C-reactive protein in childhood dermatomyositis

R. H. HAA S, R. F. D Y C K,* V. D U BO W IT Z, A N D M. B. P E P Y S*

From the Department of Paediatrics and Neonatal Medicine (Institute of Child Health), and the
*Immunological Medicine Unit, Department of Medicine, Royal Postgraduate Medical School, Hammersmith
Hospital, London W12 0HS

SUMMARY Serum levels of C-reactive protein (CRP) were determined in 9 patients with childhood
dermatomyositis. Four children were seen during clinical relapse and all had serum CRP levels less
than 1 mg/l. In addition direct immunofluorescent staining of muscle biopsies from 4 patients
showed no evidence of CRP deposition in muscle tissue. Such patients appear to be able to produce
CRP in response to acute infections, and it is suggested therefore that the pathological process in
childhood dermatomyositis may not induce a significant CRP response.

C-reactive protein is the classical acute-phase reactant, and although produced nonspecifically in
response to tissue injury there is evidence that the levels of CRP which are attained may vary signifi-
cantly between different diseases.1-3. Dermatomyositis is characterised by extensive inflammation
and muscle necrosis, but in the only case in which it has previously been reported serum CRP was not
detected, albeit by an insensitive assay.4

We report here that among 9 cases of childhood dermatomyositis, 4 (cases 1, 2, 3, 4) seen during an
active phase of their disease had serum CRP levels of less than 1 mg/l (Table 1). Three children (cases 6, 7,
8) had ‘burnt-out’ disease. One (case 7) was well apart from widespread calcinosis and no serum CRP
was detected. When subsequently reassessed with healing local skin ulcers, the CRP level was 4 mg/l. The
second child (also with calcinosis) (case 6) was well and had no detectable CRP, but the third patient
(case 8) with a paronychia on one toe had an elevated CRP of 20 mg/l. Patient 9 had minimally elevated
levels of CRP on 2 occasions, while patient 5, whose disease was well controlled by steroids, was clinically
well with a CRP of 57 mg/l when seen for review as a day patient. A subclinical infection could not be ruled
out, and a second CRP determination done 4 months later was 10 mg/l.

CRP was not detected by direct immunofluorescence in the muscle biopsies of the 4 children (cases 3,
4, 6, 7) tested or in normal muscle tissue. Complement profiles were within normal limits in all patients
in whom they were measured.

The results demonstrate that in children active involvement of muscle tissue in the inflammation and
necrosis of dermatomyositis is not associated with appreciable elevation of the serum CRP concentra-
tion. This is not apparently due to deposition of CRP in the damaged muscle, nor to inability to produce
CRP, since at least one of the patients had a significant response to an intercurrent infection.

Our observations extend the range of inflammatory disorders in which even extensive tissue damage
is associated with only a modest elevation of serum CRP. This is the case in both systemic lupus
erythematous4,8,10-13 and ulcerative colitis8 and contrasts sharply with the high CRP levels which are
regularly seen in active cases of rheumatoid arthritis2,12,15 and Crohn’s disease respectively.14

Although patients with uncomplicated SLE have been reported with high CRP values2,16 all the published
results confirm that this is exceptional.15,17 Measurement of serum CRP is therefore a valuable
aid in the diagnosis of intercurrent infection in febrile
patients with SLE,16 and this may also be true in
dermatomyositis.

Accepted for publication 4 September 1981.
Correspondence to Dr M. B. Pepys, Immunological Medicine Unit,
Department of Medicine, Royal Postgraduate Medical School, Du
Cane Road, London W12 0HS.

We thank Mrs Christine Hutson for technical assistance. This work
was supported in part by MRC programme grant G979/51 to M.B.P.
R.F.D. was supported by the MRC of Canada.
### Table 1  Clinical details and C-reactive protein levels in patients with childhood dermatomyositis

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age at onset</th>
<th>Muscle pain †</th>
<th>Muscle tenderness †</th>
<th>Stiffness joint pain †</th>
<th>Rash †</th>
<th>Malaise †</th>
<th>Total score</th>
<th>Muscle strength † % normal</th>
<th>Prednisone mg/day</th>
<th>Comments</th>
<th>Creatine phosphokinase (normal &lt; 120 IU/l)</th>
<th>ESR mm, CRP mg/l†</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Male</td>
<td>10 yr 11 mo</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>4</td>
<td>41</td>
<td>5</td>
<td>32</td>
<td>Possible relapse</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 Male</td>
<td>14 yr 10 mo</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>8</td>
<td>32</td>
<td>15</td>
<td>10</td>
<td>Relapse</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 Female</td>
<td>5 yr</td>
<td>2</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>12</td>
<td>33</td>
<td>0</td>
<td>10</td>
<td>Remission, deep venous thrombosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 Female</td>
<td>3 yr 8 mo</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>87</td>
<td>30</td>
<td>5</td>
<td>Relapse</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 Male</td>
<td>13 yr 8 mo</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>5</td>
<td>63</td>
<td>21</td>
<td>10</td>
<td>Recovering from acute relapse</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 Male</td>
<td>8 yr 11 mo</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>88</td>
<td>10</td>
<td>25</td>
<td>Remission</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7 Female</td>
<td>13 yr 2 mo</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>1</td>
<td>4</td>
<td>89</td>
<td>25</td>
<td>32</td>
<td>Inactive calcinosis, reducing steroids</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 Female</td>
<td>13 yr 8 mo</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>91</td>
<td>45</td>
<td>30</td>
<td>Burnt out disease, calcinosis, and contractures</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9 Female</td>
<td>15 yr 6 mo</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>1</td>
<td>3</td>
<td>26</td>
<td>6</td>
<td>4</td>
<td>Scleroderma features, pulmonary infiltrates</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10 Female</td>
<td>15 yr 8 mo</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>36</td>
<td>8</td>
<td>11</td>
<td>Clinically well</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Diagnosis established by clinical features of muscle weakness, rash and malaise, together with muscle tenderness, fatigue, or calcinosis, supported by measurements of serum creatine phosphokinase, electromyography, and muscle biopsy.*

†Scores of 0–3 assigned for severity of each of the features shown.

†Measured by electroimmunoassay. *Among normals 90% are < 3 mg/l and 99% < 10 mg/l.*
C-reactive protein in childhood dermatomyositis

References

1 Pepys M B. C-reactive protein fifty years on. Lancet 1981; i: 653-7.
C-reactive protein in childhood dermatomyositis

R. H. Haas, R. F. Dyck, V. Dubowitz and M. B. Pepys

Ann Rheum Dis 1982 41: 483-485
doi: 10.1136/ard.41.5.483

Updated information and services can be found at:
http://ard.bmj.com/content/41/5/483

Email alerting service

These include:
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/