[Ascorbic acid in the complex therapy of acute pneumonia].
Mochalkin NI.

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https://www.mv.helsinki.fi/home/hemila
https://www.mv.helsinki.fi/home/hemila/VitC_pneumonia.htm
https://www.mv.helsinki.fi/home/hemila/CP.htm  (Cochrane review)
https://www.mv.helsinki.fi/home/hemila/CP  (Cochrane review references)

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The Russian text is available at:

In two subsequent reports,
Mochalkin (1974) and Mochalkin (1975) published the same data, see translations:
Acute pneumonia is one of the most frequent/common diseases of the internal organs under both peace and war conditions. During flu periods, the incidence of acute pneumonia increases, sometimes up to 20% (V.V. Vlasov, 1958; N.V. Sergeev, 1961).

The widespread use of antibiotic therapy has dramatically decreased/reduced/lowered the mortality and gravity of the disease, but the problem of acute pneumonia remains because, first of all, the rate still runs high (0.3 – 0.4%) and, second, a growing number of patients has exhibited a clear tendency toward either a prolonged period of illness or a more chronic form (N.S. Molchanov, 1964, 1965; V.V. Medvedev, 1964; A.Ya. Zigelnik et al., 1964; L.V. Chireikin, 1967; V.P. Silvestrov, 1968).

Treating acute pneumonia with antibiotics was especially successful during the first decade of their use, resulting in a sharp decline in mortality (from 10-15% to less than one percent), a shorter rehabilitation period, the absence of strictly partial localization in crupous pneumonia, and the frequency of segmental deterioration (N.S. Molchanov, 1966). Drawing on extensive clinical experience, however, a number of drawbacks with antibiotic therapy have emerged, such as the progressively diminishing effectiveness of antibiotics (N.S. Molchanov, 1964), suppression of immuno-biological body reactivity (I.I. Daal-Berg et al, 1959), disruption of the body’s protein, carbohydrate and vitamin exchange (A.N. Klimov, 1959; Ya.B. Maksimovich, 1961; V.A. Novikov, 1967, et al), and toxico-allergic side reactions (A.L. Libov, 1958; H. Planeljes, A. Kharitonova, 1965; P.I. Shamarin, 1966). Such drawbacks retard the process of reverse development in pneumonia locales under treatment with antibiotics (L.I. Moskvicheva, 1961; P.K. Lipatova, 1964). Consequently, the need has emerged to find new ways of raising the effectiveness of antibiotic therapy in treating acute pneumonia.

We focused our attention on studying the exchange of ascorbic acid in the process of treating pneumonia with antibiotics, and on how it affects the clinical process and its results. The existing literature has presented this problem rather poorly, and has provided/offered no
indication/suggestion/recommendation for a rational/reasonable dose of vitamin C that could prevent both its deficiency and hyper-vitaminization. From the viewpoint of modern science, the following two scenarios are not only undesirable but even dangerous: (1) the hyper-vitaminization induced by antibiotic use or by other causes, and (2) that induced by an overdose of ascorbic acid, which can occur with prescriptions for large doses of ascorbic acids. Unjustified prescriptions for overdoses are known to disrupt the functions of certain endocrine glands (M.L. Rokhlina, 1949; E.P. Samborskaya, 1967) and even to destroy kidney glomerules followed by the onset of hypertension (V. Merezhkovsky, 1963; M.D. Mashkovsky, 1967).

The research tasks we set were as follows: (1) to study the effect of the planned therapeutic doses of certain antibiotics (penicillin, streptomycin, penicillin combined with streptomycin, and tetracycline) used in treating acute pneumonia on the patients’ vitamin C balance and on the clinical development of the disease; (2) to establish the minimal and maximal diurnal doses of ascorbic acid against the diurnal antibiotic dose necessary for maintaining the patient’s optimal vitamin C level; (3) to compare the basic clinical-laboratory parameters of patients treated with antibiotics without ascorbic acid prescriptions and those treated with antibiotics combined with various vitamin C doses.

