carcinoma to reactivate latent *M. kansasii* disease, and (2) the presentation of these two processes simultaneously in the same patient, a heretofore-undocumented finding. These observations should alert physicians treating patients with lung neoplasia to obtain sputum specimens for acid-fast organisms both prior to and during treatment.

James F. Gettler, M.D., and Wafa Al-Esad, M.D., M.P.H., Division of Infectious Diseases, Harlem Hospital Center; 
College of Physicians and Surgeons of Columbia University, New York

REFERENCES

To the Editor:

We agree with Drs. Gettler and Al-Esad that there has been no specific report on simultaneous presentation of *M. kansasii* infection and small-cell carcinoma. Indeed, we found only one publication1 with specific reference to active *M. kansasii* infection coexisting with any primary lung malignancy. In that series of 44 patients with pulmonary isolates of *M. kansasii*, six were found to have bronchogenic carcinoma; no detailed histology of the malignancy was given. The scarcity of the report is the reason we are about to submit an article for publication on this association. We do not believe, however, that small-cell carcinoma is unique in its ability to reactivate latent *M. kansasii* disease, since this carcinoma represents only one case in our series in which the two diseases coexist.

James R. Zvetina, M.D., and Nitaya Maliwan, M.D., Edward Hines, Jr., Hospital, Hines, Illinois

REFERENCE

The Wonderful Floating Features in Medical Imaging of Chronic Aspirated Motor Oil in the Lung

To the Editor:

I read the paper of Dr. Van den Plas and colleagues1 with great interest. It involved a very rare case of chronic inhalation of a motor oil substance into the lung with gravity-dependent shifting phenomenon in chest imaging. It showed that the light oil substance may move freely into any portion of the lung by gravity with notably distant migration. All three figures show the same horizontal upper level accumulations. This indicates that a large amount of this light motor oil was freely movable inside the lung. Although some of the small particles of this particular oil substance were phagocytized within the alveolar macrophages, most of them were treated as nonabsorbable material in the lung in this particular case. A large portion of the oil was distributed in the lower part of the lung no matter what position the patient was in. The large volume of light motor oil did not adhere to any part of the lung but moved freely along the bronchial tree from top to bottom as the patient's position determined. It was evident that this oil substance could freely leave the air space and flow into the small bronchi even to the segmental bronchi and then disseminate to other segmental regions of the other lung. Therefore, to me, this kind of light flowing oil substance cannot be treated as a pneumonic-inducing material because it is foreign and isolated to the lung tissue. I am strongly against using the term "itis" in such cases. This case somewhat resembles a case described in the late 1950s where we had a chance to observe closely the rapid absorption of the intrapulmonary hemorrhages in leptospirosis icterohemorrhagica.2 As soon as antibiotics were started, the diffuse mottlings or even big patches in the lung promptly absorbed within 1 to 3 days, according to whether the disease processes were in the early or late stages. That is to say, the lesions vanished very quickly, so they were not pneumonic in nature. Therefore, we concluded that this was not an inflammatory process in the lung, but a toxic effect causing diffuse intrapulmonary hemorrhages. We prefer to use the term pulmonary manifestations in leptospirosis rather than leptospirochetotic pneumonia.

Although these two conditions are quite different in nature, the noninflammatory pathologic findings in the lung were the same. Therefore, this case of motor oil accumulation in the lung should not be catalogued as lipoid pneumonia. A word for its management: I suggest that it could be treated with pulmonary lavage, and I think that this light oil substance could be wiped out easily.

At any rate, this special case illustration is very interesting and wonderful in film reading, and I think it is a treasure in modern chest imaging.

Lian Bi Hu, M.D., Chengdu, Peoples Republic of China

REFERENCES

Public Health and Tuberculosis

To the Editor:

No one can disagree with the public health measures for the control of tuberculosis set forth by Drs. Dunlap and Bailey.1 I wish, however, that these doctors had mentioned the necessity of detaining uncooperative patients until they finish a course of effective therapy. Such a long-term detention measure, with constitutional protections, has just been adopted by the New York City Department of Health.2

Medicine is truly a kinder and gentler profession than the law, and physicians are not accustomed to forcing people to do things against their will. Also, public health officials tend to be politically liberal. But to control tuberculosis, compulsion will sometimes be necessary.

I understand that federal law provides public officials who act objectively reasonably and in good faith, with immunity from suit for deprivation of rights: in detention cases, the right to liberty.

You might like to hear about my own experience as a four-time federal civil rights defendant. I worked part-time in a jail for some 11 years. Prisoners have easy access to federal courts

326 Communications to the Editor
References


Oral Anticoagulant Therapy Recommendations

To the Editor:

It is regrettable that my warning against intense warfarin regimens, such as are recommended in Britain and in North America, was not taken into consideration in the update by Hirsh et al., which appeared in the October 1992 supplement of Chest. My warning contains the recommendations made by the Federation of Dutch Thrombosis Centers, which together provide oral anticoagulation laboratory control for about 250,000 patients receiving active treatment. It is on good grounds that the Federation recommends 3.0 international normalized ratio (INR) as the target for primary and secondary prevention of venous thrombosis and thromboembolism, 3.5 INR in case of recurrence under the former regimen and for patients at risk for a cardiogenic embolism from any source (including tissue heart valve replacement) and those with atherothrombotic disease, and 4.0 INR for patients with mechanical heart valve prosthesis; the risk of hemorrhage at such levels remains acceptable.

In sharp contrast to these recommendations, Hirsh et al propose to aim at levels between 2.5 and 3.5 INR in patients with artificial heart valves. In their argumentation, ironically, they refer to a recently completed randomized trial performed by the McMaster group, which shows that the degree of protection by oral anticoagulants when aiming at 3.0 to 4.5 INR was increased by the addition of aspirin in a dose of 100 mg/d without a significant increase in major bleeding or cerebral hemorrhage. In my view, Hirsh et al. herewith support Dutch policy to aim at 3.5 to 4.8 INR without the addition of aspirin.

Similar considerations hold for their argument for an optimal therapeutic range of 2.0 to 3.0 INR in patients with tissue heart valves; from the data presented by the McMaster group, one is justified in concluding that 2.0 to 2.5 INR and 2.5 to 4.0 INR are equally ineffective in the protection of patients against systemic embolization.

Another major mistake made by Hirsh et al is the statement that in the two large-scale studies of the secondary prevention of myocardial infarction performed in The Netherlands and in Norway, both with intensive anticoagulation, there was no increase in bleeding complications. The bleeding complications observed in the Dutch study were even considered worthy of separate publication.

Emil A. Loeliger, M.D.,
Department of Hematology,
Academisch Ziekenhuis,
Leiden, The Netherlands

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