The freedom from hazard of tympanites and urinary retention permits use of this potent antihypertensive drug in circumstances where ganglionic blocking agents pose difficulty or danger. In the older patients with hypertension and failure, in nephritics with malignant hypertension, and in at least one man with sufficient prostatic obstruction to be of clinical significance, the use of bretylium tosylate has not given rise to unexpected complications.

CONCLUSION

Bretylium tosylate therapy would appear on the basis of this brief experience to be a significant advance in the treatment of hypertension; certain annoying to semi-disabling side effects produced by ganglionic blocking agents are not correspondingly significant when this drug is used. Pharmacological effects appear to be confined to sympathetic nerve interruption within the dosage limits used in this study. Subjective muscular weakness has been noted in two patients; in two others, digestive symptoms of undetermined cause, followed by vomiting and obstipation, occurred at relatively high dose. The possible presence of hyperchlorhydria from relative vagal predominance has not yet been investigated. It is stressed that in this short period of study no serious side effects have been observed, but in view of the short period of observation, this finding must be interpreted with caution.

May I acknowledge my gratitude to Dr. E. A. Fergusson, hospital superintendent, Drs. W. J. M. Cameron, G. W. Manning and M. M. Mosbaugh, and the Clinical Investigation Unit for their assistance and co-operation.

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RÉSUMÉ

En vertu de ses effets secondaires négligeables, le tosylate de bretylium semble marquer une étape importante dans le traitement de l'hypertension. Les effets pharmacologiques de ce nouveau médicament paraissent confinés à l'interruption du système nerveux sympathique dans les limites de la posologie employée au cours de cette présente étude. Parmi les 10 malades qui en ont reçu, deux se sont plaints de fatigue musculaire subjective et deux autres de symptômes digestifs de cause indéterminée suivis de vomissements et de constipation tenace lorsque la dose fut augmentée. Il se pourrait que l'hyperchlorhydrie causée par une prédominance vagale relative soit à la source de ces désordres mais l'auteur n'a pas poussé ses recherches dans cette direction. Au cours de cette courte période d'administration, aucun effet secondaire sérieux n'a été observé.

Case Reports

RENAL AND CEREBRAL SCLERODERMA*

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SCLERODERMA is now well established as a general systemic disease, so much so that the name progressive systemic sclerosis has been suggested. The following case is reported because of particular findings in two systems. The patient presented with an apparent nephrotic syndrome; this picture has previously been reported, but is uncommon. In the last few days of life, mental confusion and finally convulsions and coma occurred. Again, such symptoms have been reported, but are rare. At autopsy the kidneys, as well as some other organs, showed well-developed vascular changes of scleroderma. In the brain, there was an encephalopathy associated with similar vascular changes.

The patient, a 34-year-old housewife of Scottish origin, was admitted on January 27, 1957, to the Montreal General Hospital. She had had a cold at Christmas time, 1956, and then about January 17, 1957, she developed generalized œdema, an itching skin rash, burning micturition, nausea and vomiting and insomnia. On January 25, the family physician found that she was hypertensive and recommended admission.

She denied any illness before this last one. Aside from taking calomel on January 23, and occasional doses of castor oil, she had no history of exposure to drugs. She considered herself allergic to chocolate, which had caused nausea and vomiting on occasion, and was made rather ill by eating turkey.

After her death, it was learned from another physician that about 1941 her Wassermann test had been found positive in successive tests over some months, and that she had received full treatment with arsenic and bismuth for presumed syphilis. There was no

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TABLE I.—BLOOD CHEMISTRY

	January 28	*January 30*	*January 31*	*February 4*	*February 8*	*February 14*
Blood sugar (mg. %)	93.0					
Blood urea nitrogen (mg. %)	22.0		26	29.0	25.0	28
Creatinine (mg. %)	1.1					
Serum protein (g. %)	3.7			3.6	3.3	
Albumin (g. %)	0.7			0.65	0.5	
Globulin (g. %)	3.0			3.0	2.8	
CO_2 comb. power (mEq./l.)	32.0					18
Serum cholesterol (mg. %)		307				
Serum bilirubin (mg. %)				0.1		
Serum sodium (mEq./l.)					128.0	
Serum potassium (mEq./l.)					4.1	

history of primary luetic infection, and her husband's serology is thought to have been negative.