The group of patients comprised 140 males diagnosed with acute pneumonia hospitalized during the first two days of onset of the disease: 38 patients were between 20-30 years of age, 23 were aged 31-40, 26 were aged 41-50, 37 were aged 51-60, and 16 were over 60 years old. Depending on the mode of basic treatment, the patients were divided into three groups:

- Group I (70 patients) was treated with antibiotics without ascorbic acid (25 patients were treated with penicillin, 15 with streptomycin, 15 with penicillin and streptomycin, and 15 with tetracycline);
- Group II (39 patients) was treated with antibiotics combined with vitamin C (minimal dose*: 50 mg per 100 000 antibiotic units) (15 patients were treated with penicillin, 8 with streptomycin, 8 with penicillin and streptomycin, and 8 with tetracycline)
- Group III (31 patients) was treated with antibiotics combined with ascorbic acid (maximum dose: 100 mg per 100 000 antibiotic units) (10 patients were treated with penicillin, 7 with streptomycin, 7 with penicillin and streptomycin, and 7 with tetracycline).

All patients were tested under equal conditions of placement, care, and nutrition, and were subjected to a complex therapy which included antibiotics in the following dosages: 100 000 units of penicillin every 4 hours and 500 000 units of streptomycin 1-2 times per day intramuscularly; 100 000

* We established the minimal and maximal (optimal) doses of ascorbic acid per prescribed dose of antibiotic through experimentation on animals and testing on healthy, willing humans.
units of tetracycline (oxytetracycline) every 4 hours orally. Ascorbic acid powder was also taken orally. Both antibiotics and ascorbic acid were used for 10 days.

To monitor the effectiveness of the employed methods of treatment, we used the following parameters: dynamics of temperature normalization, erythrocyte sedimentation rate (ESR), leucocyte quantity in the peripheral blood, timing of wet rattle disappearance, duration of roentgenologically-determined changes in the lungs, and the mean period of recovery.

The study of the dynamics of changes in the ascorbic acid content of blood plasma and of its release with urine began with the establishment of the initial level. On the 5th, 10th, 15th, 20th, and 30th days of using antibiotics, we determined the vitamin C content in blood plasma (Farmer & Abbot method) and urine (N.S. Zheleznyakova method). The data obtained were subjected to statistical treatment. Table 1 below shows the absolute mean values of the concentration of ascorbic acid in blood plasma and urine.
Table 1

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of patients</th>
<th>Ascorbic acid level in blood plasma (mg%)</th>
<th>Ascorbic acid release in urine (mg/hour)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Time from treatment initiation (days)</td>
<td>initial 5</td>
<td>10</td>
</tr>
<tr>
<td>I</td>
<td>70</td>
<td>0.73</td>
<td>0.42</td>
</tr>
<tr>
<td>II</td>
<td>39</td>
<td>0.73</td>
<td>0.58</td>
</tr>
<tr>
<td>II</td>
<td>31</td>
<td>0.71</td>
<td>0.70</td>
</tr>
</tbody>
</table>

As Table 1 shows, both the vitamin C level in blood plasma and the release of ascorbic acid with urine changed sharply in the process of treatment, especially in group I where the decrease in the level of ascorbic acid was especially pronounced (up to 56.16%). It should be noted that the increase in the vitamin deficit goes hand in glove with the duration of antibiotic use. In group II, the decrease was less pronounced (up to 79.45%), while in group III, the balance of vitamin C remained practically unchanged.

From day 10 of the treatment, the cancellation of antibiotics and ascorbic acid, and hence the cessation of the acute inflammation process in the lungs, led to a tendency to restore the vitamin C deficiency in the organism. However, the patients in group I reached 93.15% of the initial level of ascorbic acid in blood plasma only by the 30th day of treatment, while the patients in group II reached 97.26% by the 20th day. The mg/hour monitoring of ascorbic acid release with urine appears consistent with the dynamic of its level in the blood.

Therefore, a course treatment of therapeutic doses of penicillin, streptomycin, and tetracycline results in a considerable decrease of the vitamin C content in blood and its release with urine.

