Table 1—Distribution of Cryptococcal Isolates in 18 AIDS Patients

<table>
<thead>
<tr>
<th>Total no. of isolates</th>
<th>30</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebrospinal fluid</td>
<td>14</td>
</tr>
<tr>
<td>Blood</td>
<td>11</td>
</tr>
<tr>
<td>Lung</td>
<td>2</td>
</tr>
<tr>
<td>Bone marrow</td>
<td>1</td>
</tr>
<tr>
<td>Urine</td>
<td>1</td>
</tr>
<tr>
<td>Catheter tip/blood</td>
<td>1</td>
</tr>
</tbody>
</table>

Mycobacterium avium-intracellulare and pyogenic bacteria. All our patients were homosexual men and therefore similar to the patients described by Kovacs et al. who found primary pulmonary cryptococcosis in only one of 27 patients. Although exposure to pigeon droppings may, as proposed by the authors, result in an increased incidence of pulmonary cryptococcosis, it is difficult to understand why homosexual men would be less exposed than patients with other risk factors for AIDS. Furthermore, Eng et al. reported ten patients with AIDS and cryptococcal disease. Eight were intravenous drug abusers and only two were homosexual men (similar to Wasser's patients with risk factors other than homosexuality). None had pulmonary involvement (primary or secondary) with Cryptococcus neoformans.

It might also be of interest to point out that histoplasmosis is being described with increasing frequency, both from endemic and nonendemic areas, in patients with AIDS. This infection seems to present as pneumonitis (four of 12 or 33 percent of our patients) more often than cryptococcosis.

Based on these data which, in a fairly large number of patients, show that primary pulmonary cryptococcosis is extremely rare, it would be premature to conclude, as the authors have done, that this is a "frequent manifestation" of cryptococcal infection in AIDS patients.

Nancy Khordori, M. D., Faheem Butt, M. D., and Kenneth V. L. Rolston, M. D., M. D. Anderson Hospital and Tumor Institute; and The Institute for Immunological Disorders, Houston

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1. Wasser L, Talavera W. Pulmonary cryptococcosis in AIDS. Chest 1987; 92:692-95

Metastases to the Right Ventricle

To the Editor:

We have read with interest the article by Emmot et al. describing a metastatic malignant melanoma to the right ventricle causing inflow and outflow tract obstruction. A prior case involving an adrenal cell carcinoma with metastases to the right ventricle has been described by our group. Our patient presented with similar clinical symptoms and physical findings. Susception of inflow and outflow tract obstruction of the right ventricle was documented by echocardiography and subsequent angiography. Surgical removal of the mass followed by both external beam radiotherapy and chemotherapy resulted in a three-month survival from the time of surgery.

With the increasing availability of two-dimensional and Doppler echocardiography, as well as magnetic resonance imaging (MRI) over the past several years, valuable information regarding the extent of these tumors and hemodynamic parameters can be obtained noninvasively. These two cases illustrate that metastases to the right ventricle causing significant hemodynamic compromise do occur and that a variety of imaging techniques provide useful information on the overall extent of the tumor.

Brian W. Carlin, M. D., San Diego; Sinda Dianzumba, M. D., and Claude R. Joyner, M. D., Pittsburgh

REFERENCES

To the Editor:

The report by Dr. Carlin et al. describing metastatic carcinoma to the right ventricle causing inflow and outflow tract obstruction indeed supports the utility of multiple imaging modalities in evaluating cardiac masses. We were unaware of this previous report at the time of preparation of our manuscript but would like to thank Dr. Carlin and his colleagues for bringing it to our attention as well as the readers of Chest. We hope that knowledge of cases such as these will enhance awareness of potential applications of the new noninvasive imaging modalities available at the present time.

James L. Vacek, M. D., and William W. Emmot, M. D., University of Kansas Medical Center, Kansas City

REFERENCE


Allergic Urticarial Eruption, Leukocytosis and Abnormal Liver Function Tests Following Nifedipine Administration

To the Editor:

Nifedipine, a potent calcium antagonist, is widely used in the treatment of hypertension and angina. We describe a case of severe urticarial allergic eruption associated with leukocytosis and abnormal liver function tests following the administration of nifedipine for hypertension.

A 59-year-old man was being treated with methyldopa and chlorthalidone for hypertension and oral theophylline and inhaled salbutamol for airways obstruction. He presented with recent onset of depression and deterioration in his airways obstruction. Methyldopa was thought to be the most likely cause of the change in affect...
and nifedipine was substituted and the chlortalidone continued. His theophylline dosage was increased and salbutamol was continued. Oral steroid therapy had to be added a week later because of a further increase in Airways obstruction. A week later, on a reduced dose of prednisolone (15 mg daily) and the other above-mentioned drugs, he was admitted with a markedly erythematous maculopapular rash on his trunk, limbs, scalp and face with vesicle formation and exfoliation from ruptured bullae. He was afebrile, had a sinus tachycardia and was normotensive.

