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Acute necrotizing rhinitis in man

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SUMMARY

The clinical picture and the course of the acute necrotizing rhinitis in man is described in its different stages.

Necrotizing rhinitis seems to be a significant factor in the etiology of some disturbances of the nasal respiratory function and of the ciliary activity of the nose, of atrophic rhinitis and of dry pharyngitis, in some cases of intranasal adhesions and of permanent mucosal defects of the septum. To our knowledge these defects have not been reported in the literature in the past, but are apparently not too rare.

The following report on acute necrotizing rhinitis in man is based on clinical observations over several years. Not included are cases of necrosis due to exposure to caustics, pressure from foreign bodies or post-operative complications, as well as malignancies, granulomatous disease or specific infections. Most of the cases occurred during influenza A_2 and B epidemics. In addition, adenoconjunctival virus, and mycoplasma were identified.

The clinical picture was quite characteristic in its course, but varied in intensity from epidemic to epidemic and from individual to individual. The clinical course can be divided into four stages:

1. The initial manifestations.
2. The height of involvement of the nose and involvement of other structures.
3. The healing process.
4. The sequelae.

In the initial stage, there was general malaise and some elevation of the temperature. The nasal mucosa showed swelling and significant increase of the vascular pattern and had a spotty reddish appearance.

In the second stage, subepithelial hemorrhages (Figure 1) were seen, which had a tendency to spread readily and in some instances considerable confluence was found within minutes. This was followed by a breakdown of the mucosa

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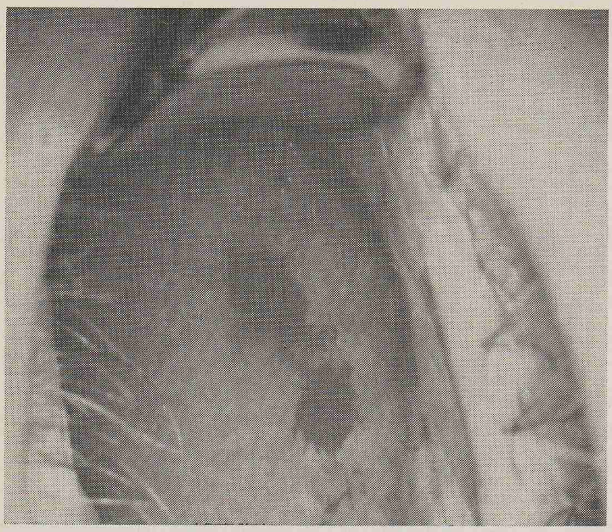

Figure 1. Subepithelial hemorrhage of the inferior turbinate. There is an increase of the vascular pattern producing a spotty appearance.

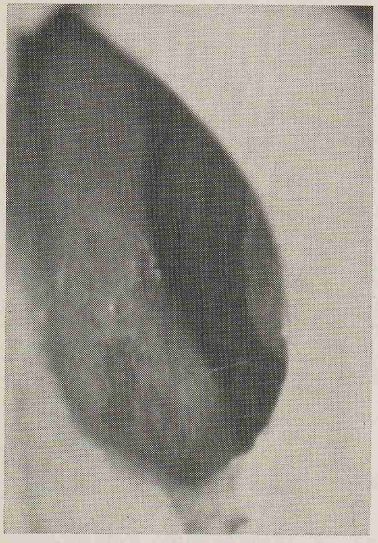

Figure 2. Ulceration and external hemorrhage of the inferior turbinate. Increase of the vascular pattern. Increase of the nasal airspace.

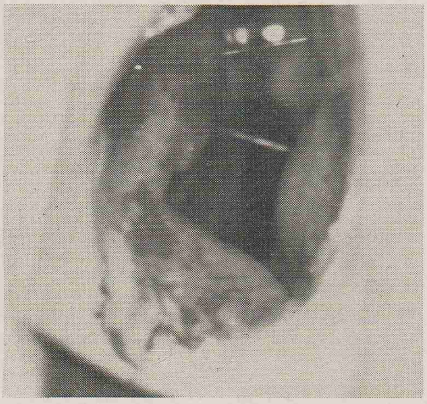

Figure 3. Ulceration and widespread external and subepithelial hemorrhage of the inferior turbinate.

