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2 Wilcox PA, Potgieter PD, Bateman ED, Benatar SR. Rapid diagnosis of sputum negative miliary tuberculosis using flexible fibreoptic bronchoscope. Thorax 1986; 41:681-84


To the Editor:

Dr. Watson's major observation appears to be regarding the value of histopathologic findings of granulomata in biopsy specimens in patients with miliary tuberculosis.1 We did not consider biopsies which demonstrated noncaseating granulomata as diagnostic for tuberculosis due to the nonspecific nature of such a finding. Tubercular granulomata (as opposed to those in other granulomatous disorders) show central caseating necrosis, which is considered the pathologic hallmark of tuberculosis.4 Hence, we had considered the biopsy as positive only in the presence of granulomata with caseous necrosis. Willcox et al2 established a rapid diagnosis in 27 of 34 patients. In six of these, the diagnosis was based exclusively on the presence of caseating granulomata, which was later substantiated by response to treatment. Although all our patients also responded to antitubercular therapy, in that sense response to treatment neither makes the diagnosis early nor definitive. Burke et al3 have similarly considered the presence of caseating granulomata as diagnostic (Table 1, patients 4 and 5).

Fibrebronchoscopy was not repeated in patients in whom a rapid diagnosis could not be established and they were put on antitubercular therapy, to which they responded favorably. Dr. Watson's observation on the high probability of miliary tuberculosis in patients with this clinical, radiologic and histopathologic appearance is relevant in our setting. In the virtual absence of granulomatous mycotic infections in India, the differential diagnosis of miliary opacities is considerably reduced. Conditions such as tropical pulmonary eosinophilia, bronchopneumonia, occupational lung disease and pulmonary alveolar microlithiasis can be ruled out by careful history-taking and other relevant investigations. The only real diagnostic dilemma is with miliary sarcoi—a rare entity in India. The clinical picture and presence of caseating granulomata in biopsy specimens clinch the issue in favor of miliary tuberculosis in the absence of smear and Mantoux positivity in most of these patients. Hence, with a high prevalence and incidence of tuberculosis in India, a physician treating patients with miliary opacities for tuberculosis in the relevant clinical setting, even without histopathologic evidence, would more often be right than wrong.

We obtained transbronchial biopsies blindly, without fluoroscopic guidance, due to lack of facilities. Although fluoroscopic guidance makes the procedure safer,4 the operator's experience may compensate for this lack of facilities to a large extent. However, there does not appear to be any evidence in the literature to suggest that it increases biopsy yield.

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3 Wilcox PA, Potgieter PD, Bateman ED, Benatar SR. Rapid diagnosis of sputum negative miliary tuberculosis using flexible fibreoptic bronchoscope. Thorax 1986; 41:681-84


5 Simpson FG, Arnold AG, Purvis A, Belfield FW, Muer MF, Cooke NJ. Postal survey of bronchosopic practise by physicians in the United Kingdom. Thorax 1986; 41:311-17

Fat Emulsion and ARDS

To the Editor:

In the June, 1989 issue of the Chest (95:1278-81), Venus et al reported the effects on hemodynamics and gas exchange after administration of fat emulsion infused intravenously (3.0 ± 0.3 mg/ kg/min) in 19 patients with ARDS. The authors described a significant reduction in PAO2/FIO2, and an increase in MPAP (mean pulmonary artery pressure), PVR (pulmonary vascular resistance) and Qva/Qt (pulmonary venous admixture). Furthermore, they found that Qva/Qt increased to a greater extent in septic vs non-septic ARDS patients, while the magnitude of increased MPAP was not influenced by the presence or absence of septicemia.

We would like to report our findings obtained after intravenous infusion of 20 percent Intralipid emulsion in six patients suffering from ARDS (Table 1) in which, in contrast to the Venus et al report, we did not observe any significant change in hemodynamic and gas exchange measurements.