On admission she appeared to be about her stated age, well nourished, with obvious œdema of face, sacrum and legs. Blood pressure was 210/100 mm. Hg, pulse 80 and regular. Over the back, chest, buttocks and thighs were seen target-shaped macules with white centres. These tended to spread and become less distinct in outline as the days went by. A dermatological consultant described the skin lesions as: "Toxic erythema probably of the erythema multiforme type, usually secondary to drugs or some systemic disease". The diagnosis of scleroderma was not particularly considered.

No papillœdema was noted, but one linear hæmorrhage was seen in the right fundus.

Several heart murmurs were noted, namely, an early diastolic at the apex, a systolic along the left border of the heart, and an early diastolic at the left sternal border. There was dullness to percussion at the lung bases, but no rales were heard. The abdomen was swollen but no masses were felt.

X-RAY AND LABORATORY DATA

Urinalysis.—On admission, specific gravity 1.016; acid reaction; protein, 160 mg. %; white cells, 5-8; red cells, 15-20; no casts.

Subsequent urinalyses gave a variable specific gravity, as high as 1.023 on February 7 and 1.024 on February 15. Reaction was neutral in one specimen on January 29; on other occasions, acid. Protein content varied from 130 mg. % on February 13 to 300 mg. % on February 8. Glucose was never present. Moderate numbers of red blood cells and white blood cells were present in all specimens. Granular casts were seen from time to time and both granular and hyaline casts in the last specimen.

Blood chemistry.—See Table I.

Hæmatology: January 28.—Red blood cell count, 5,100,000; Hb. value 16.4 g. %; white cell count 8500. Hæmatocrit 48%; differential count: polymorphonuclear leukocytes 75%; basophils 1%; lymphocytes 21%; monocytes 3%; sedimentation rate: 46 mm. in one hour (Wintrobe). L.E. cell search—negative (also negative on February 7). *February* 12.—Hb. value 14.42 g. %; hæmatocrit 49%; white cell count 6450; differential count: polymorphonuclear leukocytes 82%; lymphocytes 17%; monocytes 1%. *Stool.*—Negative for occult blood January 29, and twice on January 30. *Bacteriology.*—Urine culture, February 1: light growth of *E. coli.* Throat swab, January 30: normal respiratory flora. *Electrocardiogram*, January 28.—T wave low in limb leads. T negative in V2, slightly diphasic in V4, flat in V6. Tracing interpreted as showing non-specific myocardial changes. *Chest radiography,* January 28.—Bilateral pleural effusion, with patchy infiltration at the right base. Heart shadow not grossly enlarged. Some accentuation of vascular pattern throughout both lung fields. *Serology.*—Not performed.

COURSE IN HOSPITAL

The working diagnosis on admission was acute glomerulonephritis, with a possibility of systemic lupus erythematosus. As laboratory results came in, it became apparent that the patient was suffering from nephrotic syndrome as a phase of some chronic renal disease, which was presumed to be glomerulonephritis.

She was treated by conservative measures for the first week, in the hope that the nephrosis might clear spontaneously. Digitalis therapy, using digoxin, was instituted on January 28. Dietary salt was restricted; fluid intake was moderately restricted. On this regimen she remained œdematous, and measured output remained almost constantly below fluid intake. The blood pressure had fairly constant diastolic readings from 65 to 90, but the systolic varied from 160 to 205.

On February 4, various forms of more active therapy were instituted. She was given cortisone 300 mg. intramuscularly daily, and meralluride, 2 c.c. intramuscularly, this day and also on February 9 and 13. Penicillin, 400,000 units daily, was started. The protein supplied in her diet was increased to 100 g. daily, though it is doubtful how much of this she ate. On February 5, and again on February 8 and 10, 25 grams of concentrated human albumin was administered intravenously. This seemed to promote some slight increase in output.

On February 7 a peculiar mental change developed. She became disorientated, then withdrawn and tense; the house physician considered her to be in a schizoid, almost catatonic, state. Cortisone was discontinued, and she appeared normal the next morning, so that it was resumed at a reduced oral dose, along with propantheline bromide. On February 10 prednisone was substituted for cortisone, the dose again being relatively high, 15 mg. four times daily; this dosage was subsequently progressively reduced until it was discontinued on February 13.

