parable patients with perforated duodenal ulcers were followed up after two to twelve years. Ten patients could not be traced. Eight (11%) of the "gastric" patients, but none of the "duodenal" patients, are known to have died of carcinoma of the stomach. Seven (5%) of all the patients died of peptic ulcer.

The late prognosis among the known survivors was unaffected by the sex or age of the patients or the site of the ulcer. It was affected by the length of history prior to the perforation and by whether or not the ulcer had been previously diagnosed; in the latter group 50% suffered a further gastric operation.

The advantages and disadvantages of the three current methods of treatment are discussed, and it is concluded that no one method should be the method of choice in all cases. In any one series all three methods have their proper place. Tentative conclusions about the indications for choice of method in individual cases are suggested.

We are most grateful to our colleagues at the Central Middlesex Hospital for permission to refer to cases treated under their care, and to Mr. J. D. Fergusson, Mr. F. A. Henley, and Mr. T. G. I. James for their advice in the preparation of this paper.

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GRANULOMA OF THE NOSE AND PERIARTERITIS NODOSA

BY

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[WITH SPECIAL PLATE]

From case reports published during the past twenty-one years there gradually emerges the picture of a peculiar syndrome consisting of a chronic ulcerating granulomatous inflammation of the nose, nasal sinuses, and often pharynx, larynx, and trachea, which is followed after a time by generalized periarteritis nodosa, and which leads almost invariably to a fatal issue.

The dominant lesion at the outset of the disease causes a purulent discharge from the nose, which is usually also obstructed. The nasal mucosa presents a characteristic picture of ulcers with yellow crusts which bleed on removal, and necrosis of the bone may appear later. These symptoms are usually accompanied by low-grade fever, and within a few weeks the patient is obviously becoming seriously ill, with albuminuria, signs of renal and cardiac failure, and a rising blood

urea. The terminal illness is usually short, death occurring about six months after the onset of the nasal lesions. Necropsy shows characteristic lesions of periarteritis nodosa in many organs.

This syndrome was first described in 1931 by Klinger (1 case), and since then by Wegener (1939), 3 cases; Lindsay, Aggeler, and Lucia (1944), 1 case; Weinberg (1946), 1 case; and Howells and Friedmann (1950), 1 case. Four of these patients were males and three females; their ages ranged from 23 to 70, and the total duration of illness from six to thirteen months. In all of them the post-mortem findings confirmed the diagnosis.

We record here two cases of this syndrome seen recently. In both of these a diagnosis was made comparatively early in the course of the disease—in the first case on the microscopical appearance of the nasal granuloma, and in the second on account of the striking similarity of the nasal lesions to those seen in the first case.

Case 1

A bus inspector aged 49 was admitted to St. Nicholas Hospital, Plumstead, under the care of Mr. L. E. Gardiner, on June 18, 1951, for investigation of bilateral nasal obstruction, nasal discharge, and an aching pain over the nose of three weeks' duration. There was nothing relevant in his past history, and he had not been given sulphonamides.

Examination showed slight generalized swelling of the nose externally and a thick, blood-stained discharge; the nasal mucosa was widely ulcerated, and had a raw granular surface, covered in parts with a yellow crust. It bled easily when touched; there was no fetor. The patient's general condition was good apart from a slight pyrexia and some malaise. An x-ray film of his nasal sinuses was clear. A course of penicillin injections, 300,000 units daily for 12 days, cleared up the discharge, but did not heal the mucosal ulceration.

A biopsy of the nasal mucosa on June 25 showed appearances interpreted as possibly tuberculous, and an x-ray film of his chest on July 6 revealed tuberculous apical lesions of doubtful activity on the right side. He was given a course of streptomycin, 0.5 g. twice daily, for five weeks, with P.A.S.

Persistent failure to find the tubercle bacillus in the sputum led to revision of the diagnosis, and a further nasal biopsy (Special Plate, Fig. 1) on July 16 was sent to one of us (M.O.S.) and reported on as follows: "The material consists of fragments of granulation tissue containing a fair number of eosinophils, and a small number of multinucleated giant cells of foreign-body type and of Langhans's type. There are no areas of caseation, and no tubercle bacilli have been demonstrated; one cannot at this stage entirely exclude tuberculosis, but the condition this resembles most is not tubercle but the so-called 'giant-cell granuloma of nasal sinuses.' This condition has been reported as a presenting lesion in patients who have subsequently developed periarteritis nodosa."

