time, as was the case with our patient.

It could be hypothesized that the duration and severity of untreated hypothyroidism results in mitochondrial changes that respond less completely or less permanently. Age may also be a factor in the response obtained, as the patients in the study by Hagspiel and coworkers averaged 37 years of age and our patient with an unsustained response was 57 years old. Sustained metabolic responsiveness to thyroid hormone may decrease with age.

A number of questions remain unanswered regarding the exact effect of hypothyroidism on phosphorus metabolism in muscle. Clearly, hypothyroidism impairs the bioenergetic state of striated muscle, with changes occurring that may be monitored by ^{31}P-NMR spectroscopy and nerve conduction/EMG studies. These changes appear to be completely reversible in some cases and only temporarily or partially reversible in others. Sufficient research has not yet been done to ascertain the full nature of these implications.

Comment

Phosphorus 31–NMR spectroscopy and nerve conduction/EMG studies provide excellent methods for monitoring changes in muscle tissue in severely hypothyroid patients, both diagnostically and for determining the degree of response to thyroid hormone supplementation. The basic molecular mechanisms responsible for these changes remain unclear, as do the temporal patterns of change in different aspects of thyroid disease. Additional studies of ^{31}P-NMR spectroscopy and nerve conduction/EMG in different types of hypothyroid patients at various stages of treatment are needed to further elucidate mechanisms of the disease.

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Case reports continued

Churg-Strauss syndrome

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The neurologic manifestations of systemic necrotizing vasculitis are heterogeneous and often complex. Late onset asthma, systemic vasculitis manifest by mononeuritis multiplex, and peripheral eosinophilia—the triad of which is consistent with Churg-Strauss syndrome—developed in a 59-year-old woman. Electromyography and nerve conduction studies showed both myelinating and axonal neuropathy involving multiple nerves. Muscle and nerve biopsy showed neurogenic atrophy, demyelination, and axonal changes involving the sural nerve and inflammatory necrotizing small and medium vessel arterial changes. The patient was treated with high-dose prednisone and improved rapidly. Unusual in this case was the lack of multisystemic involvement. Churg-Strauss syndrome is a disease that is being recognized with increased clinical frequency and responds quickly to methylprednisolone treatment.

(Key words: Churg-Strauss syndrome, vasculitides, eosinophilia)

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Churg-Strauss syndrome (CSS) is a form of necrotizing vasculitis categorized under the polyarteritis nodosa group of systemic vasculitides, which also includes polyarteritis nodosa, polyarteritis nodosa of children and infants, an overlap syndrome, and Kawasaki syndrome.[1] This type of vasculitis affects small and medium arteries—as well as arterioles, capillaries, and venules—of multiple organ systems, leading to its clinical manifestations.[1-4] In the prodromal phase, patients usually are seen with a history of asthma, allergic rhinitis, or chronic bronchitis alone or in combination. In the second phase, the serum shows a leukocytosis with marked eosinophilia. The terminal phase involves a life-threatening vasculitis, and the patient has varying degrees of involvement of the heart, lungs, nervous system, sinuses, skin, gastrointestinal tract, kidneys, and muscles.[3,4] The classic triad of CSS is that of late onset asthma, systemic vasculitis, and peripheral eosinophilia.[2-4]

Report of case

A 59-year-old Lebanese woman sought treatment because of a 5-week history

of increasing malaise, fatigue, fevers, sinusitis, and anorexia with a 15-pound weight loss. Three weeks before admission, she had pain in her right arm, followed by increasing weakness and numbness of her right hand. The weakness and numbness progressed during the next 2 days to include the forearm. A week later, her left hand and arm became weak and numb. The night before admission, she began having difficulty moving her left leg and she could barely walk.

The patient's past medical history was significant for adult-onset asthma of 13 years' duration, sinusitis, seasonal allergies, and sensitivity to aspirin and penicillin. She had lived in the United States since 1970 and returned to Lebanon twice, the last visit being 4 years ago. She denied having any illness during or after these visits. She did not drink or smoke.

