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Acute Epiglottitis as Cause of Sudden Death

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Acute fulminating epiglottitis is usually a disease of children (BERENBERG and KEVY, FÄLLSTRÖM and LINDBERG, VETTO, LUDVIGSEN and VAINER, POOLE and ALTMAN, MATTESON, VON ARNDT and HANSEN, SEIGE and ALBRECHT) but may also occur, though less often (MATTESON), in adults (KJAERHEIM and URSIN-HOLM, POULSEN and SIMONSEN, LEMIERE et al., LUDVIGSEN and VAINER, KYTTÄ, MATTESON). The disease is usually characterized by mild precursory symptoms followed by a strong inflammatory swelling of the epiglottis and adjacent tissue with subsequent respiratory distress, sometimes leading to death from suffocation. Forensic pathologists (JONES and CAMPS, KJAERHEIM and URSIN-HOLM, POULSEN and SIMONSEN) have drawn attention to acute epiglottitis as a cause of sudden death, often before arrival of the doctor or during transport of the patient to hospital.

Within as short a period as 2 months we have seen fulminating fatal epiglottitis in 2 adults and in 2 children. These cases illustrate the characteristic clinical course of the disease and the morbid anatomy and are described below.

Case 1. A girl, aged 3 years and 8 months, who had previously been healthy became ill with a slightly sore throat around noon on September 11, 1965. The child was peevish and refused to eat. At 6 p.m. her body temperature was 39.8° C; her condition was otherwise unchanged. She slept quietly from 7.30 p.m. until 10.30 p.m. when she suddenly had respiratory distress. The ambulance was called for. As the distress progressed so rapidly and since the girl lost consciousness it was decided not to wait for the ambulance but to drive the child to the hospital in a private car. On arrival there at 12.45 p.m. the child was dead.

Case 2. A previously healthy girl, aged 8 years and 11 months, became ill at 11 p.m. on October 3, 1965 with mild dysphagia. The following day the symptoms persisted (unchanged) and the girl cycled to school. On return from school at 3 p.m. she complained of a sore throat and went to bed. Her temperature was not recorded. At 10 p.m. respiratory distress suddenly occurred and the child lost consciousness. On arrival at hospital at 10.50 p.m. the child was dead.

Case 3. A previously healthy woman, aged 46, became ill on October 19, 1965 with hoarseness and general malaise and went to bed. The next morning her condition was unchanged. At 8.40 a.m. that day her husband telephoned, but owing to her hoarseness she could not answer. At 10 a.m. she was found lying dead on the floor.

Case 4. A previously healthy man, aged 54, went to work at 6 a.m. on November 12, 1965. One hour later he complained of a sore throat. The pain increased rapidly and he sought medical advice without delay. He arrived at the doctor's surgery just before 9 a.m. He then found it difficult to swallow and talk. A few minutes later respiratory distress occurred and at 9.10 a.m. he was sent to hospital in a taxi. Twenty minutes later he died in the taxi, just outside the hospital.

Table

Case no.	Sex	Age	Interval (hours) between onset of symptoms and death	Symptoms	Bacteriological examination
1	K	3 years 8 months	12	Sorenses of the throat Temperature 39.8° C Respiratory distress	Haemophilus influenzae
2	K	8 years 11 months	$23^1/_2$	Difficulties in swallowing Respiratory distress	Haemophilus influenzae
3	K	46 years	26	Hoarseness "Common cold"	Haemophilus influenzae (+ pneumococci and β-streptococci)
4	M	54 years	$2^1/_2$	Soreness of the throat Difficulties in swallowing Respiratory distress	Negative

