin early intervention to prevent further complications.

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1. Introduction

Bannwarth syndrome is synonymous with Lyme neuroborreliosis. The neurological component consists of motor weakness, facial nerve palsy and peripheral radiculopathy. This is due to inflammation of the nerve fibers by *Borrelia burgdorferi*. *B. burgdorferi* is a spirochete bacteria transmitted hematogenously from the *Ixodes* tick vector to the human host.¹ The commonly known Lyme disease is diagnosed with symptoms of arthritis, heart block, and integumentary signs. The Centers for Disease Control and Prevention (CDC) estimates about 300,000 new cases annually. Lyme disease can present as early disseminated or late manifestations of disease. Bannwarth syndrome takes it a step further and emphasizes the neurologic sub set of symptoms. It is the triad of neuropathy, radicular pain, and lymphocytic pleocytosis caused by immune inflammation of nerve fibers.²

We present 2 cases of Bannwarth Syndrome in the inpatient setting. The first case explores a 73 year old gentleman presenting with radicular chest pain. The second case is a 24 year old gentleman with disseminated Lyme significant for neurological sequelae.

2. Case presentations

2.1. Case 1

The first case involves a 73 year old male with a past medical history notable for stage 2 chronic kidney disease and adenocarcinoma of the prostate status post prostatectomy presenting to the emergency department with radicular left sided chest discomfort. It was waxing and waning, unilateral, and radiating to his left upper back for the past one month. For the last 2 weeks leading to this presentation, he had accompanying numbness and tingling in his hands. Upon discussion, he resides in the countryside and spends a great deal of time outdoors during the fall season. His favorite hobbies include collecting tractors and walking his pets. Due to the unclear etiology of left sided chest discomfort, he was evaluated for cardiogenic, respiratory and musculoskeletal differentials. Upon investigation, no history of similar chest pain, no diaphoresis, no chest trauma, no smoking, no asthma, no allergies, no recent immobility or blood clots, and no gastroesophageal reflux. His vitals were non febrile at 98.0, sinus tachycardia at 110, respiratory rate of 20, normotensive at 110/80 and saturating 98% on room air.

Physical examination was notable for the patient being visibly anxious and uncomfortable. Cardiac and pulmonary examinations were normal and the

reported left-sided chest pain was non-reproducible with palpation. Extremity examination revealed no swelling or rashes. Neurological exam elicited normal reflexes and strength in all 4 extremities and normal cranial nerves, but bilateral paresthesias in the T4 dermatome were noted. His reported back pain was reproducible with palpation and increased his sensation of associated numbness and tingling at the hands and fingertips.

Evaluation for cardiac etiology of his chest pain was negative with troponin levels undetectable. Chest x ray showed no focal consolidation and was negative for pneumothorax, aortic dissection, or pleural effusion. Due to his history of outdoor activities, a thorough workup for causes of radicular chest pain were examined including, Lyme serology:

IgG Bands – Positive result is reported when any 5 or more are present (bolded are bands present): 18, 23, 28, 30, 39, **41**, 45, 58, 66, or 93 kDa.

IgM Bands – Positive result is reported when any 2 or more are present (bolded are bands present): 23, 39, **41** kDa.

This positive antibody test warranted Lyme Western Blot test, which was positive for *B. burgdorferi*. He was treated with a 14 day course of doxycycline. After the full course of doxycycline, the chest discomfort resolved. His subsequent clinical course did not show any further complications.

2.2. Case 2

A 24 year old male without significant past medical history presented to the hospital emergency room complaining of progressive upper torso pain and extremity paresthesia for the past month. One month prior, he was celebrating the 4th of July weekend in the campgrounds of a forested state park. After two weeks without any symptoms, his right hand started to hurt at the middle and ring finger distal interphalangeal joints. Over the next three days, the swelling spread to the proximal interphalangeal joints and inhibited his daily activities. He developed upper extremity paresthesias and unilateral blurry vision in the right eye. He was worried that the symptoms were worsening and pursued hospital care at the emergency department. His vitals were: Temp: 99.5 HR: 116 RR: 18 BP: 138/76 O2 Saturation: 96% room air. Chest x ray did not show any infiltrates or cardiopulmonary issues. On review of his chart, his vaccinations were up to date. Due to the lack of fever, white blood cell count, and upper respiratory infection symptoms, he was admitted for further work up. An erythematous migratory (EM) rash developed on the left upper back over the next 2 days.