The latest literature contains data on significant disruptions of the vitamin balance in patients with respiratory problems (B.H. Khamzaliev, 1956; A.A. Stroganova, 1963; R.G. Voronina, 1965; O.I. Lasitsa, 1967; V.A. Novikov, 1967, etc.), as well as on a direct connection of the disease gravity, duration, and results with the body’s provision of vitamins in general and vitamin C in particular (Z.M. Volynsky, 1954; P.A. Alisov et al., 1960; S.M. Ryss, 1963; S.I. Ashbel et al., 1967). From such a viewpoint, the differences in the dynamics of the basic clinical-laboratory parameters (in %), which characterize the process of acute pneumonia in various group presented in Table 2, become clear.

As Table 2 shows, the most favorable course of treatment of acute pneumonia is observed in
the group of patients treated not only with antibiotics but with ascorbic acid as well, especially at the optimal dosage. The patients of group I, who received antibiotics with no ascorbic acid, exhibited significantly retarded normalization of clinical symptoms. Such a comparison demonstrates/shows/suggests that the use of vitamin C facilitates the treatment of patients with acute pneumonia.

Table 2

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of Patients</th>
<th>Antibiotic (10^6 units)</th>
<th>Vitamin C (g)</th>
<th>Temperature normalization by 10th day</th>
<th>Wet rattle gone</th>
<th>ESR</th>
<th>Leucocytes</th>
<th>Roentgen monitoring</th>
<th>Duration of recovery (days)</th>
<th>Mean (SE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>70</td>
<td>0.5-1.6</td>
<td>0</td>
<td>54</td>
<td>48</td>
<td>41</td>
<td>57</td>
<td>47</td>
<td>23.7 (0.38)</td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>39</td>
<td>0.5-1.6</td>
<td>0.25-0.8</td>
<td>37</td>
<td>38</td>
<td>36</td>
<td>38</td>
<td>33</td>
<td>19.1 (0.73)</td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>31</td>
<td>0.5-1.6</td>
<td>0.5-1.6</td>
<td>31</td>
<td>31</td>
<td>31</td>
<td>31</td>
<td>29</td>
<td>15.1 (0.58)</td>
<td></td>
</tr>
</tbody>
</table>

[comment by Hemilä: the original table also contained pairwise comparisons with the calculated "t" and corresponding "p" values, but they are not copied in this table, because any pair can be so easily compared with modern statistical programs. The results were presented as percentages of the study group, but they are transformed here to the number of participants with the outcome.]

Analysis of both the literature and our experimental data shows that acute pneumonia (like other acute disorders, especially those connected with microbe invasion) results in a sharp strain of body defense mechanisms and, hence, in the increased intensity of its oxidation-restoration processes. This increased intensity, in turn, increases the organism’s requirement for ascorbic acid. The use of antibiotics only aggravates an already critical situation because the destruction of the pathogenic agent is accompanied by increased intoxication (due to endotoxin) with a further strain of oxidation-restoration reactions, which creates a growing demand for vitamin C. On the other hand, as a result of the weakening of the pathogenic agent functioning, antibiotics create favorable conditions for the mobilization of immuno-biological mechanisms, which again results in a greater demand for vitamin C.

Under such circumstances, additional use of ascorbic acid breaks this vicious circle and enables one to meet the demand in vitamin C for both further intensification of exchange reactions and for unhindered strengthening of the immune system. As a result, body reactivity increases and the period of treatment decreases.
RESULTS

(1) In the process of using antibiotics (penicillin, streptomycin, tetracycline) to treat patients with acute pneumonia both the level of ascorbic acid in blood plasma and its release with urine progressively decrease. The growth of vitamin C deficit is related to the duration of antibiotic use.

(2) The recovery process is much longer in patients treated with antibiotics without ascorbic acid. This may be one of the causes of prolonged treatment and the subsequent onset of acute chronic pneumonia.

(3) To prevent the deficit of vitamin C and its associated complications, the treatment of acute pneumonia with antibiotics should be combined with a simultaneous prescription of ascorbic acid, the diurnal dose of which we suggest to calculate on the basis of the diurnal dose of antibiotic. The minimal dose is, in all probability, 1 mg of ascorbic acid per 2000 units of antibiotic, the maximal dose, 1 mg per 1000 units of antibiotic.

(4) The choice of minimal or optimal dose of ascorbic acid, when administered jointly with antibiotics, obviously depend on the vitamin C resources of an organism in every concrete case. These resources vary with the season, nutrition, and the presence of natural vitamins in the diet.

LITERATURE

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