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Bacterial tracheitis—A true entity?

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Introduction

ACUTE laryngotracheobronchitis is an infection caused most commonly by influenza and para-influenza viruses (Eichenwald, 1976; Krugman *et al.*, 1977). The majority of patients are children below three years of age. This clinical entity is characterized by gradual onset and a benign course. Local findings are edema and moderate inflammation of the vocal cords and subglottic area. Therapy consists of humidification and general care (Eichenwald, 1976; Krugman *et al.*, 1977). The effect of administration of corticosteroid and racemic epinephrine is undetermined (Leipzig *et al.*, 1979; Westley *et al.*, 1978). We describe two children with *bacterial laryngotracheobronchitis* involving the subglottic area. The disease was characterized by an acute onset, high fever, inspiratory stridor, and the production of copious amounts of purulent sputum requiring intratracheal intubation. These two cases support a previous report describing bacterial tracheitis (Jones *et al.*, 1979) and raise some questions regarding the primary etiology of the disease.

Case No. 1

A $1\frac{1}{2}$-year-old female was hospitalized for inspiratory stridor, cough and shortness of breath which started a few hours prior to admission. Physical examination revealed a sick child with temperature of 39·8°C, respiratory rate of 60 per minute, with suprasternal and intercostal retractions. On auscultation a decrease in air entry to both lungs was noted. Fluoroscopy of the chest revealed paradoxical movements of the mediastinum, so a tracheal foreign body was suspected. On bronchoscopy the supraglottic and epiglottic structures appeared normal while the subglottic area was acutely inflamed secreting copious amount of purulent material. No foreign body was found. Serial arterial blood gases following the procedure showed mild hypoxemia with progressive respiratory acidosis. The child was treated with racemic epinephrine inhalations, intravenous hydrocortisone and ampicillin, and supplemental oxygen through a mist tent. As no improvement occurred the child was intubated and ventilated. Cultures of tracheal secretions obtained during bronchoscopy and intubation grew indole-negative proteus species and para-influenza virus type 1. Blood culture was sterile. The hemoglobin was 12·0 gr%, W.B.C. 15,000/mm^3 with a shift to the left. Chest X-ray was normal.

As soon as culture and sensitivity results became known, kanamycin was added. Gradual improvement was noted over the next few days and extubation was performed after three days. The child recovered with no sequela.

Case No. 2

A $4\frac{1}{2}$-year-old child, previously healthy, was hospitalized because of inspiratory stridor, barking cough and progressive dyspnea which started 24 hours prior to admission. Physical examination revealed an agitated child with a

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temperature of 38·8°C, peri-oral cyanosis, tachypnea of 64/min., with intercostal retractions. The lungs were clear on auscultation. Chest X-ray disclosed prominent peri-hilar markings. The hemoglobin was 11·0 gr%, W.B.C. 19,000/mm^3 with a shift to the left. Urgent bronchoscopy performed for a suspected tracheal foreign body revealed a normal epiglottis but with prominent subglottic oedematous mucous membrane secreting purulent discharge. An endotracheal tube was inserted because of respiratory deterioration (pH 7·2, pCO_2 60 mm Hg, pO_2 55 mm Hg, $FIO_2$0·4%). Cultures of deep tracheal secretions as well as blood and throat cultures yielded non-groupable hemolytic streptococcus.

Therapy included intravenous penicillin and hydrocortisone, and inhalations with racemic epinephrine. During the next days frequent suction removed copious amounts of purulent secretion from the trachea. Repeated chest X-rays remained normal throughout the hospital course. The child improved gradually and was discharged on the seventh hospital day. Viral cultures for para-influenza, influenza and adeno viruses were reported as negative.