His white cell count was 36 x 10^9/L (polymorphs 91.3 percent, lymphocytes 7.4 percent, monocytes 1.3 percent). HB 16.3 g/dl, platelets 524 x 10^9/L SMAC profile was abnormal with alk phos of 224 (30 to 100), gamma GT 158, sodium 131 mmol/L (135 to 145), K 2.8 mmol/L (3.5 to 5.0), hI 83 mmol/L (95-105), Bicarb 35 mmol/L (22 to 28), glucose 12.5 mmol/L (2.8 to 8.3) and urate 582 umol/L (150 to 470). Culture of blood, nose and throat swabs and MSU were all sterile.

Skin biopsy showed edema of the dermis with moderately dense perivascular and interstitial inflammatory cell infiltrates, composed mainly of polymorphs and lymphocytes with occasional mast cells. These features are suggestive of a drug-induced allergic eruption. With cessation of nifedipine treatment and institution of high dose prednisolone therapy, hydrocortisone cream and antipruritics, the rash improved and the white cell count and liver function tests returned to normal.

A large number of skin and appendage disorders have been described following nifedipine administration. True urticaria and urticaria-exanthema are among the commonest reactions noted with bullous eruption, exfoliative dermatitis and erythema multiforme being less common.14 Urticarial eruptions are frequently misdiagnosed clinically as erythema multiforme. However, the lesions of urticarial allergic eruptions do not have target configuration and are usually purpuric. True urticaria tends to have a shorter clinical course and can be histologically differentiated from urticarial allergic eruption.2

Nifedipine is rarely hepatotoxic and hepatic reactions are thought to be due to hypersensitivity and as such are idiosyncratic, usually occurring after a latent period of one to four weeks.4 In our patient, the mild hyperglycaemic reaction may be steroid-related. However nifedipine cannot be ruled out.4 Hypokalaemia was attributed to his diuretic therapy (and possibly salbutamol) and responded promptly to potassium supplements. The rapid resolution of the reaction following withdrawal of nifedipine despite continuing all other medications suggests a central role for nifedipine as a direct toxic effect or, less likely, as an interaction with one of the other drugs.

We believe it is the first report of histologically-confirmed urticarial allergic eruption, hepatotoxicity and leukocytosis occurring in the same patient following administration of this drug.

M. Toner, M.D.,
Department of Pathology,
St. James Hospital; and
A. White, M.D.;
J. Moriarty, M.D., and
L. Clancy, M.D., FCCP,
Royal City of Dublin Hospital,
Dublin, Ireland

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2 Reports of side effects associated with the use of drugs. 1978-85. National Drugs Advisory Board (Ireland).
3 Ackerman AB. Histologic diagnosis of inflammatory skin diseases. Philadelphia: Lea and Febiger, 1979

Endotracheal Tube Placement

To the Editor:

Recently, Stewart and co-workers1 reported their use of a fiberoptic stylus to confirm proper endotracheal tube placement. We feel that some of their statements and recommendations may lead others to neglect time-tested maneuvers used to confirm endotracheal tube placement. Also, we wish to recommend two very reliable methods.

The authors advocate placing the endotracheal tube with the tip 20 cm from the incisors. This placement is too shallow and might produce an incomplete seal when the cuff is inflated, potentially allowing an air leak and increasing the risk of aspiration. Also, positioning the endotracheal tube at this depth may increase the risk of accidental extubation during transport. The endotracheal tube should be positioned (in men with the tip 23 to 24 cm from the incisors, a distance supported by the literature.2,3 The distance in women should be a centimeter or two less.

The authors discuss using this technique in brightly lit areas and advise shielding the neck to reduce the amount of ambient light. But we suspect that it may not be possible to sufficiently reduce the ambient light to permit reliable evaluation of tube placement during intubations in the field. Moreover, in our experience intratracheal placement of the lighted stylot in obese patients or others with large necks may not appear as the distinct, circumscribed glow described in the article.

This study, conducted in an ideal setting of low light and low performance anxiety, is markedly dissimilar to the typical cardiac arrest situation. We suggest that the correct response rate using auscultation of the chest would be as high, especially if performed in the same conditions as the study. One simple procedure to perform if doubt persists regarding tube location is to confirm with laryngoscopic examination that the endotracheal tube passes through the vocal cords.

For several years, capnometry has been readily available for use in operating rooms and it soon will be (if it is not already) a standard of care for anesthesia practice. Several companies market portable battery-operated capnometers—both qualitative and quantitative—which are suitable for use in almost any setting. All reliably detect end-tidal carbon dioxide, a finding inconsistent with gastric ventilation.

Another accurate and reliable method to determine endotracheal tube location is bronchoscopy. The carina is easily identified by neophyte bronchosocopists. Semi-disposable fiberoptic catheters are currently available. These can be used to determine endotracheal tube placement and thereby eliminate the need for and cost of chest roentgenogram.

Robert A. Strickland, M.D., and
John F. Butterworth IV, M.D.,
Bowman-Gray School of Medicine,
Winston-Salem, NC

REFERENCES
1 Stewart RD, LaRose A, Stoy WA, Heller MB. Use of a lighted stylot to confirm correct endotracheal tube placement. Chest 1987; 92:900-03

To the Editor:

I welcome Dr. Butterworth's comments and criticism of our paper.

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