At the beginning of August the patient developed slight left exophthalmos, and another x-ray film of his sinuses showed left antral infection. Mucopus containing *Staph. pyogenes* and pneumococci was washed out at puncture. Subsequently a muscle biopsy from the thigh showed a small acutely inflamed artery, confirming the diagnosis of periarteritis nodosa (Fig. 2). By the second half of August he had developed gross oedema of the limbs and a peripheral neuritis of the mononeuritis multiplex type. He became severely breathless with left ventricular failure, and a chest x-ray film showed "bat's wing" changes in the mid-zones. Throughout this illness the nasal picture was unchanged.

He was transferred to Lewisham Hospital on August 28 for A.C.T.H. treatment; three intravenous infusions, two of 30 mg. and one of 50 mg., were given without apparent benefit. He died on September 1.

Investigations.—July 19: E.S.R., 52 mm. in first hour (Wintrobe 100-mm. tube). August 5: haemoglobin, 8.89 g.%; red cells, 2,800,000 per c.mm.; white cells, 8,000 per c.mm. (polymorphs 72%, lymphocytes 26%); M.C.H., 31 $\gamma\gamma$; M.C.D., 7.2 μ ; E.S.R., 61 mm. in one hour. August 14: urine contained 40 mg. of protein per 100 ml. and a few red and white cells. August 27: haemoglobin, 7.96 g.%. August 29: blood urea, 84 mg. per 100 ml. Blood-pressure readings: July 18, 160/80; August 17, 180/120; August 28, 155/90.

Necropsy Findings.—Externally, oedema of legs. The heart showed hypertrophy of the left ventricle; all main arteries were healthy. Nasal, laryngeal, and tracheal mucosae were hyperaemic, and one minute granulomatous nodule was present in the tracheal wall. The lungs were airless, oedematous, and congested; there were a few thrombi in the terminal branches of the left pulmonary artery and a small caseous tuberculous focus at the left apex. Pus was present in the antra and ethmoid sinuses. The left kidney had a small infarct; many other organs showed effects of heart failure but were otherwise normal.

Histology.—Kidney: many glomerular tufts contained small fibrin thrombi and showed minute foci of fibrinoid necrosis of the capillary loops, with rupture and small haemorrhages into Bowman's capsule. In some places there were adhesions between the tufts and the capsule, and early crescent formation was present. A small recent infarct was present in the left kidney. No acute arteriolitis was seen, but an acute necrotizing arteritis was occasionally present. Other acute arteritic lesions, typical of periarteritis, were seen in the capsule of the adrenals, the lungs, a small branch of the left coronary artery, and one of the vasa nervorum of the left anterior tibial nerve. The mucosae of the maxillary antra showed inflammatory reaction, with only one giant cell.

Case 2

A maintenance worker aged 51 was first seen on August 13, 1951, with a three-months history of nasal catarrh and obstruction, mild earache and deafness, a husky voice, and some loss of weight. There was no record of his having had sulphonamides before coming to hospital. His general condition was good. The nasal mucosa was congested without ulceration or crusting, there was mucopus in his nasopharynx, and he had chronic laryngitis. An x-ray film showed infection of the left maxillary antrum, which was punctured on three occasions, some smelly pus being washed out each time.

Not much improvement resulted from these measures, and on September 21 a raw granular area, without crusting, was seen on the mucosa of the left side of the septum. A biopsy of this showed thickened transitional epithelium and underlying connective tissue with slight chronic inflammatory reaction. At about this time he began to feel ill, with night sweats, cough, and occasional rigors. Except that he looked ill and thin, a general examination on September 29 did not show any abnormal physical signs, apart from his E.N.T. condition.