Physical examination showed a mildly cachectic woman with stable vital signs and a temperature of 101.1°F (measured orally). Findings of the head, eyes, ears, nose, and throat examination were significant for maxillary sinus tenderness bilaterally. Findings on auscultation of the heart and lungs were normal. Neurologic examination found no gross deficits, and cranial nerves were grossly intact. The patient had symmetric weakness of the upper and lower extremities—distal greater than proximal—with asymmetric weakness of the left extensor digitorum communis and the left biceps femoris. Deep tendon reflexes were hyporeflexic in the upper extremities and areflexic in the lower extremities. Plantar reflexes were flexor. Regions of hypoesthesia were noted in the left ulnar and superficial peroneal nerve distributions.

Laboratory studies showed a leukocytosis with a white blood cell count of 29,000/mm^3, with 52% eosinophils. A blood theophylline level was subtherapeutic. The erythrocyte sedimentation rate was 48 mm in 1 hour. A second complete blood cell count showed 62% eosinophils. Analysis of cerebrospinal fluid (CSF) revealed no abnormality, with total protein value of 18 mg/dL. Oligoclonal bands and myelin basic protein were absent in the CSF and serum. The absolute eosinophilic count was 10,240/mm^3 (reference laboratory normal range, 150/mm^3 to 300/mm^3). Nasal smears were negative for eosinophils. Serum protein electrophoresis showed an elevated IgE level of 1645 mg/dL (reference laboratory normal range, <1000 mg/dL). Computed tomography scans of the brain, chest x-ray film, and an echocardiogram showed no abnormalities. Sinus x-ray films showed pansinusitis. The C3 and C4 levels were normal, but the total complement (CH_{50}) level was slightly elevated. Assays for cryoglobulins, Lyme titer, and antinuclear antibody as well as blood cultures and examination of stool for ova and parasites were negative.

Trichinosis titer was weakly positive, suggesting either an early mild infection or a false-positive. This titer was thought to be a false-positive for several reasons. The patient did not eat pork for personal and cultural reasons. Five weeks of anorexia had limited her oral intake primarily to fluids, carbohydrates, fruit, and occasionally chicken. She did not have any other manifestations of trichinosis through history, physical, or laboratory evaluation.

The only medication that the patient was taking that had the possible side effect of eosinophilia was doxycycline, but her eosinophilia was well out of proportion to that seen secondary to a simple drug reaction. She had not been on long-term steroid therapy for her asthma.

The electromyogram was consistent with a mononeuritis multiplex without evidence of polyradiculopathy or plexopathy. Tissue biopsies of skin, muscle, and sural nerve showed varying stages of necrotizing vasculitis with fibrinoid necrosis and wallerian degeneration (*Figures 1* and *2*).

The patient was treated with high doses of intravenous steroids and aggressive physical therapy. She tolerated treatment well and progressed from a bedridden state to ambulation with a platform walker in 1 week. At follow-up evaluation 3 weeks after discharge, she was walking with a cane and continued to do well on a long-term tapering dose of oral prednisone.

Discussion

A great deal of progress with vasculitides has been made since 1950, when Churg and Strauss[2] reported the syndrome of allergic granulomatosis, allergic angiitis, and periarteritis nodosa which bares their names. At that time, all but 1 of 13 patients reported to have CSS had died within 5 years of reaching the terminal phase. Since the advent of steroid therapy in the treatment of this syndrome, there is a 90% survival rate at 1 year and 62% survival rate at 5 years.[4-6] The most common cause of death is myocardial infarction and congestive heart failure, with other causes including status asthmaticus, salmonella septicemia, ruptured aneurysm, renal failure, pneumonia, and small-bowel infarction.[4-6]

Although CSS has been recognized with increasing frequency over the years, it is still considered rare and has much in common with the other vasculitides.[1,4] The age of onset is between 15 and 69 years of age, with a mean of 47 years, and there is a 2-to-1 male predominance.[6] Patients usually have a history of late-onset asthma. Before steroid treatment, most patients died within a year of the terminal phase if the interval between the onset of asthma and the terminal phase was less than 4 years.[4,6]

The criterion of the constellation of eosinophilia, vasculitis, and extravascular granulomas set by Churg and Strauss is presently recognized in only a small number of cases and is currently considered too rigid.[3,4,7] Current thinking suggests that the diagnosis can be made clinically.[4] Findings on tissue biopsies are usually similar to those of polyarteritis nodosa.[2,3] The key feature is a necrotizing vasculitis with fibrinoid necrosis of capillaries, arterioles, venules, and small and medium arteries.[1-4,6] In the acute stage, there is segmental fibrinoid necrosis of vessels with infiltration of leukocytes (primarily eosinophils) in the vessel wall and perivascular spaces.[2-4] When granulomas are present, they are typically greater than 1 mm in diameter, perivascular, and contain a central eosinophilic core surrounded by macrophages and giant cells.[2,4,6]

