the sensitivity? The two patients in our series with diagnostic criteria for exudates yet high albumin gradients had coexistent criteria for a transudative etiology (collapsed lung and congestive heart failure). We wonder if any of their misclassified exudates could have also had a transudative etiology. The three patients with mesothelioma could have had trapped lung leading to transudative effusions. Also, their patients included a number with tuberculosis (two of whom were misclassified as having transudates), which we did not see in our group of patients.

The major criticism of both our sets of data is that the diagnoses are based on clinical criteria. This problem would best be solved by examining long-term outcome data on patients classified as having transudative effusions to rule out the possibility of a clinically silent exudative cause that was missed. More work should also be done to elucidate the pathophysiology of pleural effusions, especially those with potentially more than one cause.

How, then, can the serum-effusion albumin gradient be used in the evaluation of pleural effusions? We feel it can be helpful in two circumstances. First, in a patient with clinical congestive heart failure, an effusion with Light’s criteria suggesting an exudate, negative cytology, and a high albumin gradient, a period of observation with further diuretic therapy is warranted. Second, in a patient with a known malignant effusion but a high albumin gradient, a coexistent transudative cause for the effusion is suggested, and perhaps a trial of diuretic therapy for palliation is indicated.

Maj Bernard J. Roth, M.D., MC, USA, and Lt Col W Hal Cragun, M.D., F.C.C.P., MC, USA, Pulmonary/Critical Care, Madigan Army Medical Center, Tacoma, Washington

The opinions and assertions contained herein are the private views of the authors and do not necessarily reflect the views of the Department of Defense.

Churg-Strauss Angitis

To the Editor:

We read with interest in the November 1991 issue of Chest the article by Guillevin et al.1 in which the authors forcefully suggest a possible etiologic role for inhaled actinomycetes in systemic vasculitis with respiratory manifestations.

In our case, the diagnosis of Churg-Strauss angitis (CSA) was supported by pathologic specimens from lung biopsy showing vasculitis of medium-sized vessels associated with infiltration of lymphocytes and plasmocytes in the absence of fibrinoid necrosis. However, this conclusion seems questionable, as the classical histologic criteria for CSA (which incidentally are thought to be nonspecific) include the finding of vasculitis of small vessels and necrotizing extravascular granulomas, usually with eosinophilic infiltrates. Moreover, the patient in our case cannot be said to have CSA on clinical grounds, since according to the traditional format classification of the American College of Rheumatology at least four of six criteria have to be fulfilled for a clinical diagnosis of CSA to be made. Therefore, the observation in our case of only three criteria, namely, asthma, neuropathy, and pulmonary infiltrates (with the notable absence of eosinophilia of more than 10 percent, paranasal sinus abnormality, and extravascular eosinophilia), makes the suggested diagnosis untenable.

It is important to recall that, unlike American authors, Guillevin and other French investigators do not consider CSA and polyarteritis nodosa as two separate clinical entities and propose the application of common diagnostic criteria.2 Recognizing that this patient fulfilled the “French criteria” for polyarteritis nodosa, it is unclear why, accordingly, a diagnosis of polyarteritis nodosa was not established, instead of the less consistent conclusion of CSA.

Giuseppe Riccio, M.D., F.C.C.P., M. Elena Leoldt, M.D., Paolo Bottero, M.D., and Carlo Noei, M.D., Clinical Immunology Service, Ospedale G. Fornaroli, Magenta, Italy

Reprint requests: Dr. Riccio, Divisione Medicina I, Ospedale Fornaroli: USSL 72, Via Donatore di Sangue 50, 20013 Magenta (Milano), Italy

REFERENCES


To the Editor:

We agree with Riccio and coworkers that the case we reported1 should be attributed to polyarteritis nodosa (PN), not CSA. Our case satisfied only three of the classification criteria established by the American College of Rheumatology (ACR),4 despite the fact that the clinical history and symptoms obviously adhered more closely to CSA than PN. We would like to mention that the ACR criteria for CSA have a sensitivity of 85 percent and a specificity of 90.7 percent and that some patients do not fit with the published criteria. In our opinion, it is often difficult to sharply separate CSA (now called Churg-Strauss syndrome [CSS]) from PN. The Faucci classification5 was helpful in identifying three subsets in the PN group: (1) PN, (2) CSA/CSS, and (3) overlap syndrome. In CSS the pathologic manifestations vary somewhat, with only some of the patients presenting a granuloma, which is conversely present in a broad spectrum of vasculitides, including typical Küttner-Maier disease. For all these reasons, we are not so peremptory in separating PN and CSS patients, as the line of demarcation is not clear-cut.

On the basis of our experience with large groups of patients,6 we think that CSS and PN belong to the same group, expressing some common symptoms (eg, fever, neuropathy) and others that are more specific to each entity (eg, asthma in CSS, presence of hepatitis B virus markers and orchitis in PN). Hypereosinophilia is usually present in CSS but can also be present in PN without symptoms of allergy or respiratory signs. Differences between CSS and classic PN could be due to the etiologic factors observed in each disease (eg, inhaled antigens responsible for asthma and sinusitis in CSS as opposed to a nonrespiratory mode of entry for antigens in classic PN). In PN, hepatitis B virus has been considered responsible for 36 percent of cases,4 and the characteristics of the disease exclude pulmonary symptoms. Laboratory data also show that in patients with CSS and microscopic PN,7 anti-neutrophil cytoplasm antibodies (ANCAs) are often, but not exclusively, present and that in classic PN, ANCAs are rare.

