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Churg-Strauss Syndrome : Overview

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ABSTRACT:

Churg-Strauss condition is a problem set apart by vein irritation. This aggravation can limit blood stream to organs and tissues, at times forever harming them. This condition is otherwise called eosinophilic granulomatosis with polyamides.Adult-beginning asthma is the most well-known indication of Churg-Strauss disorder. The issue can likewise create different issues, like nasal sensitivities, sinus issues, rash, gastrointestinal dying, and torment and deadness in your grasp and feet. The reason for Churg-Strauss disorder is generally obscure. All things considered, a blend of qualities and ecological elements, like allergens or certain prescriptions, sets off an overactive resistant framework reaction. Rather than safeguarding against attacking microorganisms and infections, the resistant framework targets solid tissue, causing far and wide irritation.

It is characterized by the presence of eosinophilia, or increased levels of eosinophils, which are a subtype of white blood cells typically involved in parasitic infections and cancer; evidence of granulomatous necrotizing vasculitis, which is characterized by inflammation of the blood vessels, signs of tissue death (or necrosis), and collections of immune cells in or near the vessel wall; and abnormal lesions in the lungs that may mimic pneumonia. Although the lung is the most commonly involved organ, Churg-Strauss syndrome can affect any organ system, including the skin, cardiovascular system, gastrointestinal system, kidneys, and central nervous system. A hallmark of the disease is its association with asthma and rhinosinusitis, which is the inflammation of the nasal cavity and the paranasal sinuses.

Key words: Churg-Strauss , Corticosteroids, granulomatous ,Benralizumab ,cyclophosphamide

Introduction:

Allergic granulomatous agilities, also known as allergic granulomatosis and more commonly known as Churg-Strauss syndrome (CSS), is a systemic vasculitic disorder of unknown cause that was first recognized as a distinct entity in 1951 by pathologists J. Churg and L. Strauss. 1 At the Mount Sanai Hospital in Manhattan, NY, they noticed a group of 13 asthmatic patients who had fever, hypereosinophilia, and vascular anomalies. Only two people were able to recover from the sickness. Following that, three main histologic criteria were outlined: There are three types of necrotizing vasculitis: 1) necrotizing vasculitis, 2) eosinophilic tissue infiltration, and 3) extravascular granulomas.

Definition:Since the first Churg and Strauss Article, a few clinical case series have Been published.2-6 In a 1995 epidemiologic review the rate was assessed at 2.4 cases per million.7 CSS Side effects will generally show up between the Ages of 20 and 40, with a slight power in men.8 Nonetheless, writers Have contended that CSS might be particularly underdiagnosed as a result of unbending Adherence to Churg and Strauss' three Histologic criteria.Lanham et al3 Stated that these Histologic rules coincide both spatially and transiently in just a minority Of cases. Distinguishing proof of CSS by clinical measures might be more important on the grounds that the illness cycle has a commonplace Three-stage design :

1) prodromal phase of allergic disease, often allergic rhinitis evolving into asthma;
2) peripheral blood eosinophilia and eosinophilic tissue infiltration; and
3) a life-threatening vasculitic phase.
5) nonfixed pulmonary infiltrates; and
In 1990, the American College of Rheumatology prepared two classification schemes to distinguish CSS from Other vasculitides on the basis of observations of 20 patients with CSS.9 The highly specific “traditional format.
Classification” delineated six criteria:
1)Asthma;
2)peripheral eosinophilia 10% on differential leukocyte count;
3)Mononeuropathy (including multiplex) or polyneuropathy; ko
4)paranasal Sinus abnormality;
6) biopsy evidence Of extravascular eosinophils.
The presence of four or more of these Criteria yielded a sensitivity of 85% And a specificity of 99.7%. A more Sensitive “classification tree” was devised comprising three criteria: 1)Asthma, 2) peripheral eosinophilia 10% on differential leukocyte count And 3) history of documented allergy Other

than asthma or drug sensitivity.These criteria yielded a sensitivity of 95% and a specificity of 99.2%.

