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# Letters to the Editor

## Notes: a suitable case for audit

Sir,

The problems highlighted by Twigg *et al.*[1] constitute a crisis in the clerical standard of the case notes which is so profound as to negate most of the potential benefits of medical audit. The deficiencies in the clinical input would be largely corrected by a universal adoption of the problem-oriented approach to note keeping, in place of the present system of cryptic shorthand which totally fails to reflect the logic and complexity of clinical decision making. The chaotic state of filing of laboratory reports could be alleviated by investment in cumulative laboratory report sheets, colour coded according to the type of report. The problem of bulk arises mainly from the 'nursing process' input, principally because of the proliferation of A4 size proformas and charts designed to cater for various eventualities, including those of the most trivial kind. At the risk of 'political incorrectness', I would add the comment that the sheer volume of note keeping now expected of the nursing staff poses a threat not only to the sheer bulk of the case notes, but also to the time available for 'hands on' nursing care. The final problem is that of provision of adequate numbers of clerical staff. The reason why this remains insoluble is that it is rooted in the failure to perceive that the care of the case notes is an extension of patient care, and that clerical standards are a reflection of clinical standards of care. This aspect of clinical management has been largely ignored because we do not address these problems on an ongoing basis. Surely the best preventive measure would be for consultants to do discharge summaries on randomly selected cases routinely, so as to acquire first-hand experience of the defects of the system?

O.M.P. Jolobe
*Department of Medicine for the Elderly,*
*Tameside General Hospital,*
*Fountain Street,*
*Ashton-under-Lyne OL6 9RW, UK.*

### References

1. Twigg, J., Briggs, T., Parker, C. *et al.* Notes: a suitable case for audit. *Postgrad Med J* 1993, **69**: 578-580.

## Myositis related to mycoplasma infection

Sir,

The most recent of a small number of case reports proposed a link between *Mycoplasma pneumoniae* infection and myositis was published in the Journal in 1990.[1] We present a supportive case.

A 21 year old male was admitted 5 days after the onset of fevers, dizziness, abdominal cramps and headache. Additional symptoms included nausea, vomiting, diarrhoea, dyspnoea and a severe dry cough. Workup at another hospital included stool and blood cultures, hepatitis serology, abdominal ultrasound, sigmoidoscopy and lumbar puncture, all of which were negative. During this workup, serum lactic dehydrogenase (LDH) and aspartate transaminase (AST) rose from normal levels to 1,599 IU/l and 370 IU/l, respectively. On admission, the patient denied arthralgias, myalgias, sore throat and risk factors for human immunodeficiency virus.

He was ill-appearing, tachypnoeic with a temperature of 103°F. Blood pressure was 110/80 mmHg. The neck was supple with tenderness and the extremities were non-tender.

Laboratory data showed mild hypoxia with respiratory alkalosis and modest leucocytosis. Serum LDH was 1,417 IU/l, AST 615 IU/l and creatine kinase >16,000 IU/l. Urine tested positive for myoglobin. Chest X-ray showed a diffuse interstitial pattern.

Many potential aetiologies were considered and investigated including *Legionella*, toxoplasmosis, cytomegalovirus, adenovirus, enterovirus and influenza. The only positive findings were for IgG and IgM mycoplasma titres.

The patient was treated with copious intravenous fluids, alkalinization and intravenous erythromycin, and recovered fully. The patient was lost to follow-up and convalescent titres could not be obtained.

We found three English language references reporting a relationship between *M. pneumoniae* infection and myositis.[1-3] The nature of this association is not known. Antibodies to smooth muscle in patients with *M. pneumoniae* have been reported; however, this may be the result of tissue damage rather than the cause.[2] Direct tissue invasion has been hypothesized but not demonstrated.[1]

Spillane and Chessner provide an alternative view of this phenomenon.[4] The patient from Bennett's case report[1] was readmitted with evidence of myositis, this time in apparent association with hepatitis A. The authors surmised that the patient had a tendency to develop myositis as a non-specific response to acute infections.

Although of limited significance due to lack of follow-up data, we felt it important to add our case to the small number of reported associations between *M. pneumoniae* and acute myositis. Whether a specific complication or non-specific response, increasing awareness of this relationship may result in the recognition of more cases and greater insight into its pathophysiology.

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Alwin F. Steinmann
Carolyn Aks
*Division of General Internal Medicine*
*Albany Medical College,*
*Albany, NY 12208, USA.*