(Figure 2) of greater or lesser magnitude associated with frank bleeding. The breakdown was usually fairly superficial, but occasionally extended in depth. The bleeding was as a rule well circumscribed, but at times involved almost the total mucosal blanket (Figure 3). Even when wide-spread, the bleeding as a

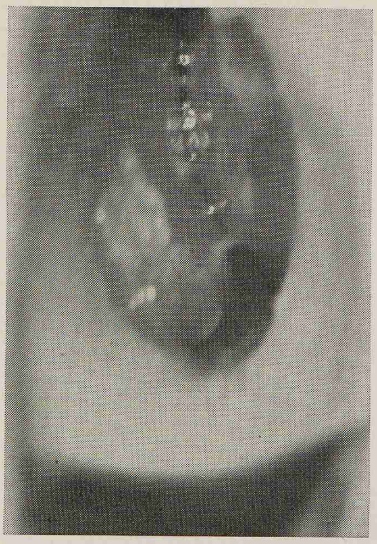

Figure 4. Ulceration with fibrin and crust formation. Irregular appearance of the mucosal surface of the inferior turbinate. Notice the abnormal wideness of the intranasal airspace. Two strands of tenacious mucus between the septum and the turbinate.

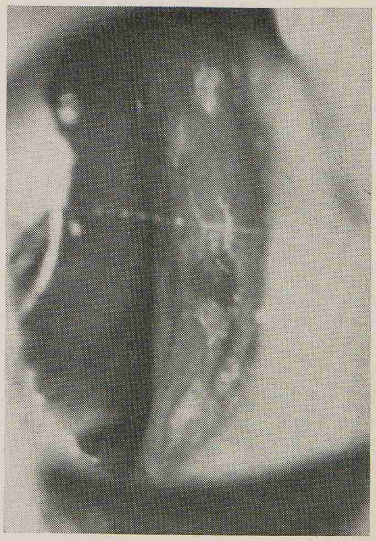

Figure 5. Spotty scarring with deposits of fibrin and crusts. Heavy crusting over the septum. A small ulceration is still present. Abnormal wideness of the intranasal airspace.

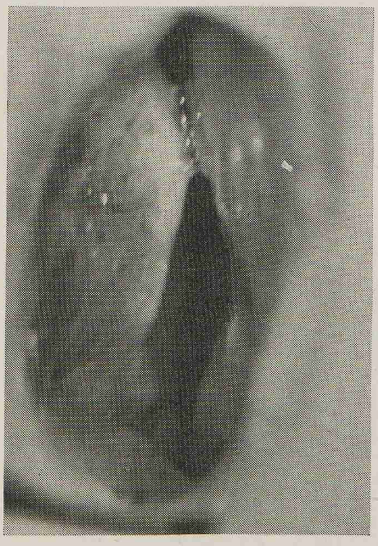

Figure 6. Scarring and crusting of the inferior turbinate and the septum. The mucosa is irregular and has a punched-out appearance. In the superior part of the picture, there is already some clear mucus in a localized area between the septum and the inferior turbinate.

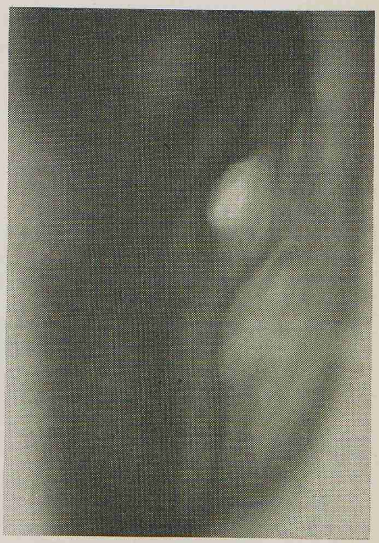

Figure 7. Embossed appearance of the mucosa, as a manifestation of subepithelial and surface scarring.

