Response to Letter re: Refeeding Syndrome in Adults Receiving Total Parenteral Nutrition: An Audit of Practice at a Tertiary UK Centre

Konstantinos C. Fragkos, 1 Simona Di Caro, 1 Shameer J. Mehta, 1 Farooq Rahman 1

1 Intestinal Failure Service, Department of Gastroenterology, University College London Hospitals NHS Foundation Trust, 250 Euston Road, London NW1 2PG, United Kingdom

Correspondence to:
Dr Farooq Rahman
Intestinal Failure Service, Department of Gastroenterology
University College London Hospitals NHS Foundation Trust
250 Euston Road, London NW1 2PG, United Kingdom
e-mail: farooq.rahman@nhs.net
Tel. +44 (0) 20344 79311, Fax: +44 (0) 20344 79217

Dear Editor,

We thank Wong and Lew [1] for their insightful comments. Refeeding syndrome (RFS) is a condition where definitions are highly heterogeneous among studies with some studies relying only on electrolyte disturbances with different cut-offs, and others also integrating clinical parameters into the definition [2]. These varying cut-offs alongside varying definitions produce an heterogeneous incidence of RFS [3]. Hypophosphatemia has been commonly used for defining RFS, which is arguably a broader syndrome that includes electrolyte abnormalities in addition to clinical symptoms [2]. In our paper [4], we explored hypophosphatemia (with cut-offs from Ahmed et al. [5]), alongside other electrolyte abnormalities, in low and high risk RFS syndrome patients receiving total parenteral nutrition, as defined by the National Institute for Health and Care Excellence guidelines [6]. Hypophosphatemia was not used as the sole defining criterion for RFS. According to the definition suggested by Wong and Lew [1] of having serum phosphate level reduced by more than 0.16 mmol/L to below 0.65 mmol/L after initiating nutrition support, six patients (7.5%) experienced RFS by 72 hours and ten patients (12.5%) experienced RFS by 168 hours. When examining hypophosphatemia with the cut-offs suggested by Wong and Lew [1], distribution is not very dissimilar to the classification we followed (Table 1), with the new hypophosphatemia incidence being 20.0%, which is expected by a decreased lower normal cut-off of 0.71 mmol/L compared to our 0.85 mmol/L. Finally, the complications suggested by Wong and Lew [1] were not analysed as part of the present study. The present study examined abnormalities in terms of metabolic measurements and not clinical signs.

Table 1. Phosphate level distribution with cut-offs suggested by Wong and Lew [1].

<table>
<thead>
<tr>
<th>Phosphate plasma levels</th>
<th>Low risk RFS (n=20)</th>
<th>High risk RFS (n=60)</th>
<th>Total (n=80)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal/High (&gt; 0.71 mmol/L)</td>
<td>20 (100.0%)</td>
<td>44 (73.3%)</td>
<td>64 (80.0%)</td>
</tr>
<tr>
<td>Hypophosphatemia</td>
<td>0 (0.0%)</td>
<td>16 (26.7%)</td>
<td>16 (20.0%)</td>
</tr>
<tr>
<td>Mild (0.55-0.71 mmol/L)</td>
<td>0 (0.0%)</td>
<td>9 (15.0%)</td>
<td>9 (11.3%)</td>
</tr>
<tr>
<td>Moderate (0.32-0.54 mmol/L)</td>
<td>0 (0.0%)</td>
<td>6 (10.0%)</td>
<td>6 (7.5%)</td>
</tr>
<tr>
<td>Severe (&lt; 0.32 mmol/L)</td>
<td>0 (0.0%)</td>
<td>1 (1.7%)</td>
<td>1 (1.2%)</td>
</tr>
</tbody>
</table>
References