About 8:30 on the morning of February 14, the patient began to have a series of grand mal seizures which lasted until 2:30 that afternoon, at which time she was given 0.06 g. phenobarbital sodium intramuscularly.

At 6:00 p.m. on February 14, her blood pressure fell, and she went into a coma from which she never recovered. Blood pressure fell from 190/70 to 120/70 mm. Hg, and then progressively down to 85/42 at 5:30

a.m. February 15. There was a temporary recovery on the morning of February 15, but after a further fall of blood pressure, it could only be maintained by administration of noradrenaline. Her temperature rose on February 15 to 103.4° F. (R). Cheyne-Stokes breathing developed during the night of February 15-16, and she died at 8:00 a.m. on February 16.

Except for a small amount of incontinence and 90 c.c. obtained by catheter, there was no urine output in the last 24 hours.

POST-MORTEM FINDINGS

At autopsy two hours after death, the body showed generalized œdema, most marked over the lower extremities. A sparse dry maculo-papular rash was scattered over the body. Some of the macules had petechial red centres. Enlarged soft discrete lymph nodes were palpable in the axillæ and groin areas. The peritoneal cavity contained 1000 c.c. of clear serous fluid. Pleural fibrous adhesions were present on both sides, being more marked on the left. The left pleural cavity contained 100 c.c. of fluid, the right 600 c.c. The pericardial sac was completely obliterated by fine fibrous adhesions.

The heart weighed 400 g. Mitral and aortic valve leaflets were slightly thickened and sclerotic, as were several mitral chordæ tendineæ. One aortic cusp was lightly calcified. The mitral valve was slightly stenosed. The myocardium was uniformly beefy red and firm, with hypertrophy of the left ventricle.

The lungs were heavy, poorly aerated, firm and lumpy, and the cut surfaces showed a corresponding patchy grey consolidation.

The left kidney weighed 230 mg., the right 200 mg. They were similar to one another with regular outlines but a tense appearance. On section the cut surfaces bulged. The capsules stripped readily and external surfaces were pale and "flea-bitten" by numerous red patches 1-3 mm. in diameter. The cortices were thick (0.8-1.0 cm.), pale, and mottled with red puncta. Medullæ, papillæ, calices, pelves and ureters showed no gross lesion. The urinary bladder was contracted and practically empty. The lining of the bladder was pale, smooth and glistening.

Two intramural fibroid tumours were seen in the uterus. A small hæmorrhagic corpus luteum was present in the right ovary.

The œsophagus was eroded superficially in its lower third; otherwise it was grossly normal.

Lymph nodes throughout were enlarged, discrete and uniformly soft. The cut surfaces were pale, moist and rather fleshy in appearance.

On miscroscopic examination the fundamental lesion was found to be a widely disseminated alteration of smaller arteries and arterioles with swelling and œdema-like pallor of the walls and narrowing of the lumen. Nuclei were increased in size and number and were distorted and irregular with some karyorrhexis.

The abnormal vessel walls had irregular staining characteristics and various evidences of degeneration such as splitting, incomplete lamination, fenestration, vacuolization, reticulation and myxoid œdema. There was no fibrinoid necrosis except in the brain; no inflammatory reaction and no aneurysmal dilatation, and only rare and questionable thrombosis. Except in the brain sections, there was no hæmorrhage. The vascular changes were strikingly manifest in the kidneys, brain, lymph nodes, spleen and heart; and less marked in the

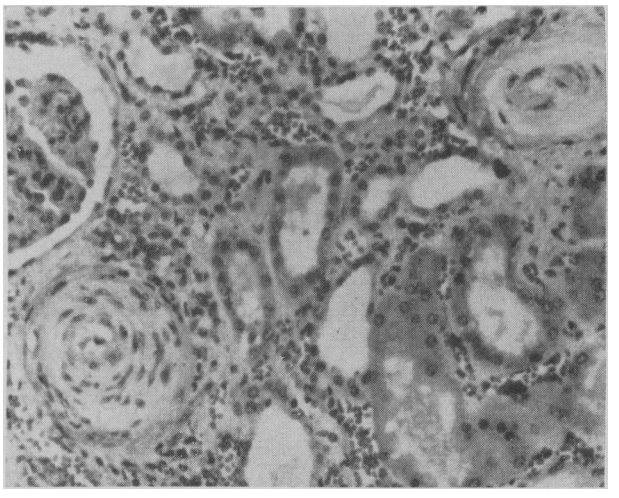

Fig. 1.—Scleroderma kidney with characteristic œdematous thickening and narrowing of small arteries (approx. ×150).