He was admitted to St. Nicholas Hospital on October 4, as he had by then developed pains and weakness in his limbs. His septal mucosa was extensively ulcerated, and was covered with dirty yellow crusts which caused bleeding on removal. His tongue was red, smooth, and dry, and his pharynx injected and covered with sticky mucus. The appearance of the nose was so like that in Case 1 that a provisional diagnosis of periarteritis nodosa was made; in keeping with this, he was found to have a widespread mononeuritis multiplex, involving all four limbs in a flaccid paralysis with equivalent sensory loss, and associated with considerable oedema. He also had a tachycardia; rales

could be heard throughout both lungs; and an x-ray film showed "bat's wing" shadows in both mid-zones. The diagnosis was confirmed by muscle biopsy.

Later, a second biopsy of his nasal mucosa showed scanty lymphocytic infiltration but no giant cells. As a further antrum puncture still produced a little pus, a polythene tube was inserted for penicillin irrigation. On October 23 cortisone therapy was begun at Lewisham Hospital, and he was given 100 mg. by injection twice a day until he died, uraemic and in heart failure, five days later.

Investigations.—October 23: haemoglobin, 56%; red cells, 3,120,000; white cells, 28,000 (lymphocytes 20%, polymorphs 78%, monocytes 2%); blood urea, 128 mg. per 100 ml. October 27: blood urea, 270 mg. per 100 ml. Blood-pressure readings: October 13, 190/110; October 24, 175/105.

Necropsy Findings.—Externally, some oedema of ankles. The pleural cavities had some free fluid and fibrinous adhesions. The heart showed slight right and left ventricular dilatation, but was otherwise healthy. All lobes of lungs

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FIG. 1.—Serous exudation in liver of child aged 1½ years. (H. and E. $\times 150$.)

FIG. 2.—Serous exudation in the space of Disse. High-power of Fig. 1. Two sinusoids are shown widely separated from the columns of liver cells by serous exudation, which has markedly distended the perisinusoidal spaces. (H. and E. $\times 560$.)

FIG. 3.—Liver of child aged 2 years showing intralobular fibrillary collagen from previous attack and a serous exudate from current attack. (Picro-Mallory connective-tissue stain. $\times 600$.)

FIG. 4.—Wax-like eosinophilic coagulum in a perisinusoidal space, from the liver of a child aged 4 years. The coagulum gives the tinctorial reactions of collagen. (H. and E. $\times 680$.)

FIG. 5.—Serial section of same case as Fig. 4. Stained with picro-Mallory connective-tissue stain. Note the blue-staining coagulum in the perisinusoidal space: the sinusoid is clearly seen. ($\times 630$.)

FIG. 6.—Serial section of same case as Fig. 5, stained with modified Foote-Wilder reticulin stain: the reticulin fibres are swollen and stained black, but the "blobs" of coagulum are golden yellow in colour and are laminated. ($\times 520$.)

FIG. 7.—Liver of child aged 4 years. Stained with a modified Foote-Wilder reticulin stain. There is perivascular cuffing around the centrilobular veins with radiating perisinusoidal reticulosis. ($\times 600$.)

FIG. 8.—Serous exudate distending the space of Mall of a portal triad in the liver of a child aged 2½ years. (H. and E. $\times 370$.)

FIG. 9.—Liver of a child aged 2½ years, showing perivascular deposition of coagulum around an interlobular vessel extending between two portal triads. (Picro-Mallory stain. $\times 360$.)

FIG. 10.—Widespread deposition of eosinophilic coagulum in the space of Disse throughout the lobule of a child aged 3 years 3 months. The coagulum has a sparse cellular infiltration indicating early fibrosis. (H. and E. $\times 600$.)

FIG. 11.—Liver from a child aged 2 years showing hepatic fibrosis; stained with a modified Foote-Wilder reticulin stain. Note the centrilobular cuffing and the perisinusoidal and perilobular reticulosis. ($\times 70$.)

FIG. 12.—Liver from a child aged 2 years showing hepatic fibrosis. There are connecting bands between portal triads and centrilobular veins. (H. and E. $\times 150$.)

FIG. 13.—Shows marked hepatic fibrosis with lobule distortion and nodular reconstruction from liver of child aged 2 years. (Modified Foote-Wilder reticulin stain. $\times 140$.)