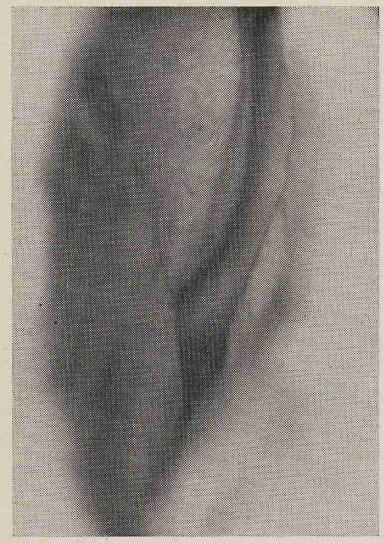

Figure 8. The mucosa has a fairly "normal" appearance. The middle turbinate shows an increased vascular pattern. The inferior turbinate is still somewhat irregular and spotty (12 weeks after onset). "Normal" wideness of the intranasal airspace.

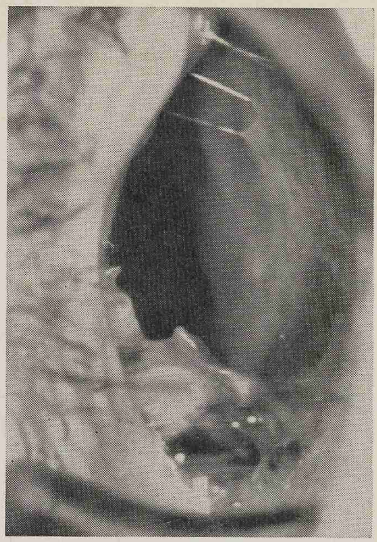

Figure 9. Abnormally wide nasal airspace. Heavy crusting on the floor.

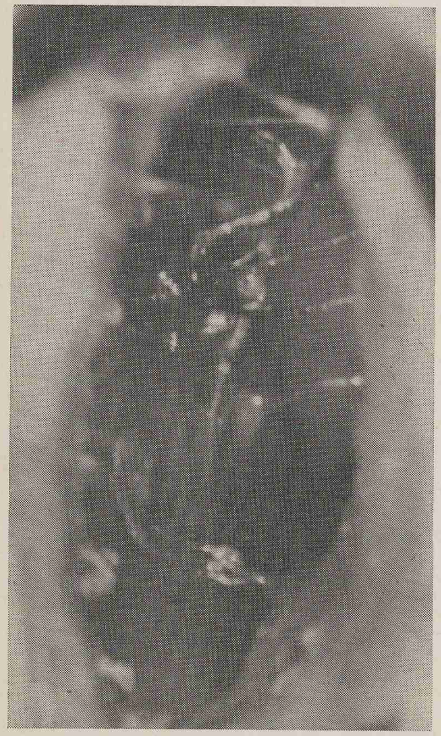

Figure 10. Abnormally wide nasal airspace. Atrophy of the mucosa with crusts and strands of dry secretion and blood between the septum and the turbinates.

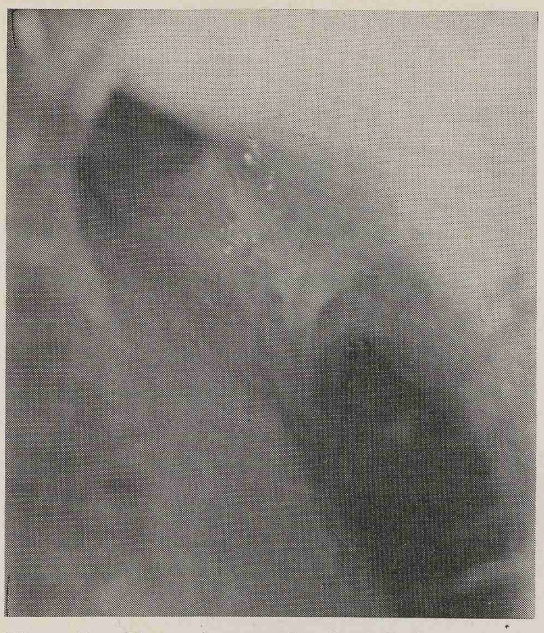

Figure 11. Heavy intranasal adhesion between the septum and the turbinate (region #4).