Physical examination was notable for the patient being diaphoretic and uncomfortable. HEENT examination was notable for bilateral non-tender cervical lymphadenopathy and blurred vision in the right eye; however, pupils were equal and round and reactive to light and accommodation bilaterally. Cardiac and pulmonary examinations were normal. Extremity examination revealed no swelling or rashes. Neurological exam elicited normal reflexes and strength in all 4 extremities and normal cranial nerves, but paresthesia was noted during sensation testing of the C5 dermatome. Extremity examination was remarkable for paresthesias reported in the forearms bilaterally, with spread of the joint arthritis from the right ring and middle finger DIP to the right MIP and PIP joints and associated worsening swelling and redness. Also noted was asymmetric tenderness to grip strength in the right ring and middle fingers. Skin examination revealed 5 non-tender bulls-eye lesions of 6 cm diameter each on his back.

2.3. Labs revealed

WBC: 6.4 g/dl; Hgb: 14.8 g/dl; Platelets: 237 g/dl; Na: 139 meQ/L; K: 4.1 meQ/L; BUN: 13 meQ/L; Cr: 1.0 meQ/L.

IgG Bands – Positive result is reported when any 5 or more are present (bolded are bands present): 18, 23, 28, 30, 39, 41, 45, 58, 66, or 93 kDa.

IgM Bands – Positive result is reported when any 2 or more are present (bolded are bands present): 23, 39, 41 kDa.

Imaging of the right hand showed no acute abnormality or radiopaque osseous lesions. EKG showed 2nd degree AV block Mobitz type 1 with progressive lengthening of PR intervals and sudden dropping of QRS complexes. This finding resulted in the performance of a transthoracic echocardiogram which was normal. The diagnosis of Disseminated Lyme Infection was made with positive IgM and IgG tests for Lyme Bands. 8 weeks of intravenous ceftriaxone with continued infusion in the outpatient setting was completed. Toward the one month mark, the erythema migrans rash resolved. The neuropathies and radicular pain steadily decreased until they disappeared. At one month's follow up, repeat EKG showed normal sinus rhythm without further complications.

3. Discussion

Bannwarth Syndrome is caused by *B. Burgdorferi*, accounting for the majority of cases in North

America, and *Borrelia afzelii* and *Borrelia garinii* for cases in Europe and Asia.³ There are approximately a quarter of a million cases of Lyme disease diagnosed across the United States each year. Bannwarth syndrome is also known as Lyme neuroborreliosis and is considered the subset of neurologic manifestations of Lyme disease.³ Although the neurologic manifestations vary from person to person, the triad of painful radiculopathy, neuropathy, and lymphocytic pleocytosis are the most common presenting symptoms.⁴

The first description of Bannwarth syndrome was a case report from France that was authored by Garin and Bujadoux-Bannwarth in 1922.⁴ Dr. Bannwarth described the tick bite associated with meningoradiculitis as the most typical neurologic presentation in this disorder. Of note, patients with these neurologic symptoms were diagnosed with lumbar puncture to confirm the lymphocytic pleocytosis. 60 years later, Lyme arthritis was made a separate diagnosis caused by *B. Burgdorferi*.⁵ It took several years to broaden the term Lyme Arthritis to Lyme disease as a general term to involve all organ systems affected. In 1979, Reik and colleagues included the neurologic system into the discussion of Lyme disease which mimicked the neurologic description by Garin and Bujadoux-Bannwarth over 50 years ago.⁵ Bannwarth was finally recognized as an accepted diagnosis for these neurologic symptoms and lymphocytic pleocytosis. In 1983, the spirochete, *B. Burgdorferi*, was identified as the causative agent in American patients for Lyme disease.⁶

Radicular pain is one of the triad in Bannwarth Syndrome. The pain is initially felt between the scapula but can later involve other extremities. The pain can interfere with sleep and can cause hypo and hyperaesthesias.⁷ The burning radicular pain is exacerbated at night time secondary to painful skin dystrophy and direct injury to the sympathetic nerves.⁸ The resulting severe perivascularitis causes thrombosis of the external neural layer of the vasa nervorum is the radicular back pain characteristic in Bannwarth. For the gentleman in case one, the radicular pain traveled along the C6 and C7 dermatomes.