œsophagus, pancreas and liver. They were also presumed to be present in other locations including skeletal muscle, where distinction between post-mortem degeneration and true pathological alteration was difficult.

In the kidneys (Fig. 1), changes as noted above were seen in the subarcuate arteries and arterioles. There was also swelling of capillaries of the glomerular tufts with frequent small "wire-loop" effect and increased cellularity. The epithelial and endothelial nuclei were increased in size and number, with some irregular distortion and karyorrhexis. Syncytial epithelial hyperplasia was occasionally present. The glomerular tufts contained little blood. The capsular spaces were patent; they were either empty or else contained scant amorphous protein. The capsules were thin and well preserved. The convoluted tubules were dilated and contained abundant amorphous protein. A few hyaline casts and a rare granular cast were present. One tubule containing several polymorphonuclear leukocytes was found, and in a number of tubules there was regenerative epithelial proliferation. Interstitial tissue was not increased. Some œdema was present, and, rarely, local infiltration of inflammatory cells. These were chiefly lymphocytes, no polymorphonuclear leukocytes being identified.

Throughout the myocardium the typical vascular lesions were seen, with scattered areas of degeneration of muscle fibres. There were occasional minute stellate fibrous scars. No inflammatory cellular infiltration was present and no Aschoff bodies were found.

In the œsophagus there was localized acute exudative inflammatory reaction with superficial ulceration. This confused the histological picture but specific vascular lesions were present as described elsewhere, and, in addition, diffuse atrophy and fibrosis of the internal circular muscular coat.

The skin showed mild swelling of dermal collagen bundles and light infiltration of lymphocytes about adnexa and vessels. There were only slight vascular changes of a questionable character. The subcutaneous tissue showed no lesion.

In the lungs extensive bronchopneumonia was present, with leukocytic exudate but with moderate fibrinoid and large mononuclear cell components. Specific vascular changes were not evident.

Examination of Brain

The brain was large, weighing 1520 g. before fixation, and the convolutions were flattened against the dura. There was no significant degree of temporal lobe or cerebellar herniation. However, the mammillary bodies were displaced downwards, indicating some degree of downward movement of the brain stem, probably not enough to produce significant upper brain-stem compression. The major arteries and veins were not abnormal, nor were the meninges.

In the coronal sections of the cerebrum small vessels appeared unduly prominent. A small petechial hæmorrhage was recognized in the white matter of the left parietal lobe, and a small pale softening of irregular outline in the left thalamus. On sectioning the brain stem, another similar pale infarct was seen in the right restiform body. The ventricles were small.

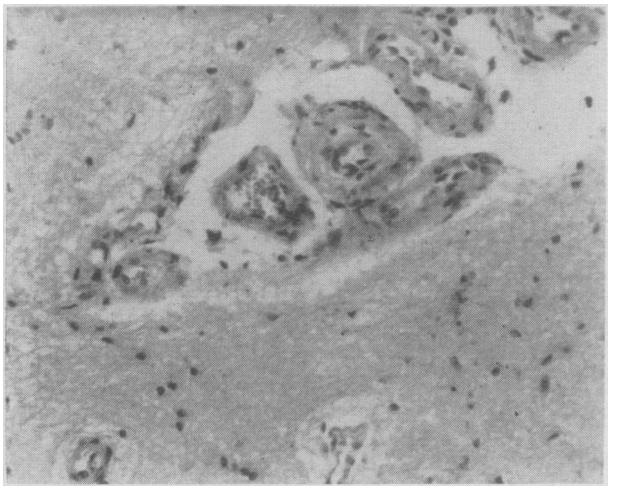

Fig. 2.—Scleroderma brain; œdematous stenosis of small arteries of meninges and cortex (approx. ×150).