K. R. HILL, KATERINA RHODES, J. L. STAFFORD, AND R. AUB: SEROUS HEPATOSIS

H. J. M. STRATTON, T. M. L. PRICE, AND M. O. SKELTON: GRANULOMA OF NOSE AND PERIARTERITIS NODOSA

FIG. 1.—Nasal mucosa of Case 1, showing chronic inflammatory reaction and multinucleated giant cells. (H. and E. $\times 110$.)

FIG. 2.—Muscle biopsy of Case 1, showing acute inflammatory reaction in wall of small artery. (H. and E. $\times 90$.)

showed red areas of infarction. The pancreas had two small infarcts in its tail; and the spleen showed irregular pale-pink areas throughout the pulp due to necrosis in areas of infarction. The kidneys had no macroscopic vascular nodules; there was one infarct. Periarteritic nodules were seen along small branches of the superior mesenteric and left gastric arteries. There was flat ulceration in nasal cavities—none anywhere else in respiratory tract. Microscopically the kidneys were similar to those of Case 1. Periarteritis was seen in small arteries of the kidneys, liver, lungs, mesentery, ileum, sciatic nerve, soleus muscle, and in arterioles at centres of malpighian bodies of spleen. Right antral mucosa was normal; left antrum and nasal cavities showed chronic non-specific inflammatory changes. Other body organs were essentially normal.

Discussion

As Howells and Friedmann (1950) point out, there are in the literature several cases of periarteritis nodosa in which chronic sinus infection was noted in the previous clinical history without its significance being emphasized. Upper-respiratory-tract infection probably plays a more significant part in periarteritis nodosa than is generally

recognized, and the important feature of the particular syndrome here described is that it is sufficiently clear-cut to enable a diagnosis to be made early. In both our cases the subsequent development of periarteritis nodosa was predicted on the appearances of the nasal mucosa. The microscopical appearances of the fully developed granuloma with its large multinucleated giant cells (Special Plate, Fig. 1) are very striking. Giant cells, however, were not seen in the mucosa in Case 2, either in the biopsy specimen or in the fragments of nasal and sinus mucosa procured at necropsy.

The essence of this syndrome is that the upper-respiratory-tract lesions are the first, and at the outset the dominating, feature of the condition. The periarteritis nodosa picture develops subsequently. This course of events is shown in our two cases. Several cases have been quoted in the literature as examples of this syndrome which do not fulfil these criteria—for instance, the case described by Rössle (1936-7) in which the nasal lesions were incidental in the disease process and were not the initial changes; and the case of Banowitch, Polayes, and Charet (1942) in which a mediastinal mass of giant cell granuloma affected the lung and was associated with periarteritis nodosa. There were no nasal lesions in this case.

Limiting the discussion of the syndrome to those cases exhibiting upper-respiratory-tract ulceration with the subsequent development of periarteritis nodosa, one may speculate on the possible significance of this pattern of the disease process. It is a well-known theory that periarteritis nodosa results from a hyperergic antigen-antibody reaction in the tissues, particularly affecting the vessels but also leading to the development of the granulomatous lesions in the extravascular connective tissue, which sometimes show giant cells of foreign-body type. Such granulomatous lesions have been produced experimentally by Gøddard (1947), in his researches on the Arthus phenomenon, by injecting egg albumen into the skin of guinea-pigs; and numerous others—for example, Klinge (1933), Bergstrand (1946), and Pagel (1951)—have described the occurrence of granulomata in the connective tissue in periarteritis nodosa and in acute rheumatism and many others of the so-called "collagen diseases"; thus the nasal lesions in this syndrome may be regarded as special types of paravascular granulomata in the sense of the term used by Pagel, and are in accord with the theory that the disease is a manifestation of hypersensitivity.

There seem to be two possible explanations of the pattern of development of this syndrome. It may be that the patients have already developed generalized periarteritis and that the nasal lesions are merely the first to become apparent. There is no evidence to show whether or not generalized periarteritis nodosa was present at the time these patients first complained of nasal symptoms, and the clinical history at least suggests that the generalized disease followed later; but the patients were not submitted to a general investigation on their first attendance at the E.N.T. department with nasal lesions, and there are no data on this point.