Churg-Strauss syndrome was first described in 1951 as a pathological syndrome of allergic granulomatosis and agilities .There are three main Classification schemes: Lanham's criteria , which rely On clinical features, and the criteria of the American Colleges of Rheumatology and the Chapel Hill Consensus Conference , which emphasize pathology .The syndrome is often associated with the presence of Perinuclear antineutrophilic cytoplasmic antibodies (PANCA), which target primarily myeloperoxidase. It is not Known if these antibodies correlate with the clinical Course or prognosis of the disease. Reports have suggested that leukotriene receptor antagonists, used for treating asthma, may cause ChurgStrauss syndrome (5–14). However, it remains unclear if Corticosteroid dose reduction by these drugs unmasks Pre-existing Churg-Strauss syndrome, or if they have a Primary causative effect. Consequently, we sought to determine whether the Presence of ANCA in patients with Churg-Strauss syndromes denotes a more vasculitic clinical picture, and Whether leukotriene receptor antagonists modify the Clinical presentation.

DISEASE SPECTRUM

The clinical indications of CSS reflects contribution of different organ framework's . In Churg and Strauss' Unique article,1 cardiovascular, digestives, urinary, and sensory system contributions were noted every now and again in These patients who had hypereosinophilia, vasculitis, and asthma. Frequently persistent's with CSS whine of constitutionals side effects like fever, Discomfort, anorexia, and weight reduction. In The review concentrate by Guillevin et Al6 of 96 patients with CSS, 71% of the Subjects experienced quick, huge Weight reduction (5% of body weight) and 57% showed steady fever surpassing, 38° C for a long time.

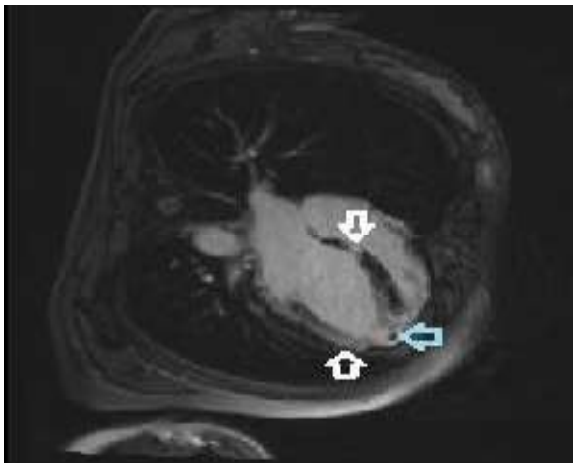
Pulmonary

One of the central defining features of CSS is asthma. Asthma may precede Systemic vasculitis by as many as 30 Years (mean latency, 8 years).2 Typically, asthma exacerbations increase in Frequency and severity preceding the Onset of vasculitis.3,10 In the review by Lanham et al,3 58% of patients with CSS were free of asthma in the early Stages of their vasculitic illness. Onset Of asthma coincided with the onset of Systemic vasculitis in 20% of the cases Reported by Chumbleyp et al2 In the Postvocalic phase, bronchospastic Symptoms may dominate clinically. Asthma may persist in more than 80% Of patients during long-term remission Of CSS. Nonfixed, patchy pulmonary infiltrates' were noted on chest radiographs In 27% to 63% of patients.2,3 Radiographic findings are often associated With fever, cough, and dyspnea. Pulmonary ,infiltrates show no predilections for any specific area of the lung And may or may not be symmetrical or Bilateral. In patients with CSS with Pulmonary infiltrate, results of bronchoalveolar lavage typically show eosinophilia ranging from 6% to 66%.12 Alveolar hemorrhage has been reported In CSS.13 Pulmonary nodules may also Be present, although unlike those in Waggener granulomatosis, they rarely caviatate.3 Pleural effusion occurs in 7.3% to 29% of patients, and pleural fluid may Contain abundant eosinophils. In a systematic review of pulmonary Computed tomographic Patients with CSS, Worthy et al14 reported parenchymal opacification in Almost 60% with the majority of these (N 6) in a predominantly Peripheral distribution.