Comment

Croup is usually caused either by acute bacterial epiglottitis or viral laryngotracheobronchitis or acute spasmodic laryngitis of undetermined etiology (Eichenwald, 1976; Krugman *et al.,* 1977). The two cases described herein, as well as the eight cases reported previously (Jones *et al.,* 1979), raise the possibility that acute laryngotracheobronchitis may be caused by bacteria. The clinical presentation of the present two cases and those previously described was characterized by a rapid onset of laryngeal obstruction, high fever, leukocytosis and prostration. These findings suggest acute epiglottitis. However, the young age of most of the children (eight out of the ten children were under three years of age) and the findings of a normal epiglottis but inflamed subglottic area suggested a different entity. Furthermore, the copious amount of pus secreted by the subglottic mucosa and trachea seems to characterize this disease. Secretions of this nature are seen neither in acute epiglottitis nor in viral laryngotracheobronchitis, except in rare cases (Howard *et al.,* 1972). In the present two cases pneumonia was ruled out by the absence of typical physical findings and repeated normal chest X-rays. The etiology of the disease is not readily apparent. In one of our cases a gram-negative bacillus was isolated (by a sterile technique) and in the other a hemolytic streptococcus was isolated from cultures of blood, throat and tracheal secretions. In six previously reported cases, *Staphylococcus aureus* was isolated from the secretions, and *Haemophilus influenza* and *Streptococcus pyogenes* in one case each, suggesting that upper respiratory tract flora are responsible for most cases.

In one of our cases para-influenza virus type 1 was isolated and in the other no virus was cultured. In the eight previously reported cases no attempt was performed to isolate viruses.

The true etiology of bacterial tracheitis is still unclear. Bacterial superinfections are rare in acute viral laryngotracheobronchitis and, in fact, most authors do not recommend antibacterial therapy in this setting (Davis and Wedgwood, 1965; Tercero-Talavere and Rapkin, 1974). However, the concept that viruses may enhance the susceptibility of the respiratory tract to bacterial invasion has long been accepted (Douglas, 1976; Glasgow, 1972). It is recognized

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that bacterial lung infections occur as a complication of respiratory viral diseases (Foy *et al.*, 1979). Viruses can alter the normal upper respiratory flora and in addition compromise the host capacity to clear bacteria, decrease phagocytosis of alveolar macrophage and suppress immune response (Glasgow, 1972).

Our two cases support Jones *et al.'s* (1979) theory that bacterial tracheitis exists. Our one case with concomitant isolation of virus and bacterial pathogens may suggest that the bacteria were superinfecting a viral croup. Since in previous reports no viral cultures were obtained it is unknown whether these cases were also a complication of viral disease or an initial bacterial infection. Further studies designed to obtain viral and bacterial cultures, as well as serological tests, should clarify this question. Regardless of whether the initiating cause is viral or bacterial, patients with laryngotracheobronchitis should have cultures performed from the blood and tracheal aspirate if the disease takes an unexpectedly severe course, simulating epiglottitis and necessitating intubation. Antibiotic therapy should be started and aimed at organisms demonstrable in the gram stain performed from a tracheal aspirate specimen. Initial therapy should probably be aimed against *Staphylococcus aureus* and other upper respiratory tract flora.

REFERENCES

DAVIS, S. D. and WEDGWOOD, R. J. (1965) *American Journal of Diseases of Children,* **109,** 544.
DOUGLAS, R. G. (1976) *Hospital Practice,* **11,** 43.
EICHENWALD, H. F. (1976) *Hospital Practice,* **11,** 81.
FOY, H. M., CONNEY, M. K., ALLAN, I. and KENNY, G. E. (1979) *Journal of the American Medical Association,* **241,** 253.
GLASGOW, L. (1972) *The New England Journal of Medicine,* **287,** 42.
HOWARD, J. B., MCCRACKEN, G. H. and LUBY, J. P. (1972) *The Journal of Pediatrics,* **81,** 1148.
JONES, R., SANTOS, J. I. and OVERALL, J. C. (1979) *Journal of the American Medical Association,* **242,** 721.
KRUGMAN, S., WARD, R. and KATZ, S. L. (1977) C. V. Mosby Company, Sixth Edition, 248.
LEIPZIG, B., OSKI, F. A., CUMMINGS, C. W., STOCKMAN, J. A and SWENDER, P. (1979) *The Journal of Pediatrics,* **94,** 194.
TERCERO-TALAVERE, F. I. and RAPKIN, R. H. (1974) *Clinical Pediatrics,* **13,** 1074.
WESTLEY, C. R., COTTON, E. K. and BROOKS, J. G. (1978) *American Journal of Diseases of Children,* **132,** 484.

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