We maintain that the discrepancies between our results and that of Venus et al are due to different protocols involved in the two studies. 1) We administered the same amount of 20 percent Intralipid (500 ml) twice as fast (4 hours instead of 8). 2) Our patients had less severe ARDS (mean value of Qva/Qt was 13.9 vs 20.6 percent). 3) We measured hemodynamics and gas exchange in our patients more often than Venus et al did. We believe that intervals of 8 and 3 to 4 h between measurements (immediately prior to and following completion of Intralipid infusion, respectively, in the report) are too long for such critically ill and unstable patients.

In brief, it is likely that the conflicting results could be attributed mainly to the infusion rate. Skie et al have suggested that during slow lipid infusion (eg, 3 mg/kg/min for eight hours, similar to Venus et al's report) there may be a net increase in vasodilatory and anti-inflammatory prostaglandins (resulting in a release of HPV and hence an increase of Qva/Qt). In addition, after the administration of a bolus or rapid infusion of lipids (eg, 8 to 10 mg/kg/min), fatty acid substrate may overwhelm the effect of vasodilatory prostaglandin (PGE2 and PGL2) production, resulting in increased production of vasopressor and inflammatory prostaglandin metabolites (eg, thromboxane A2).

An intermediate fat emulsion infusion rate, like in our experiments (500 ml of 20 percent Intralipid in 4 h, about 6.0 mg/kg/min), may result in an equilibrium between vasodilating and vasoconstricting prostaglandin production without an effect on hemodynamics or gas exchange in patients with ARDS. Hence our findings might suggest that fat emulsion administration to critically ill patients should be done relatively fast, if we wish to prevent effects on gas exchange.

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Ch. Roussos, M.D.,
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University of Athens Medical School,
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Table 1—Cardiopulmonary Effects of Intralipid Infusion in Six Patients with ARDS (4 Septic, 2 Noneptic)*

<table>
<thead>
<tr>
<th></th>
<th>Before Control</th>
<th>15 min</th>
<th>30 min</th>
<th>1 hr</th>
<th>2 hrs</th>
<th>4 hrs</th>
<th>5 hrs</th>
<th>8 hrs</th>
</tr>
</thead>
<tbody>
<tr>
<td>PaO₂/FiO₂</td>
<td>239 ± 40</td>
<td>236 ± 42</td>
<td>241 ± 42</td>
<td>207 ± 25</td>
<td>242 ± 39</td>
<td>228 ± 43</td>
<td>233 ± 35</td>
<td>229 ± 32</td>
</tr>
<tr>
<td>PaO₂</td>
<td>110 ± 13</td>
<td>109 ± 14</td>
<td>111 ± 14</td>
<td>97 ± 9</td>
<td>114 ± 16</td>
<td>105 ± 14</td>
<td>109 ± 12</td>
<td>107 ± 11</td>
</tr>
<tr>
<td>Qva/QT</td>
<td>13.9 ± 5.3</td>
<td>13.3 ± 5.9</td>
<td>13.5 ± 5.9</td>
<td>14.4 ± 2.1</td>
<td>9.9 ± 3.2</td>
<td>13.9 ± 3.4</td>
<td>12.4 ± 4.4</td>
<td>12.6 ± 4.7</td>
</tr>
<tr>
<td>MPAF</td>
<td>30.3 ± 4.1</td>
<td>30.0 ± 3.8</td>
<td>30.1 ± 3.8</td>
<td>28.7 ± 2.6</td>
<td>28.0 ± 2.6</td>
<td>28.1 ± 2.5</td>
<td>28.2 ± 2.5</td>
<td>28.7 ± 2.9</td>
</tr>
<tr>
<td>CO</td>
<td>5.3 ± 1.3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5.3 ± 0.5</td>
<td>5.2 ± 0.5</td>
<td></td>
</tr>
<tr>
<td>PAPD-PW</td>
<td>8.0 ± 5.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>8.3 ± 3.0</td>
<td>7.0 ± 3.5</td>
<td></td>
</tr>
<tr>
<td>PVR</td>
<td>262 ± 121</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>251 ± 88</td>
<td>244 ± 94</td>
<td></td>
</tr>
<tr>
<td>SVR</td>
<td>1066 ± 67</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1190 ± 42</td>
<td>1133 ± 60</td>
<td></td>
</tr>
</tbody>
</table>

*Mean ± SEM.

p<0.05.