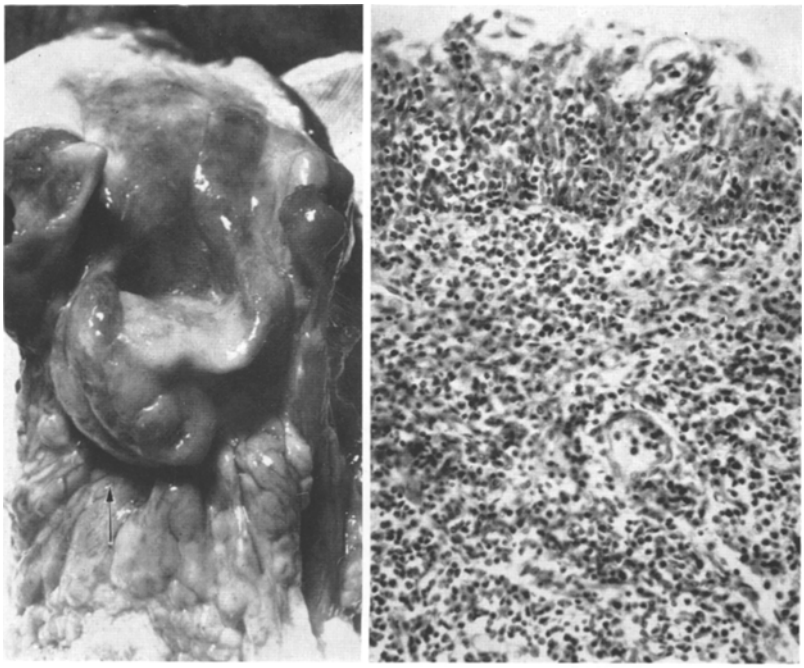
Autopsy

Gross Findings. — Autopsy was done within 5 hours after death in one case (No 4), and after 20—36 hours in the others.

The autopsy findings were largely the same in all 4 cases. The changes were mostly confined to the larynx and were characterized by a marked swelling and reddening of the epiglottis, particularly of its free edges and the lingual surface, with consequent narrowing of the airway. The swelling also involved the rest of the larynx, though less severely. In one case (No 2) the laryngeal mucosa showed small superficial ulcerations and was coated with an uneven layer of mucus. In another case (No 4) the swelling had caused polypous excrescenses of the epiglottis and adjacent parts of the larynx (Fig. 1 A). The mucosa of the trachea and the bronchus was slightly reddened in case 3 but pale in the others. The lungs were congested and oedematous but showed no focal changes. The internal organs showed signs of acute congestion. There was acute splenitis to a varying degree in all 4 cases. There were no morphological signs of generalised infection or septicaemia. A few petechiae were seen in the conjunctivae and beneath the pleurae in case 1. Otherwise no

petechial haemorrhages were seen in the skin of the face, in the conjunctiva, in the oral or nasal mucosa, in the thymus or beneath the serous membranes of the heart or lungs.

Microscopical Findings. — In all 4 cases the epiglottis showed severe oedema with numerous neutrophilic leukocytes (Fig. 1 B). The inflammation involved both the mucosa and the submucosa as well as the underlying muscle layers. The picture



A B

Fig. 1 A. Case 4. Marked swelling and reddening of the epiglottis and adjacent larynx with a polypous excrescense (arrow)

Fig. 1 B. Intense inflammatory cell reaction involving mucosa (*up*) and underlying tissue with numerous polynuclear leukocytes

was dominated partly by oedema and partly by an inflammatory cell reaction. In case 2 the surface epithelium of the epiglottis showed ulceration; in the other 3 cases the epithelium was intact. The mucosa of the trachea and the bronchi showed signs of mild inflammation in one case (No 3) but in none of the remaining 3. No inflammatory reactions were seen in the lungs in any of the 4 cases. The microscopical examination otherwise verified the gross findings.

Bacteriological examination

Culture of tracheal secretion obtained *post mortem* showed an abundant growth of *Haemophilus influenzae* in cases 1, 2 and 3. In addition culture of tracheal secretion in case 3 gave scanty growth of betahaemolytic streptococci belonging to

Lancefield's group A and culture of lung tissue in that case besides *Haemophilus influenzae* abundant growth of pneumococci.

In case 4, culture of secretion from the epiglottis and from the trachea gave no growth of *Haemophilus influenzae*; normal commensals were the only finding. Fourteen day cultures of foetal kidney cells at 33° C and at 37° C including haemadsorption at 37° C for virus, proved negative, as did culture of amniotic cells at 37° C. In case 4 culture of blood, of splenic tissue and subarachnoidal secretion gave no growth of bacteria.

In cases 2 and 3 culture of direct smears of arterial blood on haematin agar plate gave abundant confluent growth of *Haemophilus influenzae*. Culture of the blood was not performed in case 1. In cases 2 and 3 culture of lung tissue, splenic tissue and CSF gave abundant growth of *Haemophilus influenzae*.

The isolated strains of *Haemophilus influenzae* from cases 2 and 3 were studied serologically and found to belong to the capsular type b, *i.e.* the type commonly found in patients infected with *Haemophilus influenzae*. The strains kept their capsule for 6 months, during often repeated subculture. Culture filtrates of these strains were found to contain two precipitating soluble-antigens (tested with diffusion-in-gel technics). One consisted of capsular substance, the other of a undefined antigen. The culture filtrate, from which substrate material had been removed by filtration on Sephadex G-200, proved toxic for rabbits and guinea pigs on intravenous respectively intraperitoneal injection. The rabbits died 4—10 hours after the injection and began to show signs of distress about 1 hour after the injection. They remained still and became atonic. Respiration became difficult with a terminal maximum use of the accessory muscles of respiration. No convulsions occurred. Autopsy showed dilatation of the abdominal and heart vessels and in some cases a few small punctate haemorrhages under the serous mucosa of the heart and lungs. Histological examination revealed congestion of the internal organs, especially of the lungs and spleen, sometimes with haemorrhage. The animals, which had received only a sublethal dose, were ill for the first day, after which they appeared healthy.

