Subglottic Edema in Acute Epiglottitis in Children

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Analysis of airway radiographs of 20 children with proven acute epiglottitis revealed that five (25%) had, in addition to supraglottic edema, localized subglottic edema radiographically indistinguishable from that seen in croup. In all five patients the etiologic organism was *Hemophilus influenzae* type B.

Acute epiglottitis is a cause of rapidly progressive laryngeal obstruction that usually occurs in children, most commonly in the preschool years [1–5]. The etiologic organism in most cases is *Hemophilus influenzae* type B [1, 6, 7]. Although the term epiglottitis has become familiar and is widely used to identify this condition, supraglottitis is probably more appropriate because of the usual involvement of all of the supraglottic structures, including epiglottis, aryepiglottic folds, and false vocal cords. However, it is not generally appreciated that extension of inflammatory edema into the subglottic region can occasionally occur in acute epiglottitis. We report subglottic involvement in five patients with epiglottitis due to *H. influenzae*, documented in all cases with radiographs of the airway.

Subjects and Methods

From 1970 to 1977, 44 children were admitted to St. Louis Children's Hospital with acute epiglottitis. All hospital charts and radiographs were reviewed to analyze the clinical data and determine the presence of subglottic edema. In all cases the diagnosis was confirmed by direct visualization of the inflamed, edematous epiglottis. Twenty patients without airway radiographs and four with radiographs in the lateral projection only were excluded from further analysis. Of the remaining 20 children, all had airway radiographs in the anteroposterior and lateral projections at the time of initial presentation and prior to any type of intubation.

The lateral radiographs were analyzed for the well known signs of epiglottitis, including edema of the epiglottis and aryepiglottic folds, and obliteration of the laryngeal ventricle secondary to involvement of the false vocal cords [8–13]. Subglottic edema was evaluated in the anteroposterior projection, since we feel that the lateral projection is not as sensitive an indicator of mild narrowing. Only localized subglottic narrowing was considered abnormal, since it is known that collapse of the extrathoracic trachea can occur during inspiration in cases of obstruction at or immediately above the level of the glottis [14]. No attempt was made to grade the severity of subglottic narrowing, but cases of questionable narrowing were considered normal. Compared with the shouldered appearance of the upper margins of the normal subglottic trachea (fig. 1), subglottic edema was manifested by symmetric fusiform narrowing localized to the area immediately below the true cords.

Results

All patients with airway radiographs had typical features of epiglottitis in the lateral projection (fig. 2). Of the 20 children with adequate anteroposterior and lateral preintubation airway radiographs, five had localized subglottic edema in addition to the usual signs of epiglottitis (fig. 3). The clinical data in these five patients are summarized in table 1. In all five there was bacteriologic confirmation of *H. influenzae* type B infection. Stridor was described in two patients, but in neither was it the dominant clinical feature. Three patients were managed with nasotracheal intubation and two with tracheostomy. All made uneventful recoveries.

Discussion

Acute epiglottitis as a clinical entity distinct from croup, or laryngotracheobronchitis, has been recognized for nearly 40 years [6]. In croup, which is usually of viral etiology, there is localized subglottic edema with no swelling of the supraglottic structures. In acute epiglot-

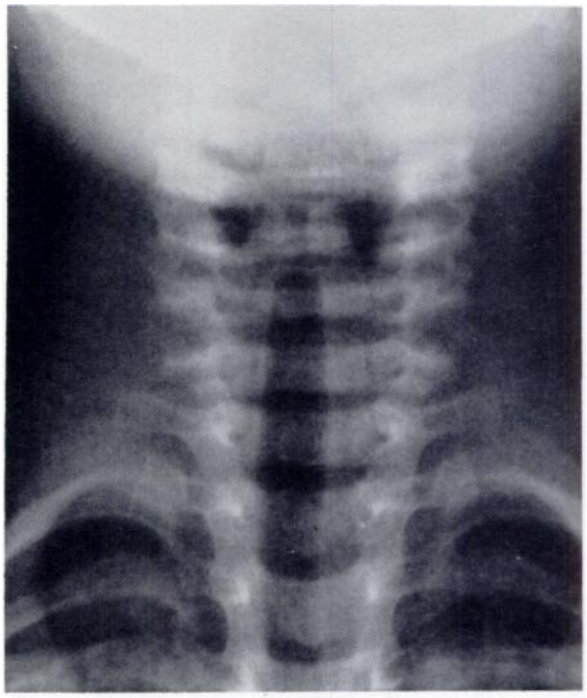

Fig. 1.—Normal anteroposterior airway radiograph in 19-month-old child showing true vocal cords at level of inferior extent of air-filled pyriform sinuses. Subglottic airway has shouldered appearance, with superolateral convexity of upper margins.

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titis there is marked inflammatory edema of the epiglottis and other supraglottic structures. The subglottic airway is usually uninvolved. It has been postulated that the close adherence of the mucosa to the vocal cords prevents involvement of the cords and extension into the subglottic region [4, 5]. However, microscopic extension of the inflammatory process into the laryngotracheobronchial tree has been described in some autopsied patients [7]. There are also scattered clinical reports that mention the occasional presence of subglottic edema in acute epiglottitis [15, 16].