REFERENCE


To the Editor:

I would like to thank Dr. Roussos and colleagues for their comments on our report regarding effects of Intralipid infusion on hemodynamic and gas exchange of patients with ARDS (Chest 1989; 95:1278-81).

I read Roussos et al’s findings in six patients with interest. I cannot explain why, in contrast to our report, Roussos et al’s patients did not show any significant change in hemodynamics and gas exchange after administration of 500 ml Intralipid. Based on the limited information about Roussos et al’s study group, it seems that those patients did not suffer from severe lung injury or sepsis. Several studies have shown that sepsis causes impairment in plasma clearance rate of intravenous fat emulsion. This may explain the difference in results. It is noteworthy that mean values for PVR, MPAF and PAD-PW suggest the presence of pulmonary hypertension in Roussos et al’s study subjects. Also, two hours through the infusion, Roussos et al’s data shows a significant decrease in Qva/QT (from 13.9 ± 5.3 to 9.9 ± 3.2) without any significant change in PaO₂, PaO₂/FiO₂ or MPAF. I would be interested to know Dr. Roussos thoughts about the reasons for the observed decrease in intrapulmonary shunting only in data obtained 2 hours through Intralipid infusion.

I do not believe that the faster infusion rate utilized in your patients can explain the discrepancies between our results. In fact, the present data in the literature suggest that fast infusion of fat emulsion will cause more pronounced hypoxemia. There is some information (Hageman and Hunt. Clin Chest Med 1986; 7:69) that suggest that the observed pulmonary changes during fat emulsion infusion is due to stimulation of prostaglandin synthesis. It is also suggested that effects of IV fat emulsion may vary depending on the dose and duration of infusion. During slow infusion, arachidonic acids will convert to endoperoxide intermediate PGE₂, and then preferentially use PGE₂ and PGF₂α routes of conversion. PG₂α is a potent pulmonary and systemic vasodilator. On the contrary, during fast infusion, excessive amount of arachidonic acid may overwhelm the enzymes for PGE₂ and PGF₂α route and cause a net increase in vasoconstrictive prostaglandins (e., Thromboxane). The prostaglandin mediated pulmonary vasoconstriction then causes increase in V/Q mismatch which can explain the observed hypoxemia during fat emulsion infusion.

We have tested this hypothesis recently by comparing the cardiopulmonary effects of Intralipid infusion in two groups of critically ill patients. Group 1 received 500 ml of 20 percent Intralipid solution over six hours. Group 2 received 500 ml of 20 percent Intralipid solution over 24 h. MPAF and Qva/QT did not change in group 2 patients, while it increased in group 1 patients. We also measured the metabolites of PG₁₂ (6 Keto PGFa) and thromboxane (TXB₂) in these patients. In group 2 patients, the level of 6 Keto PGFa significantly increased. These results and other available data in the literature suggest that slow infusion of Intralipid may prevent the observed hypoxemic effect in critically ill patients by allowing the increase in vasoconstricting prostaglandins.

As can be seen, our experience suggest the exact opposite of your group conclusion. We believe that fat emulsion administration to critically ill patients should be done slowly.

Bahman Venus, M.D., F.C.C.P.,
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Scimitar Syndrome

To the Editor:

In the Boentgenogram of the Month entitled "Dextrocardia?" Mannes et al report a case of an anomaly of the right upper lobe bronchus.¹

The chest film and bronchogram are typical of the venolobar syndrome (also known as hypogenetic right lung or scimitar syndrome). Radiographic findings in this syndrome are a small right lung composed of two lobes with the main pulmonary artery superior to the right main bronchus (mirror image isomerism) and secondary cardiac dextroposition.² Associated findings may include partial anomalous pulmonary venous return below the diaphragm (a scimitar), pulmonary sequestration and diaphragmatic anomalies including hernia, evagination, cysts and accessory diaphragm.³

Although patients are often asymptomatic, dyspnea on exertion and repeated bronchopulmonary infections can be seen.⁴

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