Infantile Scurvy: A Historical Perspective

Kumaravel Rajakumar, MD

ABSTRACT. Scurvy, a disease of dietary deficiency of vitamin C, is uncommon today. Among diseases, scurvy has a rich history and an ancient past. The Renaissance (14th to 16th centuries) witnessed several epidemics of scurvy among sea voyagers. In 1474, James Lind, a British Naval surgeon, performed a carefully designed clinical trial and concluded that oranges and lemons had the most antiscorbutic effect. Eventually, with the provision of lemon juice to the sea voyagers, scurvy became rare at sea. Infantile scurvy appeared almost as a new disease toward the end of the 19th century. The increased incidence of infantile scurvy during that period was attributed to the usage of heated milk and proprietary foods. Thomas Barlow described the classic clinical and pathologic features of infantile scurvy in 1883. Between 1907 and 1912, Holst and Frolich induced and cured scurvy in guinea pigs by dietary modification. In 1914, Alfred Hess established that pasteurization reduced the antiscorbutic value of milk and recommended supplementation of fresh fruit and vegetable juices to prevent scurvy. Such pioneering efforts led to the eradication of infantile scurvy in the United States. A brief history of infantile scurvy is provided. Pediatrics 2001;108(4). URL: http://www.pediatrics.org/cgi/content/full/108/4/e76; scurvy, infantile scurvy, history, nutrition.

Scurvy, as a disease entity, has an impressionable and indelible history in the annals of medicine. The review of recorded history suggests scurvy to be an ancient disease. Medical writings from antiquity to the Middle Ages describe the classic signs and symptoms of scurvy. Critical appraisal of such literature raises the question of whether other diseases were mistaken for scurvy.

The Renaissance witnessed an unprecedented sense of adventurism and exploration by sea. The early sea voyages were often prolonged and lacked adequate supplies of fresh fruits, vegetables, or animal foods. Such dietary deprivations led to explosive outbreaks of scurvy among the sailors and to the eventual failure of many expeditions.

EARLY HISTORY OF SCURVY

One of the earliest outbreaks of scurvy at sea was sustained by the crew of Vasco da Gama during his 1497 expedition to India. Da Gama began his expedition from Lisbon on July 9, 1497, with a fleet of 4 ships and a crew of 140 men. It took them 6 months to round the Cape of Good Hope. By the time da Gama’s crew landed on the southeast coast of Africa, most of them were afflicted with scurvy. Da Gama recorded: “Many of our men fell ill here, their feet and hands swelling, and their gums growing over their teeth so that they could not eat.” As they sailed farther up the east coast of Africa, they met local traders, who traded them fresh oranges. Within 6 days of eating the oranges, da Gama’s crew recovered fully and he noted, “It pleased God in his mercy that . . . all our sick recovered their health for the air of the place is very good.”

From India, da Gama returned across the Arabian Sea. Within 12 weeks of sailing, his crew was again afflicted and weakened by scurvy. Da Gama commented: “We addressed vows and petitions to the Saints . . . it pleased God in his mercy to send us a wind which in the course of six days, carried us within sight of land . . . at this we rejoiced as . . . we hoped to recover our health there as we had done before . . . the Captain-Major sent a man on shore to bring off a supply of oranges which were much desired by our sick.” Da Gama lost more than half of his crew by the end of his journey. His crew sustained scurvy when they had been at sea for 10 weeks or more. They recognized oranges to be an effective antiscorbutic by the second outbreak. The experience of da Gama in dealing with scurvy did not become common knowledge, and over the next several centuries, scurvy remained as the scourge of the sea explorers.

James Lind’s experiment to test the potency of various antiscorbutic remedies is an important milestone in the history of scurvy. Lind performed his famous experiment in 1747 while a naval surgeon aboard the British naval ship Salisbury. He chose 12 patients with severe scurvy and housed them in a sick bay. The basic diet was similar for all the patients. The 12 men were divided into 6 groups and each group was given a different remedy. The antiscorbutic remedies tested were cider, elixir of vitriol, vinegar, seawater, 2 oranges and 1 lemon for 6 days, and a medicinal paste made of nutmeg and a variety of other ingredients. The patients receiving the oranges and lemons made a remarkable recovery and were well in 6 days and were appointed to nurse the rest of the sick. Lind was able to conclude that citrus fruits had the most effective antiscorbutic potency. It took another 50 years before the British navy made the provision of lemon juice routine onboard. Such a measure resulted in a sharp decline of scurvy among the British naval sailors.