Neuropathy is a second symptom in the triad. Peripheral neuropathy is common and the origin of symptoms depends on the initial tick bite. At times, facial nerve palsies and ocular nerve palsies can occur. Cranial nerve involvement leads to facial nerve palsy in approximately 10% of patients.⁸ The closer the initial bite site is to the face, the greater likelihood of facial nerve involvement due to the

shorter distance to those nerves.⁸ Patients bitten in the head or neck manifest 13% more neurologic symptoms than those bitten peripherally.

Lymphocytic pleocytosis is the third symptom in the triad. It is defined as a leukocyte count greater than 5×10^6 cells/L and a protein concentration >45 g/L.⁹ The classic signs of meningitis including fever, vomiting and neck stiffness are only 20% of patients with Bannwarth CSF involvement.⁹ CSF abnormalities that are seen include protein elevation, relative IgG increase, and oligoclonal bands. A lymphocytic pleocytosis with plasma cells and occasional granulocytes were regularly found.⁹

The diagnosis of Bannwarth Syndrome involves both clinical investigation and objective findings to support the differential. Clinical criteria involve geographical areas with high populations of the *Borrelia* organisms, radicular pains, facial or ocular nerve palsies, or CSF involvement. There are no sensitivity or specificity criteria established as of yet for interpretation of banding patterns in CSF.¹⁰ Due to the possibility of passive immunoglobulin diffusion across the CSF barrier or the presence of serum antibodies in CSF as a result of a traumatic lumbar puncture, detection of antibodies does not solely confirm diagnostic *B. Garinii*.¹⁰ Therefore, clinical skills are the leading method for diagnosis at this time.

The differential includes autoimmune and other infectious causes. Guillain-Barre syndrome can be dominated by severe back and limb pain. Facial diplegia is seen in at least half of the cases. It is a postinfectious autoimmune demyelinating disorder localized to the peripheral nerves.¹¹ The difference is the ascending manner of the paralysis in Guillain Barre rather than the asymmetric and localized neuropathies in Bannwarth.¹¹ In addition, poliomyelitis falls in the differential of asymmetric nerve pain. The difference is that polio is more likely to have gastrointestinal symptoms, fever and an unvaccinated history.¹¹ Similarly, infections with other neurotropic viruses such as Echovirus and Coxsackievirus are less likely as there are gastro-intestinal symptoms or pleurodynia.¹¹

A major complication associated with Bannwarth includes heart block. The nervous supply of the myocardium is directly affected by inflammation of the His-Purkinje bundle.¹² All types of atrioventricular blocks are a possibility, but progression of Lyme disease correlates with the severity of initial atrioventricular block. Type 2 atrioventricular block tends to progress to Type 3 complete dissociation between the atria and ventricles when sufficient treatment is not administered.¹²

Treatment is defined as Ceftriazone 2 g daily IV for disseminated infections and oral doxycycline

100 mg twice a day for localized infections were started at the same time.¹² Recovery is measured over time based on resolution of neurologic symptoms. The antibiotics destroy the *Borrelia* organisms and steadily decrease the neural tissue inflammation. Worsening of symptoms warrants specialist management and longer hospital courses. The ultimate goal of treatment on the heart is to stop 2nd degree heart block from converting into complete dissociation of atria and ventricles.¹² The short term treatment for dissociated heart block and type 2 block is temporary pacing.

4. Conclusion

The purpose of this case report is to spotlight the neurologic findings in Bannwarth syndrome. The neurological symptoms that warrant thorough history for Bannwarth Syndrome include radicular pain, neuropathies, and facial/ocular palsy. It is important to differentiate Lyme disease from Bannwarth syndrome because the neurologic symptoms dictate duration of treatment. Compared to the common 5–7 day course treatment of Lyme's disease, Bannwarth Syndrome requires at least 3 weeks of treatment. The neurologic physical exam should be included during lyme disease for suspicion of Bannwarth and to broaden the differential. As physicians, we should interrogate these neurologic manifestations and begin early intervention to prevent further complications.

Conflict of interest

The authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest.

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