The common systemic signs seen with

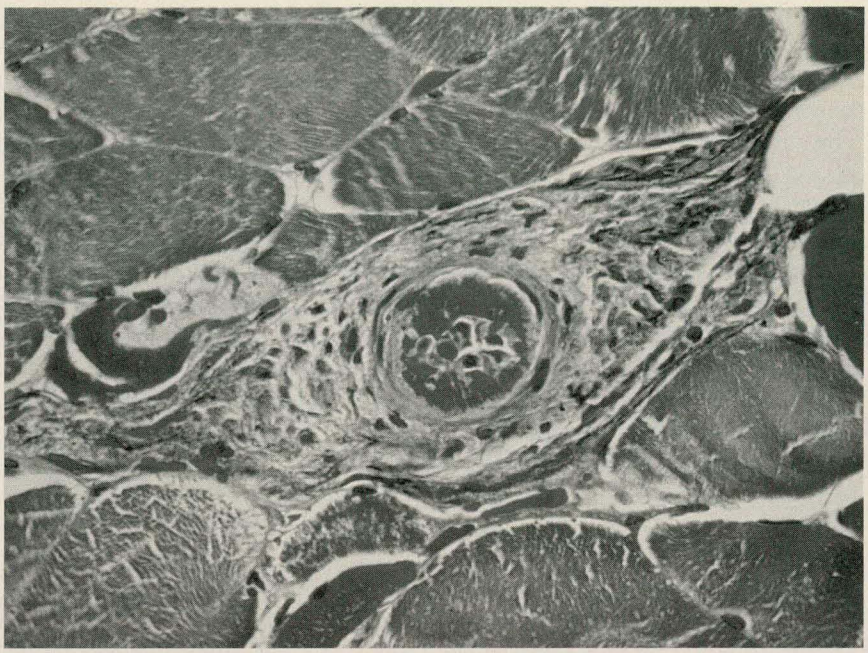

Figure 1. Paraffin-embedded section stained with trichrome showing fibrinoid necrosis of small artery with increased connective tissue and periadventitial fibroblastic proliferation that penetrates from blood vessel wall, resulting in atrophy and necrosis of adjacent myofibers (original magnification ×100).

CSS include malaise, lassitude, weight loss, fever, arthralgias, and myalgias.[3-5] Cutaneous manifestations are a prominent component and include rash, palpable purpura, and tender nodules often seen in the scalp and symmetrically in the extremities.[2-4,6] Livedo reticularis and cutaneous infarctions have also been reported.[4]

Asthma is the most frequent respiratory complaint seen in CSS. It can precede CSS by up to 30 years, although the symptoms of asthma abate before the onset of CSS in a majority of patients.[2-4] Pulmonary infiltrates are a central feature in CSS, seen in 72% of cases, with extensive eosinophilic infiltration of the alveoli and interstitium.[3,4] Chest x-ray films show that the infiltrates are usually transient, patchy, and without predilection for one area. Bilateral nodular infiltrates have been reported, but they rarely cavitate as seen with Wegener's granulomatosis. Allergic rhinitis leading to nasal obstruction, recurrent sinusitis, and nasal polyposis are the most frequent upper respiratory tract symptoms.[4]

Cardiac involvement accounts for 48% of deaths in CSS.[3,4] Left untreated, myocardial infarction and intractable cardiac failure can result.[2-4] Hypertension is seen in 75% of patients.[3,4] Acute and constrictive pericarditis and cardiac tamponade have also been reported as well as extensive replacement of the myocardium by granulomas and scar tissue.[8,9]

Gastrointestinal involvement is also common in this syndrome. Abdominal pain occurs in up to 59% of patients. Mesenteric vasculitis, hepatic artery aneurysms, cholecystitis, diarrhea, and gastrointestinal tract bleeding have been reported. Submucosal eosinophilic infiltration in the bowel walls can produce large nodular lesions and lead to obstruction. This obstruction, with serosal involvement, can produce an eosinophilic peritonitis with ascites containing large amounts of eosinophils.[3,4]