Cardiovascular

Cardiac involvement in patients with CSS is a main cause of morbidity and The most frequent cause of death in Reported series.1–3 Manifestations included cardiac arrest, myocardial infarction, valvular heart disease, congestive Heart failure, pericardial effusion, and Acute or chronic constrictive pericardites .3,17–22 Early intervention is desired Because reversibility occurs infrequently. In Churg and Strauss's initial Study,1 6 of 10 postmortem examinations revealed coronary vasculitis. In The review of literature by Lanham et Al,3 almost half of 50 CSS cases with Known causes of death were attributable to congestive heart failure or myocardial infarction, or both.Coronary angiography results for Patients with CSS are usually free of Arteriographic abnormalities.23–25 In One case report, the authors described Alternating areas of narrowing and dilatations in the smaller branches of the Right coronary artery. In a second Case, multiple vessel wall microenterprises and abrupt terminations were Evident and diagnostic for vasculitis. Eosinophilic endomyocarditis (ie, Inflammation characterized by

eosinophilic infiltration of cardiac tissue) has Been noted in numerous disorders, including CSS.20,27,28 Infrequently, this Has progressed to endomyocardial fibroses, ventricular thrombus formations, and obliterative and restrictive Cardiomyopathy.4,20,22,28 –30 In addition To intracardiac thrombus, large- and Medium-vessel arterial and venous Thromboembolism have been described in association with CSS.31,32 Hypercoagulability may be promoted By eosinophilic cationic protein or released of von Willebrand factor from Endothelial cells in areas of vasculitis, Or both. Ames et al31 described cases of Left subclavian vein thrombosis, right Lower extremity deep venous thrombosis, and left cerebral artery infarction.Fig-Cardiac Involvements of Esoninophile Granulomalotosis.



Renal

Historically, renal manifestations in CSS have been generally thought to be mild and not a prominent feature. Lanham et al3 reported that 49% of patients' in their series exhibited mild to moderate renal impairment and 10% had acute renal failure. Chumbley et al2 reported that in a series of 30 patients with CSS, fewer than 4% had renal failure. The generally beaning nature of renal manifestations distinguishes CSS from other necrotizing vasculitides such as Wegener granulomatosis.However, articles have suggested that Renal involvement may be more significant than previously noted.40,41 In the reviews by Clutterbuck et al40 of 19 inpatient's with CSS, 5 patients had renal Disease as the presenting problem, 3 had Nephrotic syndrome, and 4 had concentrations' of serum creatinine 6.6 mg/dL (500 mol/L). Overall, 84% of the study Population had renal involvement: two Patients required hemodialysis; macroscopic hematuria was present in 68% of the patients and proteinuria was present In 63% of patients. Hypertension was Noted in more than one-third of the patients and may have been a contributing Factor to the renal impairment glomerulonephritis with features that are necrotizing, crescentic, or both.3 For 13 renal biopsies in the Study by Clutterbuck et al,40 results of showed focal segmental glomerulonephritis. Churg and Strauss's1 autopsy findings included diffuse or focal interstitial nephritis and necrotizing vasculitis with granulomatous nodules.,

Dermatologic

Subcutaneous nodules on the right elbow of the patients with churg syndrome Cutaneous eruptions occur in up to 70% of patients with CSS.42– 44 Subcutaneous and dermal nodules on the extensors and flexor surface of extremities Are common (Fig 2).45 Crotty et al42 Reviewed 14 patients with CSS and Reported nodules (N 9), papules (N 5), vesicles (N 4), and purpura (N 3). Other findings included skin Infarction, facial edema, urticaria,Macular erythema, and ulceration. Histologic findings were divided into extravascular granulomas (50%), leukocytoclastic vasculitis (33%), and Cutaneous polyarthritis' nodes (17%).Clinically, cutaneous lesions were.