Fig. 2.—Case 5. Lateral airway radiograph showing markedly swollen epiglottis with encroachment on valleculae. Subglottic edema is not as apparent in this projection as in fig. 3*E*.

Our study was performed to determine the frequency of subglottic edema, as determined by airway radiographs. Of the 20 children with radiographs suitable for analysis, five (25%) had subglottic edema, indistinguishable from the type seen in croup. In all five cases the causative organism was *H. influenzae* type B. While the coexistence of croup as a cause for the subglottic edema cannot be excluded, it is very unlikely on the basis of analysis of the case histories (table 1), with the possible exception of case 5. In two of the five children stridor was described, but it was not further characterized.

Except for a single report of subglottic narrowing detectable on a radiograph of a child with acute epiglottitis [15], reports dealing with radiographic findings in this condition have not mentioned the presence of subglottic edema. They have emphasized the lateral projection, and in some institutions this is the only view obtained in cases of possible acute upper airway obstruction. In our study we analyzed subglottic narrowing in the anteroposterior projection, not only because we have found detection of mild narrowing to be unreliable in the lateral projection, but also in order to exclude cases of inspiratory extrathoracic tracheal narrowing due to supraglottic obstruction [14]. It is understandable that subglottic edema would not be readily detectable clinically, due to its obscuration by the striking supraglottic pathology.

The presence of subglottic edema in our patients had no effect on their management or clinical course. We report our findings to document a little known feature of acute epiglottitis and in order to emphasize the importance of the lateral airway projection to differentiate

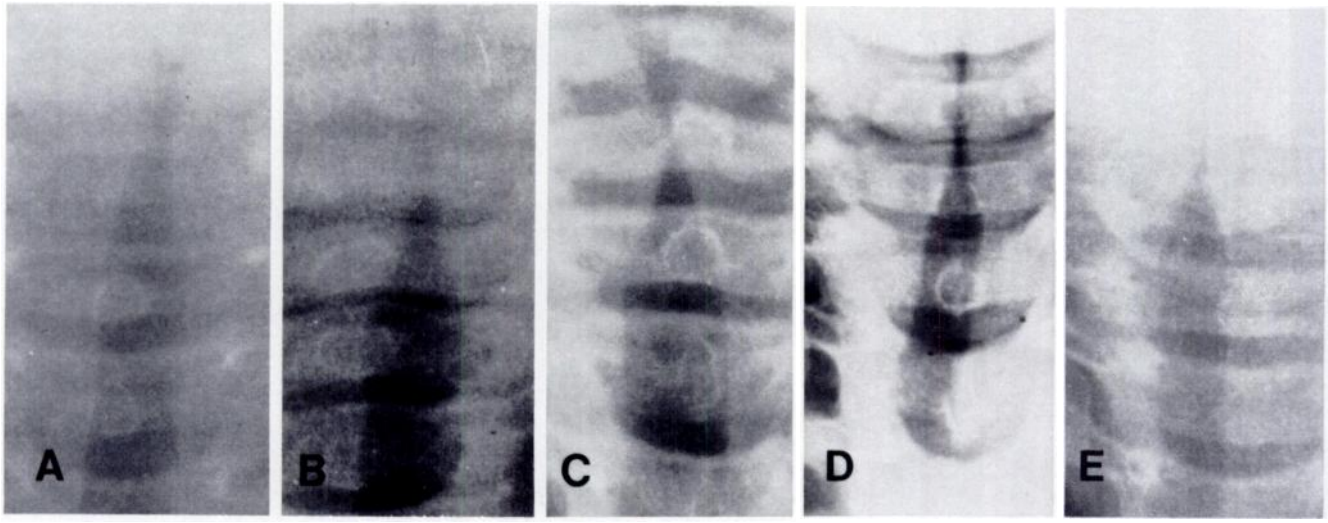

Fig. 3.—Anteroposterior airway radiographs of five patients who exhibited subglottic edema showing in all cases, fusiform narrowing localized to immediate subglottic region, indistinguishable from croup. *A*, Case 1. *B*, Case 2. *C*, Case 3. *D*, Case 4. *E*, Case 5.

TABLE 1
Summary of Clinical Data

Case No.	Sex and Age (years)	Clinical Presentation	Stridor	Bacteriologic Data
1	M, 3	Known seizure disorder; admitted with seizure and 1 day history of fever; respiratory distress developed immediately after admission	No	Blood culture, *H. influenzae* type B
2	M, 1 2/3	Fever for 1 day; acute onset of respiratory distress a few hours before admission	No	Blood culture, no growth; blood countercurrent immunoelectrophoresis, positive for *H. influenzae* type B
3	M, 5	Mild sore throat for 5 days; fever for 1 day; acute onset of difficulty swallowing and worsening sore throat about 12 hr before admission	No	Blood culture, *H. influenzae* type B; tracheal aspirate, normal flora
4	M, 3	Onset of fever and drooling 12 hr before admission; 4 hr history of stridor	Yes	Blood culture, *H. influenzae* type B; epiglottis culture, normal flora
5	M, 3	Sore throat and fever beginning 2 days before admission; stridor for 16 hr	Yes	Blood culture, *H. influenzae* type B; blood countercurrent immunoelectrophoresis, positive for *H. influenzae* type B

epiglottitis from croup. In the anteroposterior projection these two very different conditions may be indistinguishable.

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