Renal involvement in CSS is rare. The lesion is one of focal segmental glomerulosclerosis with necrotizing features and is similar to Wegener's granulomatosis. Mild hematuria and albuminuria have also been reported.[2] A bladder neuropathy and granulomas of the prostate, ureters, and penis have also been described in CSS.[3,4]

Eosinophilia is a hallmark sign of CSS, but cases have been reported without it.[3,4] Three basic proteins in the eosinophil have been isolated which have potential neurotoxic effects: major basic protein (MBP), eosinophil cationic protein (ECP), and eosinophil-derived neurotoxin (EDN).[10-13] The MBP is a nonspecific protein that can damage vascular endothelial cells and neutralize heparin, leading to a thrombotic and embolic phenomenon. The ECP leads to a hypercoagulable state by neutralizing heparin and potentiates factor XII–dependent coagulation pathways. The EDN damages neurons of the central nervous system (CNS) and peripheral nervous system (PNS) directly. The mechanism is unknown. Intrathecal injection of EDN has been shown to selectively damage Purkinge's cells of the cerebellum, white matter regions of the cerebellum, spinal cord, and brainstem, causing Gordon's phenomenon (muscle rigidity, ataxia progressing to severe weakness, and paralysis).[10-13] Eosinophils and their contents can produce neurotoxicity five ways[11]:

- direct infiltration into the nerve,
- direct cytotoxicity or antibody-dependent eosinophilic cytotoxicity,
- direct secretion of eosinophilic products into neurons or into the circulation and then to the nerve,
- cerebral infarction secondary to the hypercoagulable state caused by MBP and ECP, and
- neurotoxicity secondary to eosinophilic damage to remote organ systems, such as the heart and lungs.

Neurologic involvement in the systemic necrotizing vasculitides can be both central and peripheral.[1-5,14,15] In a study of 25 patients with systemic vasculitis, Moore and Fauci[16] found CNS involvement in 40% and PNS involvement in 60%. The CNS manifestations include disorientation and confusion as seen in encephalopathies, seizures, and psychosis.[3,4] Occlusion of vessel walls from fibrinoid necrosis can result in cerebral infarction. Weakening of the vessel walls can lead to aneurysm formation and rupture resulting in subarachnoid hemorrhage and hematoma. Strokes occur most-

Figure 2. Toluidine blue–stained semithin section of left sural nerve in longitudinal section showing myelin ovoids, ballooned or collapsed myelin profiles, marked decrease in myelinated axons, amorphous lipid, fibroblastic proliferation, and connective tissue, interstitial edema, and increased Schwann cell nuclei. No inflammatory infiltrates are seen (original magnification ×250). All characteristics are consistent with wallerian degeneration.

ly in the cerebral hemispheres, but they can also be seen in the cerebellum, brainstem, and spinal cord. Cranial nerve involvement is relatively infrequent, but involvement of nerves II, III, IV, VII, VIII, and XII have been reported, with the most common lesion being ischemic optic neuritis.[1,3,4,5,14] Other neurophthalmologic manifestations of CSS include eosinophilic granulomas of the eyelids or conjunctiva, scleritis, episcleritis, panuveitis, amaurosis fugax, retinal ischemia or infarcts, and central or branch retinal artery occlusions.[14,15]

Approximately 60% of all patients with CSS have features of a peripheral neuropathy.[3,16] The etiology is thought to be secondary to compromise of the vasa nervorum leading to ischemia and wallerian degeneration. Four types of peripheral neuropathy have been reported in CSS:

- mononeuritis multiplex,
- extensive mononeuritis,
- distal sensorimotor polyneuropathy, and
- cutaneous neuropathy.[3,16]

Of the four neuropathies, mononeuritis multiplex is the most prevalent. Most commonly, symptoms start with pain, followed by motor and sensory deficits in the distribution of more than one peripheral nerve, developing during a period of hours to days, but of short duration. Extensive mononeuritis multiplex is less common, results in more severe deficits, and evolves and resolves slower than mononeuritis multiplex. There can be flaccid weakness, quadriparesis, and a multimodality sensory loss in one or more extremities with distal greater than proximal involvement. Many patients have sensory levels in mid arm and thigh. It is thought that this entity is the result of simultaneous infarction of multiple peripheral nerves in all four extremities. In case reports by Moore and Fauci,[16] analysis of CSF and myelograms showed no abnormality.