A second possibility is that the upper-respiratory-tract lesions represent the relatively localized site of an attack on the body defences by some injurious agent of bacterial or chemical origin, and that widespread arterial lesions develop subsequently in a body now sensitized. This view is favoured by Wegener (1939) in his survey of the condition.

Another problem concerns the possible relationship between this disease and the condition known variously as "malignant granuloma of the nose" (Woods, 1921) or "progressive lethal granulomatous ulceration of the nose" (Stewart, 1933). The majority of the cases of this disease, which is almost invariably fatal, have not been examined after death, and still fewer have been examined histologically. However, Case 1 of the six cases described by Woodburn and Harris (1951) as examples of this disease and the third of Williams's (1949) three cases showed periarteritis nodosa at necropsy. Woodburn and Harris's Case 1 had

T. NICOL AND A. ABOU-ZIKRY: LEGENDS TO PLATE

FIG. 1.—Photomicrograph of section of spleen of intact male guinea-pig which received one subcutaneous injection of trypan blue daily at 4 p.m. for six days and was killed the following day. Shows a moderate number of dye-bearing cells in the sinuses of the red pulp. The dye-bearing cells appear black in the photomicrograph. (Weak eosin. $\times 200$.)

FIG. 2.—Photomicrograph of section of spleen of intact male guinea-pig which received 0.5 mg. oestradiol benzoate intramuscularly daily at 10 a.m. for four weeks plus one subcutaneous injection of trypan blue daily at 4 p.m. during the last six days before being killed. Shows numerous dye-bearing cells in the reticulum and in the dilated sinuses of the red pulp. The dye-bearing cells appear black in the photomicrograph. (Weak eosin. $\times 200$.)

FIG. 3.—Photomicrograph of section of spleen of intact male guinea-pig which received 0.5 mg. oestradiol benzoate intramuscularly daily at 10 a.m. for three weeks plus one subcutaneous injection of trypan blue daily at 4 p.m. during the last six days before being killed. Shows a large number of dye-bearing cells in a dilated sinus of the red pulp. The dye granules, which appear black in the photomicrograph, sometimes mask the nucleus of the cell. (Weak eosin. $\times 650$.)

FIG. 4.—Photomicrograph of section of spleen of orchidectomized guinea-pig which received 0.5 mg. oestradiol benzoate intramuscularly daily at 10 a.m. for four weeks, plus one subcutaneous injection of trypan blue daily at 4 p.m. during the last six days before being killed. Shows sinuses of red pulp dilated and empty of cells. Only a few dye-bearing cells can be seen in the reticulum. The dye-bearing cells appear black in the photomicrograph. The splenic vein in this animal contained many dye-bearing cells. (Weak eosin. $\times 200$.)

FIG. 5.—Photomicrograph of section of prostate of intact male guinea-pig which received 0.5 mg. oestradiol benzoate intramuscularly daily at 10 a.m. for four weeks, plus one subcutaneous injection of trypan blue daily at 4 p.m. during the last six days before being killed. Shows dye-bearing cells in the areolar tissue between the acini. The acini are surrounded by a thickened layer of fibrous tissue. The dye-bearing cells appear black in the photomicrograph. (Carbolfuchsin. $\times 200$.)

FIG. 6.—Photomicrograph of section of prostate of orchidectomized guinea-pig which received 0.5 mg. oestradiol benzoate intramuscularly at 10 a.m. for four weeks, plus one subcutaneous injection of trypan blue daily at 4 p.m. during the last six days before being killed. Shows a densely fibrous prostate. The acini are collapsed and the lining epithelium is thrown into folds. (Weak eosin. $\times 200$.)

two years' history of nasal obstruction, ultimately developing ulcerating lesions of the nose and trachea, and died after one year's illness; at necropsy an acute necrotizing arteritis was found in many organs. We feel that these two cases are perhaps examples of nasal granuloma with periarteritis, as described here.

We have been able to examine slides of nasal lesions and post-mortem specimens of four cases of classical malignant granuloma of the nose. No evidence of periarteritis nodosa was seen, and the local lesion was a chronic non-specific inflammation. Therefore we have no evidence to enable us to decide whether the classical destructive "malignant granuloma of the nose" is another manifestation of the condition previously discussed or something entirely different.