Divided into three groups by Strauss et :

1)erythematous maculopapules resembling erythema multiforme;
2) hemorrhagic lesions ranging from petechial to extensive ecchymosed; and
3) tender, subcutaneous or cutaneous nodules. Histologically, lesions in the first two categories typically display a leukocytoclastic vasculitis, whereas subcutaneous nodules often exhibit the
"characteristic granuloma" of CSS. The nodules are the most persistent And commonly resolve incompletely With scarring within 2 to 3 months. Davis et al46 reported extravascular.Necrotizing granulomas in 15 of 29 cutaneous biopsy specimens from patients With CSS. However, the Churg-Strauss Granuloma is nonspecific and can be Found in other disorders, such as Waggener

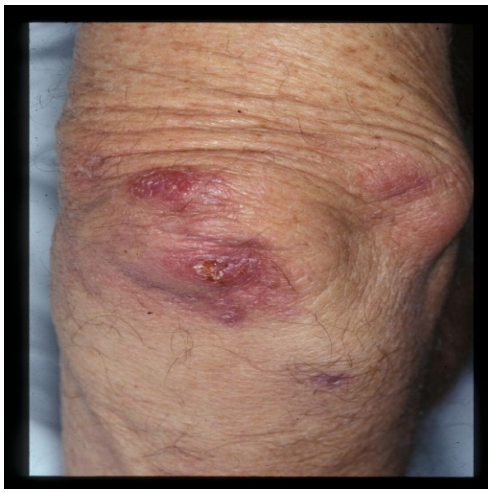


Fig-2 subcutaneous nodules on the right elbow of the patients with churg stratus syndrome.

granulomatosis, systemic lupus Erythematosus, and rheumatoid arthritis. In Finn and Winkelmann's47 reviews of 27 patients with extravascular Necrotizing granuloma, only 26% were Eventually diagnosed with Churg Strauss syndrome

Gastrointestinal :

Gastrointestinal tract manifestations of CSS are varied and reflect the presence Of eosinophilic gastroenteritis and necrotizing vasculitis. In the study of 96 Patients with CSS by Guillevin et al,6 31% had gastrointestinal tract involvements at presentation, most of which Was abdominal pain. Emergency laparotomy for four of these patients Showed acute appendicitis, omental Hematoma, diffuse bowel ischemia And ischemic colitis. Lanham et al3 reported abdominal pain, diarrhea, and Bleeding in 59%, 33%, and 18% of Cases, respectively.Several reports exist of small bowel Necrosis, ulceration, infarction, andPerforation.48 –53 Although granulomatousagilities, with eosinophilic infiltration may be seen in small bowel biopsy Specimens, the

absence of these finding's does not exclude CSS. Further Examination of mesenteric vessels in The attached mesentery may be necessary to document eosinophilic infiltrations and necrotizing vasculitis.Colitis may also be a feature of CSS and, clinically, may resemble ulcerative colitis. Leen et al54 described bloody diarrhea and lower abdominal pain in a 23-year-old man with asthma Endoscopy showed patchy erythem rom the rectum to the proximal ascending colon and severe proctitis. Rectal biopsy specimens had marked eosinophilic infiltration of the submucosa with necrotizing granulomas.

Urologic

Although uncommon, obstructiveUropathy in patients with CSS may occurs secondary to ureteral stenosis.55,56Cortellini et al55 described progressiveAnuria that developed in a 22-

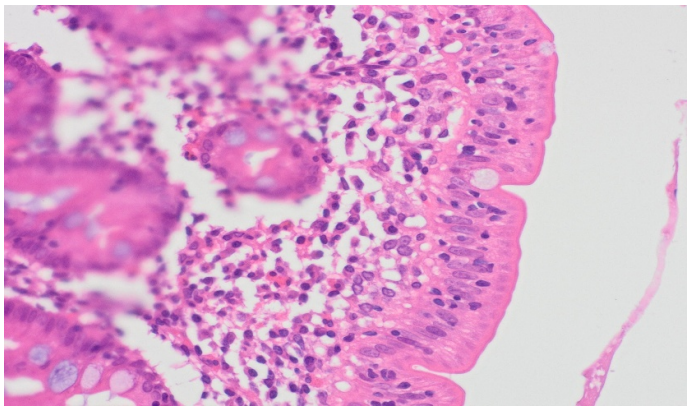


Fig-3-Cross section of an artery in a patient with Churg-Strauss syndrome.

year-oldMan with CSS who had bilateral ureteral obstruction. Scar tissue from possibly healed granulomas on the leftUreter and eosinophilic infiltration ofThe right ureter were found postoperatively. Granulomatous involvement ofThe prostate gland in CSS has also beenReported to occur as an obstructive Uropathycondition.