As scurvy was becoming rare at sea during the
INFANTILE SCURVY: A BRIEF HISTORY

Francis Glisson, a Cambridge professor, made one of the earliest recorded descriptions of infantile scurvy. In his famous treatise on rickets, published in 1650, he referred to scurvy occurring conjointly with rickets: “Scurvy is sometimes conjoined with this Affect (rickets). It is either hereditary, or perhaps in so tender a Constitution contracted by infection, or lastingly, is produced from the indiscreet and erroneous Regimen (diet) of the Infant, and chiefly from the Inclemency of the Ayr (air) and Climate where the Child is educated.”7

During the next 200 years, the literature is devoid of references to the occurrence of infantile scurvy. The lack of reporting of infantile scurvy after the Glisson era probably reflects a true diminished occurrence of scurvy. The advances in agriculture with the widespread adoption of potato as a staple and as a weaning food reflected bone pathology.10 By postmortem studies, Barlow established that subperiosteal hemorrhage was the anatomic basis for limb affection in infantile scurvy. Barlow implicated the diet as an etiologic factor. His antiscorbutic regimen included raw-meat juice, fresh milk, orange juice, and access to as much fresh air as possible. He ventured to state that proprietary “infant foods” could not be trusted as “sole aliment for any lengthened period, however useful they may be as adjuncts.”10 Soon infantile scurvy became a recognizable entity and was referred to as “Barlow’s disease.” By the end of the 19th century, infantile scurvy was readily recognized and was being frequently observed in the United States and Great Britain.

INFANTILE SCURVY IN THE UNITED STATES

In 1889, Dr Northrup, of New York, had seen a case of infantile scurvy. His failure to diagnose the condition resulted in the death of the infant. He realized the true nature of the infant’s illness on postmortem examination. That was the first case of infantile scurvy recorded in the medical literature of the United States.14 By 1894, Northrup had collected 114 cases of infantile scurvy and reported them at a meeting of the New York Academy of Medicine. He had observed that “use of proprietary foods and condensed milk produces more scurvy than all other causes combined.”14

L. Emmett Holt’s descriptions of scurvy in his 1897 textbook illustrate the advances in understanding of the clinical presentations of infantile scurvy.15 Holt
classified infantile scurvy into 3 clinical types: a fatal severe form, a severe form culminating in full recovery on identification and treatment, and a mild form without gum involvement or limb swellings. According to Holt, the mild version of infantile scurvy was perhaps the most common, and was often not recognized because of lack of classic gum involvement. Holt, in his discussions on the causes of infantile scurvy, refers to the use of heated infant formulas and concluded that it was the faulty formula and not the process of heating, which caused infantile scurvy. Holt concluded, “proprietary infant-foods are most certain to produce scurvy, when they form the exclusive diet.”\(^{15}\)

As infantile scurvy became more prevalent, a collective investigation of the problem was conducted by the American Pediatric Society in 1898.\(^{16}\) A total of 379 cases were collected and analyzed. Most of the patients in this series were white and were between the ages of 7 and 14 months. Most patients were from a well-to-do background. Dietary history was available for 356 cases. Proprietary foods were part of the diet of 214 patients. Sterilized, pasteurized, or condensed milk had been given to 165 cases.

The members of the research committee concluded that most patients had received proprietary foods and that prolonged usage of unsuitable foods led to the development of scurvy. Tentative as these conclusions were, 1 of the committee members disagreed and a minority report was added. In the minority report “chronic plomaine poisoning” attributable to absorption of toxins was considered to cause scurvy, and sterilizing, pasteurizing, or cooking of the milk were not implicated in the causation of scurvy. These conclusions reflect the confusion regarding the causation of scurvy in the minds of the American pediatricians of that era.