Summary

Two cases are described in which periarteritis nodosa was preceded by an ulcerating granuloma of the nasal mucosa. Seven similar cases, and possibly two others, have been collected from the literature.

The significance of the nasal lesions is discussed and the possible connexion pointed out between this condition and the so-called "malignant granuloma of the nose."

We wish to thank Mr. L. E. Gardiner for permission to publish Case 1; Dr. I. Friedmann, Dr. A. G. Stansfield, Mr. N. R. Jory, and Dr. R. H. D. Short for allowing us to see tissues from cases of "malignant granuloma" of the nose; Mr. S. E. Andrews for the photographs; and Mr. H. Eastwood and Miss K. M. Vale for technical assistance.

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The Lebanon Hospital for Mental and Nervous Disorders at Asfuriyeh, near Beirut, was opened in 1900, and since then has passed through many excitements and upheavals, but the work has gone on unbroken. Apart from a financial crisis during 1946-7, the hospital has grown steadily, the peak number of in-patients being 501 during the summer of 1938. There are now some 23 buildings (contributed by four countries) on an estate of 38 acres beautifully situated on the foothills of Mt. Lebanon, about five miles from Beirut. The present number of beds is 450, but, with the opening this year of a large new building, there will again be room for 500 patients. The latter come from 14 or 15 countries and are of a dozen different religions. They are of all types and classes, from the rich to the destitute. The hospital also runs a neuropsychiatric clinic in Beirut, which is doing important work for early cases. Developments since 1948 include a training school for Lebanese and Near Eastern student nurses, with an Asfuriyeh certificate in psychiatric nursing. Psychiatric instruction to doctors is of longer standing, and the present medical director, Dr. W. M. Ford Robertson, is clinical professor of psychiatry at the American University of Beirut, and two senior psychiatrists also share in the work of teaching. There are also special courses at the hospital for graduate doctors and nurses of various nationalities. A copy of the annual report of the hospital can be obtained on application to the Lebanon Hospital office, Drayton House, Gordon Street, London, W.C.1.

SEGMENTAL ASPIRATION PNEUMONIA AND BRONCHIECTASIS*

BY

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[WITH SPECIAL PLATE]

In a previous publication we described the clinical features of a large series of cases of segmental aspiration pneumonia (Robertson and Morle, 1951) and suggested that the syndrome of primary atypical pneumonia could be largely explained on an aspiration basis. Reference was then made to bronchographic studies undertaken in these cases of segmental pneumonia. It was stated that a wide range of bronchial abnormality might occur within such lesions and that these changes might be serious and sometimes irreversible.

These changes are now described and their significance is discussed in relation to the natural history of bronchiectasis.

So far we have carried out bronchography about 600 times on over 300 cases of segmental pneumonia. From so large a series we feel that enough evidence is forthcoming to substantiate our view that many, if not most, of the cases of bronchiectasis originate from these segmental pneumonias occurring in association with upper respiratory infections.

Bronchograms

There are many degrees of deviation from the normal, and in a few borderline cases it has been found difficult to decide whether a bronchogram was abnormal or not. This difficulty, which appears to have worried other authors (Evans and Galinsky, 1944), is a matter of some importance, as changes of such minimal nature may represent the initial stages of disease, which may later become fully established.

We classified a bronchogram as abnormal if one or more of the following features were present. (a) Dilatation: this varied in degree from minimal changes in calibre to gross and obvious dilatation. (b) Crowding: approximation and crowding together of the bronchial shadows. (c) Failure to Fill: failure to obtain satisfactory filling of the peripheral bronchi of the region studied.

Further features noted in those bronchograms classified as abnormal on the basis of the above three criteria included distortion and angulation of the peripheral bronchi and apparent irregularity of the bronchial walls. Some elucidation of the three main abnormal features is perhaps necessary.

Calibre changes vary from loss of the normal degree of taper of the bronchus as the periphery is reached to severe and obvious dilatation. In an estimation of the significance of an apparently dilated bronchus there are several features to be taken into consideration. An illusion of dilatation may be caused by solid filling of a group of bronchi. Allowance must be made for the magnification of the

*The substance of this work formed a paper given at a meeting of the Thoracic Society in July, 1951.