For all these reasons we believe that, without denying the great
interest of the ACR classification, vasculitides of the PN group should be considered as a common trunk, a close family of disorders, with their various expressions related to the antigens involved and their pathogenic consequences.

Loïc Guillevin, M.D.,
Department of Internal Medicine,
Hôpital Aucois,
Bobigny, France

REFERENCES


Antithrombotic Therapy for Venous Thromboembolism

To the Editor:

I would like to call your readers' attention to some errors in our article,1 which appeared in the supplement to the October 1992 issue of Chest. In lines 4 through 7 of the right-hand column on page 417S, the sentence should read "Urokinase is recommended to be given as a 4,400 IU/kg of body weight loading dose followed by 4,400 IU/kg hourly for 12 h." In Table 4 on the same page (see Table 1 below), the correct dosing regimen for urokinase in

Table 1 — Thrombolytic Agents in Venous Thromboembolism*

<table>
<thead>
<tr>
<th></th>
<th>Streptokinase</th>
<th>Urokinase</th>
<th>Tissue plasminogen activator</th>
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<tr>
<td>Stop heparin infusion. Start thrombolytic infusion when aPTT or thrombin time (TT) is ≤1.5 times control.</td>
<td>250,000 IU loading dose</td>
<td>4,400 IU/kg loading dose</td>
<td>100 mg (56 million IU) over 2 h</td>
</tr>
<tr>
<td>Streptokinase</td>
<td>100,000 IU/h maintenance</td>
<td>4,400 IU/kg/h maintenance</td>
<td></td>
</tr>
<tr>
<td>Urokinase</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tissue plasminogen activator</td>
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<tr>
<td>After terminating thrombolytic infusion, restart heparin infusion without a loading dose or with a small loading dose when aPTT or TT ≤1.5 times control.</td>
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</table>

*Duration of therapy: Streptokinase is recommended for 24 h infusion in pulmonary embolism, 48-72 h in deep venous thrombosis. Urokinase is recommended for 12-h infusion in pulmonary embolism, 24-48 h in deep venous thrombosis. Tissue plasminogen activator is recommended for a 2-h infusion in pulmonary embolism at a total dose of 100 mg.

pulmonary embolism should be shown as 4,400 IU/kg loading and 4,400 IU/kg/h maintenance for 12 h.

Thomas M. Hyers, M.D., F.C.C.P.,
Division of Pulmonology and Pulmonary Occupational Medicine,
St. Louis University School of Medicine,
St. Louis

REFERENCE


Risk of Aspiration Pneumonia in the Elderly

To the Editor:

In the elderly, aspiration is a common and serious problem. The morbidity and mortality that result from aspiration pneumonia are being recognized as major geriatric health problems. Recently, a marked depression of the cough reflex, one of the respiratory defense mechanisms, was shown in patients with aspiration pneumonia.1 We therefore examined the swallowing reflex, the cough reflex, and the mucociliary transport system and determined the critical levels of depression in the defense systems that lead to aspiration pneumonia.

The ten control subjects (mean age, 77 ± 3 [SE] years) were healthy volunteers and led active daily lives. The ten patients with dementia due to cerebral arterial sclerosis (mean age, 79 ± 3 years) were hospitalized, but they did not suffer from aspiration pneumonia. The ten patients with aspiration pneumonia (mean age, 78 ± 5 years) had all suffered at least one episode of observed aspiration with chest x-ray evidence of an inflammatory response in the dependent pulmonary segments. Computed tomographic scans and magnetic resonance images revealed various degrees of cerebral atrophy and lacunar infarction, but did not show any abnormalities in the bulbar region in both patient groups.

Cough response to citric acid was evaluated by determining the cough threshold as reported previously.1 The swallowing reflex was induced by a bolus injection of 1 ml of distilled water into the pharynx through a nasal catheter without the knowledge of the subject, as described by Nishino et al.4 The swallowing action was identified by visual observation of the characteristic laryngeal movement. The swallowing reflex was evaluated by the latency of response, which was timed from the injection to the onset of the swallowing action. Nasal clearance was examined by the saccharine particle method, which is reported to reflect tracheobronchial clearance.5 To eliminate any diurnal variation in responses, three sets of experiments were done at the same time in a random fashion on 3 days within 1 week.

The latent time of swallowing was 1.2 ± 0.1 (SE) s in the controls, 5.2 ± 0.6 s in the patients with dementia, and 12.5 ± 0.3 s in the patients with aspiration pneumonia. The threshold concentration of citric acid was 2.6 ± 0.4 (SE) mg/ml in the controls and 37.1 ± 16.7 mg/ml in the patients with dementia; all of them coughed at some point. By contrast, seven of the ten patients with aspiration pneumonia did not cough, even at the highest concentration of citric acid (360 mg/ml). All of the patients with aspiration pneumonia had a latent time of swallowing longer than 11 s and a threshold concentration of citric acid higher than 180 mg/ml (Fig 1). However, nasal clearance time did not differ significantly among the three groups (p > .30).

The present study shows the combined depression of the cough and swallowing reflexes in patients with dementia due to cerebral arterial sclerosis and in patients with aspiration pneumonia, the