Pathogenesis

Churg and Strauss1 relied on pathologic criteria in their 1951 landmark Study to describe CSS: 1) necrotizing Vasculitis, 2) eosinophilic tissue infiltrations, and 3) extravascular granulomas. These lesions may coexist or occurs in isolation. In the retrospective Study of patients with CSS by Guillevin et al,6 only 48% of muscle biopsy Specimens had vasculitis.Small arteries affected segmentallyBy necrotizing vasculitis are typical, although medium-sized arteries may be affected as well.3 Histologic findings mayRange from eosinophilic perivascular Cuffing to panmural necrotizing vasculitis (Figs 3 and 4). In addition, veinsMay have pathologic changes.58,59 In theStudy by Guillevin et al,6 17 skin biopsy samples (40%) showed necrotizing vasculitis and 11 (26%) exhibitedLeukocytoclastic vasculitis.

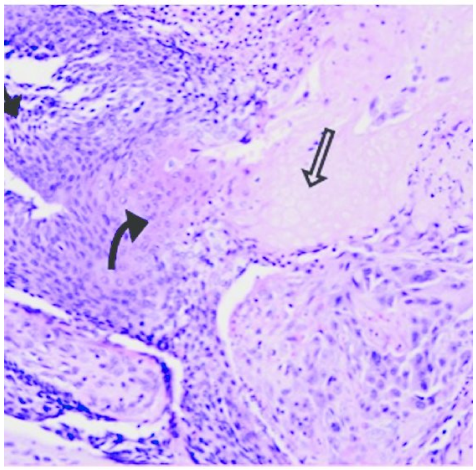


Fig-4-Further magnification of cross section in Figure 3. The irregularity of the intimal lining is apparent (hematoxylin-eosin; 400).

The pathogenesis of CSS is unknowns and several theories have beenPut forth. Of note, antineutrophil cytoplasmic antibody with a perinuclear Fluorescence pattern (p-ANCA) is detected in up to 60% of CSS cases, the Majority of which are against myeloperoxidases (MPO).63,64 MPO-ANCAs Are also found in idiopathic crescentic Glomerulonephritis, polyarthritisnodes, and microscopic polyangiitis.65 The mechanism by which MPO-ANCAs are involved in the pathogenesis Of CSS and whether titers correlate With disease activity remains unclear.Some have proposed that the immunoglobulins (Ig) subclasses IgG1 and IgG3 of anti-MPO antibodies may Crosslink Fc receptors with surface bound MPO on neutrophils and

Thereby lead to neutrophil activation And endothelial cell damage.66 Minami Et al65 described a patient with CSS and Crescentic glomerulonephritis who had markedly increased levels of MPOSNCA.

After treatment with prednisolones, clinical manifestations of the illness gradually resolved and levels of MPO-ANCA decreasedImmunopathogenic causes have alsoBeen proposed.69 In 30 of 96 patientsWith CSS, Guillevin et al6 identifiedDesensitization (47%), discontinuationOf oral corticosteroid therapy (27%),And vaccinations (13%) as 3 main triggering factors to the onset of the vasculitis. Therefore, they suggested usingExtreme caution when vaccinating orDesensitizing patients with unstable orSevere asthma. Also, isolated reportsHave implicated inhaled antigens in thePathogenesis of CSS. Guillevin et al70Described a patient who developedThree distinct episodes of CSS temporally related to contact with pigeonsAnd whose lung biopsy specimensShowed actinomycetes. Schmitt et al67Studied serologicMarkers in 16 patients with CSS duringInactive and active phases of the illness'.

In patients with active disease,Levels of ECP, soluble interleukin-2 receptors (sIL-2R), and soluble thrombomodulin were all markedly increasedCompared with the remission phase.Soluble thrombomodulin is a keyMarker for endothelial cell damage,And the study revealed close correlation with levels of sIL-2R. Further,Increased levels of sIL-2R during remissions (ie,1,000 U/mL) were associated with relapse.67Similarly, Shiota et al68 described aPatient whose serum interleukin (IL)-5Levels correlated with the activity ofHer CSS. Administration of prednisolone resulted in reduction of eosinophils counts, clinical improvement, And normalization of IL-5 levels.