**CAUSE OF INFANTILE SCURVY**

Without doubt, the explosive increase of infantile scurvy during the latter part of 19th century coincided with the advent of usage of heated milks and proprietary foods.\(^{17}\) Bacterial contamination of raw milk was responsible for significant mortality and morbidity among infants during that time.\(^{2}\) Usage of heated milk led to noticeable decline in infant mortality. The advent of heated milks was heralded as a great advance in infant feeding. Unfortunately, the process of heating the milk led to loss of vitamin C. Exclusive usage of heated milks with no other antiscorbutic supplementation led to the development of scurvy. Proprietary foods were touted as being comparable to breast milk in infant feeding. Being farinaceous and of poor nutritional quality, they were a poor substitute for breast milk. They were extensively adopted by the well-to-do. Such indiscriminate use of wealth led to increased incidence of scurvy among infants from higher socioeconomic strata.

**CONCLUSION**

The eventual solving of the malady of infantile scurvy can be tied to the monumental work of Holst and Frolich.\(^{18,19}\) Between 1907 and 1912, they induced and cured scurvy in guinea pigs by dietary modification. Guinea pigs exposed to a diet of cereal grains developed scurvy. Addition of fruits, fresh vegetables, or their juices to the grains prevented the occurrence of scurvy in guinea pigs. Their study was instrumental for several advances in the understanding of etiology and treatment of scurvy.\(^{20}\)

In 1914, Alfred Hess, a pediatrician at the Hebrew Asylum in New York, had noted several cases of scurvy among infants fed on pasteurized milk.\(^{21}\) The increased incidence of infantile scurvy at the Hebrew Asylum had coincided with the elimination of orange juice from the diet. The New York Milk Commission was of the opinion that pasteurized milk heated to 145°F retained its chemical constituents. On the basis of such an assumption, orange juice was excluded from the infants’ diet. Hess was able to effect a cure for scurvy by providing raw milk or orange juice or potatoes. His experiments clearly established that pasteurization resulted in the loss of antiscorbutic potency of milk. Hess recommended that infants receiving heated formulas be supplemented with fresh fruit or vegetable juices to prevent scurvy. Such advances in the understanding of scurvy led to the eventual eradication of infantile scurvy.

**REFERENCES**

10. Barlow T. On cases described as ‘acute rickets’ which are probably a combination of scurvy and rickets, the scurvy being an essential, and rickets a variable, element. *Med Clin Trans (London)*. 1883;66:129–220
**Infantile Scurvy: A Historical Perspective**

Kumaravel Rajakumar  
*Pediatrics* 2001;108;e76  
DOI: 10.1542/peds.108.4.e76

<table>
<thead>
<tr>
<th>Updated Information &amp; Services</th>
<th>including high resolution figures, can be found at: /content/108/4/e76.full.html</th>
</tr>
</thead>
<tbody>
<tr>
<td>References</td>
<td>This article cites 12 articles, 2 of which can be accessed free at: /content/108/4/e76.full.html#ref-list-1</td>
</tr>
</tbody>
</table>
| Subspecialty Collections       | This article, along with others on similar topics, appears in the following collection(s):  
Nutrition /cgi/collection/nutrition_sub |
| Permissions & Licensing        | Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at: /site/misc/Permissions.xhtml |
| Reprints                       | Information about ordering reprints can be found online: /site/misc/reprints.xhtml |

PEDIATRICS is the official journal of the American Academy of Pediatrics. A monthly publication, it has been published continuously since 1948. PEDIATRICS is owned, published, and trademarked by the American Academy of Pediatrics, 141 Northwest Point Boulevard, Elk Grove Village, Illinois, 60007. Copyright © 2001 by the American Academy of Pediatrics. All rights reserved. Print ISSN: 0031-4005. Online ISSN: 1098-4275.
Infantile Scurvy: A Historical Perspective
Kumaravel Rajakumar
*Pediatrics* 2001;108:e76
DOI: 10.1542/peds.108.4.e76

The online version of this article, along with updated information and services, is located on the World Wide Web at:
/content/108/4/e76.full.html