The cutaneous neuropathy is described as a hypoesthesia to pain, temperature, and touch, in the distribution of small cutaneous nerves. The regions most commonly affected are individual digits and the dorsum and lateral sole of the feet. The mechanism is believed to be a decrease in blood flow through nutrient vessels in these regions. The sensorimotor neuropathy seen with the necrotizing vasculitides is similar to that seen with diabetes and alcoholism, and these disorders need to be ruled out. There is a symmetric distal decrease in all sensory modalities, along with varying amounts of distal weakness. Brachial and lumbar plexopathies have also been reported in CSS.[3]

The pathogenic mechanism leading to the vasculitic phase of CSS is currently unknown, but immune complex deposition appears to play a role. One theory is that small IgE-containing immune complexes may initiate the vasculitis in a manner comparable to an IgA-induced alternate pathway activation.[3,10,17] Evidence to support this theory comes from a decrease in IgE serum precipitants after treatment with steroids.[3,10] Another report showed the presence of C3 complement and IgA deposits with immunofluorescence of a vessel wall in a patient with CSS, suggesting another possible mechanism for local tissue injury.[18]

The preferred treatment in CSS is 40 mg to 60 mg of methylprednisolone daily for several weeks, followed by a tapering dose during several months.[1,3,4] Dosage is highly variable, depending on the extent of the symptoms and clinical response. Improvement is usually rapid and, as in our patient, dramatic. Studies suggest that efficacy of treatment can be better seen by monitoring the eosinophilic count rather than by clinical observation.[5] For the more resistant cases, azathioprine, cyclophosphamide, chlorambucil, and plasmapheresis have been used with positive results.[1,3,4]

Comment

The patient described here had CSS with the classic triad of late-onset asthma, systemic vasculitis, and peripheral eosinophilia; however, her lack of multisystemic involvement was unusual. Significant progress has been made in the treatment of this syndrome since it was first described in 1950. Early recognition and

treatment often lead to dramatic and rapid improvement.

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Case reports continued

Acute appendicitis secondary to non–0 group 1 *Vibrio cholerae*

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Acute appendicitis is the most common abdominal surgical condition and is usually associated with colonic flora. The patient described had acute appendicitis associated with an uncommon microorganism. This report underscores the importance of obtaining an adequate occupational, travel, and dietary history.

(Key words: Acute appendicitis, non–0 group 1 *Vibrio cholerae*)

Acute appendicitis is the most common abdominal condition requiring surgery. It is most often initiated by either obstruction of the appendiceal lumen secondary to a fecalith or mucosal ulceration. Luminal obstruction causes bacteria to multiply and invade the mucosa. The most common organisms include aerobic and anaerobic colonic flora, particularly *Bacteroides fragilis* and *Escherichia coli*.[1]

We describe a case in which acute appendicitis was associated with an uncommon bacterium.

Report of case

A 52-year-old military officer who recently returned to New York City from the Philippines sought medical attention because of acute midabdominal pain, nausea, and fever. Physical examination demonstrated right lower quadrant rebound tenderness. Oral temperature was 100°F, and the leukocyte count was 15,800/mm^3. The patient received cefoxitin, 1 g every 8 hours, and gentamicin, 80 mg every 8 hours, intravenously, and he was taken to the operating room with a diagnosis of acute appendicitis. Surgery confirmed the diagnosis of acute suppurative and gangrenous appendicitis. The suppurative fluid prepared with Gram's stain demonstrated gram-negative bacilli. The intravenous administration of the antibiotics was continued for 2 days. The patient recovered and was discharged from the hospital on the third postoperative day. He was placed on ampicillin therapy, 500 mg taken orally every 6 hours.

After the patient's discharge from the hospital, the organism was identified as serotype non–0 group 1 *Vibrio cholerae,* which was sensitive to ampicillin (<4 μg/mL), ceftriaxone (<4 μg/mL), ciprofloxacin (<1 μg/mL), gentamicin (<4 μg/mL), and trimethoprim-sulfamethoxazole (<0.5 μg/mL). The patient's recovery was uncomplicated.

Discussion

Bacteria of the genus *Vibrio* are aquatic aerobic gram-negative bacilli that require specialized medium supplemented with salt for isolation. The organisms are found in both fresh and salt water. Humans become infected by eating undercooked shellfish, particularly oysters, or when

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