It has Been proposed that IL-5 may act as a Catalyst in enhancing eosinophil maturation, migration, infiltration, and degranulation in tissues.Immunopathogenic causes have also been proposed.69 In 30 of 96 patientsWith CSS, Guillevin et al6 identified Desensitization (47%), discontinuationOf oral corticosteroid therapy (27%),And vaccinations (13%) as 3 main triggering factors to the onset of the vasculitis.

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Diagnosis

To diagnose Churg-Strauss syndrome, doctors usually request several types of tests, including:

Blood tests. A blood test can detect certain antibodies in your blood that can suggest, but not confirm, a diagnosis of Churg-Strauss syndrome. It can also measure the level of eosinophils, although other diseases, including asthma, can increase the number of these cells.

Imaging tests. X-rays and CT scans can reveal abnormalities in your lungs and sinuses. If you develop signs of heart failure, your doctor may also suggest regular echocardiograms.

Biopsy of affected tissue. If other tests suggest Churg-Strauss syndrome, you might have a small sample of tissue removed for examination under a microscope. The tissue can come from your lungs or another organ, such as skin or muscle, to confirm or rule out the presence of vasculitis.

Symptoms

Churg-Strauss syndrome varies greatly from person to person. Some people have only mild symptoms. Others have severe or life-threatening complications. Also known as EGPA, the syndrome tends to occur in three stages and gets progressively worse. Almost everyone with the condition has asthma, chronic sinusitis and elevated counts of white blood cells called eosinophils.

Other signs and symptoms might include:

Loss of appetite and weight loss
Joint and muscle pain
Abdominal pain and gastrointestinal bleeding
Weakness, fatigue or a general feeling of being unwell
Rash or skin sores
Pain, numbness, and tingling in your hands and feet

Treatment and Clinical Management

Corticosteroids : The mainstay of treatment for CSS continues to be corticosteroid therapy. The specific dosing of corticosteroid required to achieve remission has not been well studied. For patients with acute multiorgan involvement, such as acute renal failure or respiratory depressions, high-dose (ie, 1 g) methylprednisolone given intravenously may be indicated for up to 3 consecutive days.11 For the more common, non– life-threatening presentation, 40 mg to 60 mg daily of prednisone given orally may suffice. Prednisone therapy is continued until no evidence of disease remains and is then gradually tapered. Additional immunosuppressive treatments has been used concurrently with corticosteroid medication.

Guillevin et al100 conducted several prospective, randomized studies of patients with polyarthritis nodosa (PAN) or CSS. In a 1991 study, 71 patients with PAN and CSS received corticosteroid medication and plasma exchanges (PLEX); patients were randomly assigned to receive cyclophosphamide or not. Survival at 10 years was no different, but patients in the cyclophosphamide group exhibited better clinical response and lower incidence of relapse. In a 1992 randomized study of 78 patients with PAN or CSS, patients who received PLEX in addition to glucocorticoids therapy showed no benefit with respect to disease activity and survivals over those who received glucocorticoids therapy alone.101 The incidence of relapses in both groups was much higher than in the previous study which used cyclophosphamide. Two additional studies' of patients with PAN or CSS showed that patients did not benefit from the addition of PLEX to therapy with corticosteroids medication plus cyclophosphamide.102,103 PLEX is not

recommended routinely for initial management of patients' with PAN or CSS. Azathioprine has been used successfully in patients with CSS, although it has not been studied in prospective trials.2,10,40 Tatsis et al104 reported on three patients with CSS who responded poorly to cyclophosphamide therapy and were corticosteroid-dependent.

The substitution of subcutaneous interferon- for cyclophosphamide led to remission of disease and reduction of the corticosteroid dose. McDermott and Powell105 reported on a patient whose CSS failed management with azathioprine and cyclophosphamide and who remained steroid-dependent. However, when maintenance cyclosporine A was added, oral steroid therapy was tapered off and this patient has remained in clinical remission.