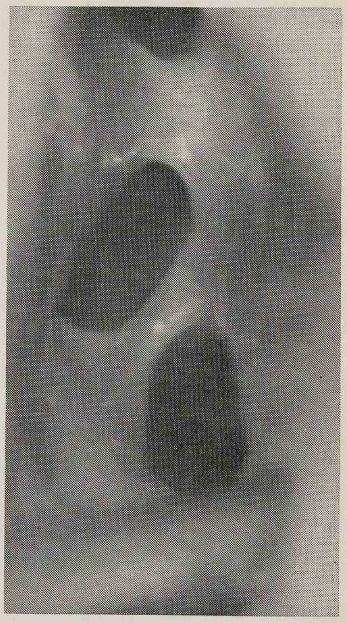

Figure 12. Intranasal adhesions between the septum and the turbinate (region #4).

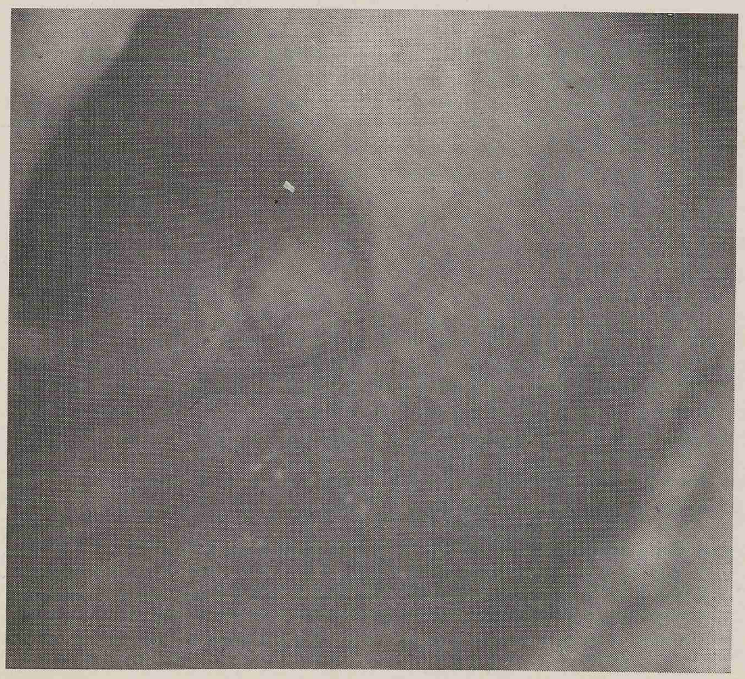

Figure 13. Permanent mucosal defect of the nasal septum. The perichondrium is preserved. Blood vessels on the perichondrium are seen.

rule was not severe. However, in some cases it was substantial. The bleeding had a tendency to occur in spells and to stop spontaneously. It was in this stage that bleeding was also seen into the ears, the soft palate, the pharynx and the larynx.

In the third stage, the healing process, the defects of the mucosa became covered with fibrin and later with crusts (Figure 4 and 5). It was common to see dry and crusty areas next to islands of normal appearing mucosa covered with clear mucus (Figure 6). The ciliary transport was definitely impaired. As the defects healed, differences in color and in the level of the mucosal surface could be seen creating an irregular appearance (Figure 6 and 7).

Some patients showed remarkable recovery and the appearance of the mucosa and its function returned to "normal" (Figure 8). In the majority of the patients, however, healing took place with some deficit. The most common sequela was scarring with varying degrees of disturbance of the ciliary activity. There was an increase of the nasal airspace, as a manifestation of atrophy and subsequently an increase of the dryness of the nose, the pharynx and the larynx, and the resulting disturbance of the nasal function seen in the wide nasal airspace (Figure 9 and 10). One patient developed significant metaplasia in the anterior third of the septum. Granulations were seen only infrequently. They subsided spontaneously. Intranasal adhesions were encountered, but could be easily separated when they were seen in the early stage. Three patients, who stayed away from medical care for a longer period of time, developed heavy adhesions (Figure 11 and 12).