Discussion

In the 4 human cases described the acute epiglottitis was fulminant. Death occurred within 26 hours of the onset of symptoms; in one (case 4) within $2^1/_2$ hours. The course was insidious: a relatively short period of soreness of the throat was followed first by dysphagia in association with hoarseness (case 3, probably mixed infection) and fever (case 1) and then by sudden respiratory distress resulting in death within an hour or so.

The cases occurred between September 11 and November 12, 1965. During the autumn and winter acute infections of the respiratory tract are prevalent. Severe cases of infection with *Hemophilus influenzae* have often occurred during influenza epidemics (Alexander, Cooke). Regarding our cases no epidemic of influenzae occurred during the autumn of 1965 in the area in question; Coxackie virus B_3 and B_5, however, were viruses most commonly found in cases of acute virus infections in October 1965.

The pathological changes found at post morten in the respiratory tract, were of characteristic spread and appearance, *i.e.* marked swelling

of the mucosa of the epiglottis, particularly its free edges and the lingual surface. The findings at autopsy were otherwise negative and not characteristic. The internal organs were acutely congested. Such congestion was also found in the experimental animals given intravenous or intraperitoneal injections of bacteria-free culture filtrate from the *Haemophilus influenzae* strains isolated from 2 of the cases.

In 3 of our cases in which the patients survived for more than a few hours after the onset of symptoms there was a varying degree of acute splenitis but no evidence of a generalized infection. In 2 of our 3 cases culture of various organs, body fluids and secretions gave abundant growth of *Haemophilus influenzae*. No histological signs of septicaemia were seen in these cases. There thus appeared to be a discrepancy between the general spread of the bacteria in the body and the occurrence of secondary infectious foci.

Haemophilus influenzae is known to be the commonest cause of acute epiglottitis. Epiglottitis has even been described as a pathognomonic sign of infection with *Haemophilus influenzae* (VETTO, ALEXANDER) probably in association with bacteriemiae (ALEXANDER). In case 4, where no pathogenic bacteria were found on culture, culture for virus was also negative.

The possibility of viral infection being a cause of acute epiglottitis has been discussed (VON ARNDT and HANSEN, SEIGE and ALBRECHT). A negative culture of material, removed 5 hours post mortem, for virus is much less informative than a corresponding negative culture for bacteria. If an appropriate culture for *Haemophilus influenzae* is negative, this bacterium is probably of no aetiological significance. Culture of samples for virus by available methods deos not exclude infection with the virus in question.

The obstruction of the airways demonstrated at post mortem together with historical data, suggest that death in our 4 cases was due to suffocation. Apart from the laryngeal changes, the morphological evidence of suffocation was slight. Petechial haemorrhages in the conjunctiva, in the skin of the face, in the oral and nasal mucosa, in the thymus, beneath the serous membranes of the heart and of the lungs, expected in cases of death due to suffocation, were not seen in 3 cases and were only scanty in one (case 1). A toxic effect of the bacteria with heart failure as a consequence may have contributed to the fatal issue in the 3 cases (No 1, 2 and 3) where culture gave growth of *Haemophilus influenzae*. Our animal experiments give some support to this view.

The histological picture of epiglottis was largely the same in all of our cases and was characterized by marked oedema, with abundant deposition of neutrophilic leukocytes. The picture was thus that of a bacterial infection. There was no morphological evidence of an allergic inflammation in the form of eosinophilia or fibrinoid necrosis.

Summary

Acute epiglottitis as a cause of sudden death in 2 adults and 2 children within a period of 2 months is reported. The cases illustrate the characteristic course of the disease and its morbid anatomy. Culture of necropsy specimens gave abundant growth of *Haemophilus influenzae* in 3 cases, including 2 in which blood, CSF, lung and splenic tissue yielded heavy growth of this organism.

Zusammenfassung

Es werden 4 plötzliche Todesfälle (2 Erwachsene, 2 Kinder) als Folge einer akuten Epiglottitis beschrieben, die innerhalb von 2 Monaten zu Sektion kamen. Die Fälle illustrieren den charakteristischen Krankheitsverlauf und die patologisch-anatomischen Befunde. Bei der bakteriologischen Untersuchung wurde bei 3 Fällen Haemophilus influenzae nachgewiesen (Kultur), davon bei 2 in Blut, Liquor, Lunge und Milz.

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