Other immunosuppressive drug

For people with mild symptoms, a corticosteroid alone may be enough. Other people may need to add another drug to help suppress their immune systems. Mepolizumab (Nucala) is currently the only drug approved by the U.S. Food and Drug Administration for treatment of Churg-Strauss syndrome. However, depending on the severity of disease and the organs involved, other medications may be required.

Examples include:Azathioprine (Azasan, Imuran)
Benralizumab (Fasenra)
Cyclophosphamide
Methotrexate (Trexall)
Rituximab (Rituxan)

Home remedies:

Long-term treatment with corticosteroids can cause a number of side effects. You can minimize these problems by taking the following steps:

Protect your bones. Ask your doctor how much vitamin D and calcium you need in your diet, and discuss whether you should take supplements.

Exercise. Exercise can help you maintain a healthy weight, which is important when you're taking corticosteroid medications that can cause weight gain. Strength training and weight-bearing exercises such as walking and jogging also help improve bone health.

Eat a healthy diet. Steroids can cause high blood sugar levels and, eventually, type 2 diabetes. Eat foods that help keep blood sugar stable, such as fruits, vegetables and whole grains.

METHODS

Following institutional survey board endorsement, potential subjects were recognized by electronic pursuit of the Mayo Center Rochester records data set from 1990 to 2000 utilizing the terms Churg-Strauss condition, Churg strass vasculitis, unfavorably susceptible granulomatous dexterities, and hypersensitive granulomatosis. Close to 100% (156/157) of recognized patients had agreed to survey of their records. One patient was erroneously recognized. Twenty-nines had just dermatological proof of Churg-Straus granulomas. Clinical assessment had barred ChurgStrauss condition in 21 patients, of whom 7 had different kinds of vasculitides. Of the leftover 105 patients, deficient records blocked another 6, leaving 99 evaluable patients with Churg-Strauss disorder. A portion of these patients have been remembered for past investigations or reports of Churg-Strauss syndrome.Clinical information were disconnected from the clinical records, which included documentation of short term, ongoing and crisis division visits at Mayo Facility Rochester, and any correspondence from other clinical suppliers. Pathologic information were acquired from examples surveyed at the facility. ANCA testing was performed by backhanded immunofluorescence (19). Patients seen after 1993 were tried by a calculation that included testing for myeloperoxidase-ANC .The presence of explicit organ contribution was acknowledged whether there was clinical proof of sickness, especially whenever upheld by radiographic discoveries, histopathology, or other testing like electromyography (mononeuritis multiplex, neuropathy normal for vasculitis) or echocardiography (valvular association or left ventricular systolic brokenness turned around by immunosuppression). Patients needed to meet the models of something like one of three Characterization plans American School of Rheumatology standards , Lanham's measures or the Church Slope Agreement Gathering definition As per the Sanctuary Slope Meeting explanations, Proxy boundaries for granulomatous aggravation Of the aviation routes (sinusitis or pneumonic invades) and Vasculitis (neurologic, cardiovascular, or renal illness reliable With vasculitis) were acknowledged (4,21). Without these substitute's, just 1 patient would have met the Sanctuary Slope Definition. Not entirely settled for U.S. occupants (86/91) utilizing the Government backed retirement Passing Record (February 2003) and was contrasted and 2002 U.S. life tables subsequent to Adapting to sex and progress in years.

CONCLUSION

CSS is distinguished from other vasculitides by the presence of asthma, Peripheral eosinophilia, mononeuropathy or polyneuropathy, paranasal Sinus abnormality, nonfixed pulmonary infiltrates, and biopsy evidence Of extravascular eosinophils. Cardiac Involvement is the main cause of Death in this syndrome, and neurologic complications, such as mannerisms multiplex, are some of the most Frequent findings. Although the exact Cause of CSS is unknown, the numerous factors associated with eosinophilic degranulation may contribute Significantly to the pathophysiology of CSS. Whether a direct association exists between LT antagonists and CSS Remains unclear. The majority of reported cases can be explained by concurrent withdrawal of glucocorticoid Therapy. Glucocorticoid therapy remains the mainstay of treatment with The use of added immunosuppression In certain situations to induce remission and to prevent relapse. The prognosis for the majority of patients with CSS is good; poor prognosis may be Predicted by the severity of specific Organ system involvement.

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