The most striking histological change was found in the arterioles and small arteries of a diameter of approximately 50 to 200 microns (Fig. 2). Many of these vessels had a loose mucinous fibroblastic thickening of the intima, sometimes so severe as to occlude completely the lumen of the smallest vessels. Thrombi were present within the lumen of some of these vessels. This change was not recognized in the cerebellum or ependyma, and varied greatly in intensity in the meninges, cerebrum, basal ganglia and brain stem. In some areas groups of small arterioles, approximately 50 microns in diameter, showed smudgy necrosis, without any accompanying cellular reaction. A few of these vessels appeared to have leaked, and small clumps of red blood corpuscles were seen close to them. Scattered petechial hæmorrhages were found in some sections of the cortex; these clearly arose from small veins and appeared agonal. The veins were congested, but no other change was present, and the larger arteries appeared essentially normal. About two dozen small pale infarcts were found scattered through the cortex, white matter and brain stem, without any cellular reaction, and often contained surviving plump astrocytes within them. One infarct was seen with a considerable microglial and polymorphonuclear leukocytic reaction surrounding it. In addition, in other areas small groups of swollen nerve cells with central chromatolysis and ectopic large pale nuclei were present. The histological evidence of ischæmia was relatively trivial in comparison with the severe cerebral illness and the extent of the vascular change.

DISCUSSION

Renal Scleroderma

In the past 20 years scleroderma involving the kidneys has been described in several reports.[1-12] Most of the earlier cases, some of which will not be referred to here, have been summarized by Moore and Sheehan.[12] In recent years there have been some comprehensive reviews of the subject, variable numbers of cases being reported.[12-14]

The early impression was that the condition was a rarity, but more recently Piper and Helwig[15] have reported that in five out of 31 cases death was due to renal failure. Fisher and Rodnan[14] found renal involvement in nine of 11 cases studied. Leinwand, Duryee and Richter[16] state that all of their patients who came to autopsy had some involvement of the kidney. This was apparently appreciated by Osler who, in his text-book, stated that patients with scleroderma "are apt to succumb to pulmonary complaints or to nephritis".

In the course of these observations an appreciation of the morphological appearance of a "scleroderma kidney" has developed. Grossly, the cut surface contains nodular elevations with an appearance like subacute pyelonephritis. There are scattered red, congested or hæmorrhagic areas and occasional infarcts. The microscopic changes have been set down systematically by Calvert and Owen,[8] who enlarged upon the earlier description of Moore and Sheehan.[12] With some further modifications, the description is as follows:

1. In the intralobular arteries, proximally, there is a concentric mucoid intimal thickening with gross reduction in the lumen. Talbott *et al.*[1] speak of this thickening as being due to "a sparsely nucleated, patchy basophilic mucinous material which had the appearance of Wharton's jelly".

2. In the intralobular arteries, peripherally, fibrinoid necrosis of media and intima is present. This necrosis is also seen in some of the afferent arterioles.

3. Renal cortical ischæmic lesions develop, including fresh to old infarcts.

4. Areas of tubular degeneration and intertubular fibrosis are also seen. The convoluted tubules particularly are affected; the collecting tubules are relatively free.[14]

5. Glomerular changes are extremely variable. Typically the fibrinoid necrosis involves the walls of the afferent arterioles and glomerular loops.[13] Frequently, as in this case, occasional "wire loop" lesions, usually considered typical of lupus erythematosus, have been noted.[2, 10]

This overlap in the pathology of renal scleroderma and other related conditions has also extended in other directions. A similarity to periarteritis nodosa has been noted by some,[3, 4] and still others

have observed similarity to malignant hypertension.[14, 17, 18, 21]

Along with the accepted pathological appearance, a reasonably uniform clinical course has become apparent. Early in the course of a case of scleroderma, symptoms and signs suggesting renal involvement may be scanty or absent. It now appears that in the later stages hypertension is a common development,[13, 14] though previously it was considered uncommon.[12] The hypertension is rapidly followed by retinopathy, cardiac failure, convulsions, profound oliguria and uræmia. The progression of the disease from the appearance of hypertension to death is usually so rapid that there is little time for clinical evidence of renal disease to become fully apparent.