One of the most interesting sequelae of the acute necrotizing rhinitis was the formation of permanent defects of the septal mucosa (Figure 13 and 14). There was loss of the mucosa and submucosa, while the perichondrium was preserved. Examination with the operating microscope revealed fine blood vessels on the perichondrium. The size of the defect varied between 2 and 5 mm. Most defects were oval in shape with regular, slightly thickened borders. In some cases, the mucosa was slightly overhanging producing a shallow pouch (Figures 13 and 14). The defects were located in the anterior third of the septum except in one patient. They were found on one side of the septum in 27 patients, while 4 patients had bilateral defects. One patient had 2 defects on the same side; one in the anterior and one in the middle third of the septum. The ciliary mucus transport over the affected area was absent and occasionally accumulation of clear mucus was found over the defect. The mechanism of the development of these defects is not yet known. No change of the defects was noticed in the long-term follow-up observations, and no perforations of the septum were seen. To our knowledge, these lesions have not previously been described in the literature. (Dr. Roberto Prado Perez of Mexico City informed me at the meeting in Kasas City that he has recently seen a patient with a mucosal defect

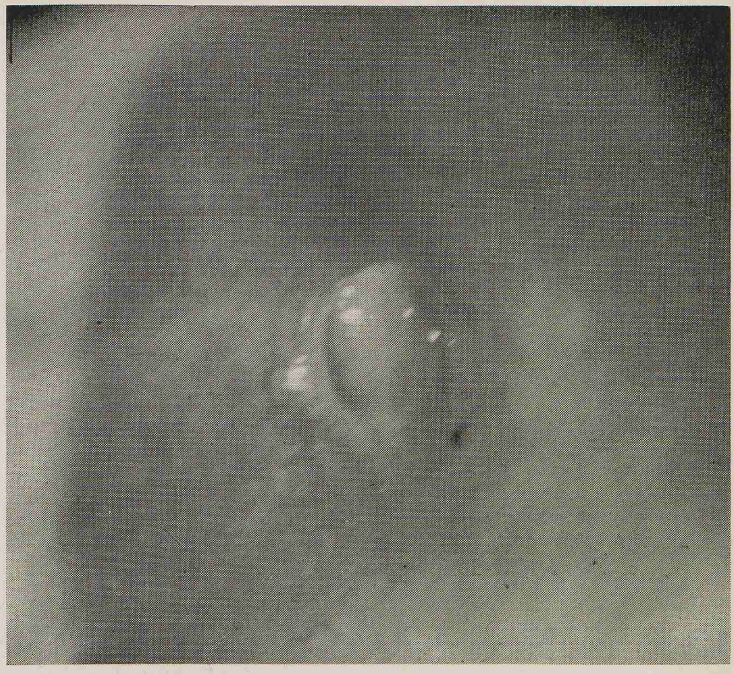

Figure 14. Permanent mucosal defect of the nasal septum, covered with clear mucus. The mucus remained stationary until it was moved by blowing the nose.

of the septum. [Prado Perez, 1977]). Judging from our material, the acute necrotizing rhinitis seems to be a significant factor in the etiology of some disturbances of the nasal respiratory function and of ciliar activity, of atrophic rhinitis, in some cases of intranasal adhesions and in permanent mucosal defects of the septum.

Animal experiments for the study of desquamating rhinitis with laryngotracheitis virus in chicken have been reported by B. G. Bang and F. B. Bang (Bang and Bang, 1963 and 1967). These authors saw functional restoration of mucociliary lining of the nasal fossa after the infection had cleared up. They reported that eight specimens showed complete metaplasia of the olfactory region. The recuperative power in our human material seems to be less than in these laboratory animals.

ZUSAMMENFASSUNG

Das Krankheitsbild und der Verlauf der akuten nekrotisierenden Rhinitis des Menschen ist beschrieben. Die akute nekrotisierende Rhinitis scheint ein bedeutsamer ursächlicher Faktor in Veränderungen der Nasenatmung und der ciliaren Aktivität, in der atrophischen Rhinitis, in einigen Fällen von intranasalen Adhesionen und dauernden Defekten der Septummukosa zu sein. Soweit

wie wir es wissen, diese Defekte sind noch nicht in der Literature beschrieben worden, aber kommen anscheinlich nicht zu selten vor.

SUMARIO

El cuadro clínico y el curso del rinitis de necrosis agudo en el ser humano está describido en sus distintas etapas.

El rinitis de necrosis parece que es un factor significante en la etiologia de algunos desordenes de la función de la respiración nasal y de la actividad ciliar de la nariz, del rinitis atrófico y del faringitis seco, en algunos casos de adherencias intranasales y de defectos permantes de la mucosa del tabique nasal. Al conocimiento nuestro estes defectos no han sido reportados anteriormente en la literatura, pero aparentemente no son muy raros.

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