There have been frequent variations from this typical clinical course. In some cases renal involvement, along with skin lesions, was the first thing observed.[15] In two cases reported by Piper and Helwig,[15] as in the present case, scleroderma was not anticipated at all from the clinical course, but renal involvement was found at autopsy.

Recently several attempts have been made to deduce the natural history of the disease from the appearance of the various lesions. Moore and Sheehan[12] felt that the initial lesion was the dilatation of segments of the intralobular arteries and associated thickening of the intima. This seems to develop only a few weeks before death and leads to ischæmic atrophy of large portions of the renal cortex. The subsequent lesion is the fibrinoid necrosis of the distal intralobular arteries and afferent arterioles. This shows no healing at autopsy and probably develops only a few days before death. The acute ischæmic lesions in the related parenchyma also develop in the last few days of life and lead to the final deterioration of renal function and death.

Rodnan, Schreiner and Black[13] and Sokoloff[17] postulate that the renal lesions are an end result of localized vasomotor activity of the renal vessels, similar to that of Raynaud's phenomenon in the digital vessels. This abnormal state of vasoconstriction and dilatation leads to hypertension and the terminal fibrinoid necrosis. As there is a marked similarity between the renal lesions of scleroderma and those of malignant nephrosclerosis, it is possible, on the other hand, that the hypertension might also be the cause of the final stages. Obviously renal biopsies in diagnosed cases of scleroderma will be of value in clarifying the time-relationship of the pathological processes.

In several case reports and articles[5, 18-21] comment has been made upon an apparent relationship between steroid therapy for scleroderma and the development of the clinical signs of renal involvement leading to death; and considerable concern has been expressed about the possible harmful effects of steroids. It must be borne in mind that the typical hypertensive crisis has occurred in many patients, independent of such therapy. As yet no definite conclusions can be drawn as to whether steroids may induce these renal changes or merely accelerate a process which has already begun. If there is any connection between steroids and the development of renal involvement, possibly prednisone and further refined steroids may be less prone to produce such effects.

The case herewith presented differs in several respects from the typical one of renal scleroderma. First, although there were dermatitis and minimal vascular changes in the skin at autopsy, there was no characteristic sclerodermatous affection of the skin itself. Secondly, unlike almost all other reported renal cases, this one had a clinical picture in which symptoms and signs of renal disease were so prominent that scleroderma was not suspected *ante mortem*, though lupus erythematosus was considered in the differential diagnosis. Finally, in following a course like that of nephrotic syndrome, this case seems to have been almost unique. In only a few previous cases[3, 5, 7, 13] were œdema and severe albuminuria pronounced features; to a certain extent these cases resembled the present one. In the final stages this patient ran the typical course of hypertension, renal failure, convulsions and death, though neither the blood pressure nor blood urea level was excessively abnormal. It is probable that the neurological lesions also entered into the production of the convulsions.

In this case, as in those referred to above, it is possible that the fatal process was accelerated by steroid therapy.

Cerebral Involvement

There are very few detailed descriptions of clinical manifestations of involvement of the central nervous system by scleroderma, and only an occasional pathological description. It is doubtful that the condition of facial hemiatrophy, occasionally observed by the dermatologists to be associated with morphœa, is due to true scleroderma; but in 1922, Osborne[22] reviewed the literature of this condition and referred to recorded cases back to 1886; the evidence in the literature and in his own cases suggested involvement of the central nervous system.

Some other isolated neurological and neuropathological descriptions have been recorded.[9] Richter[23] described a patient with generalized scleroderma in whom the most prominent feature was peripheral neuropathy.

Earlier, convulsions in the course of scleroderma were considered rare and a possible indication of neurological involvement. Now, as was discussed in the previous section, convulsions have been reported frequently in the later stages of the renal cases, and are possibly related to the hypertension and uræmia.

In the present case the brain showed evidence of disseminated ischæmic encephalopathy with many minute infarcts which were mostly too recent to have developed a cellular reaction, and a generalized swelling of the brain, secondary to this

ischæmia. The cerebral illness began quite suddenly with a psychotic episode nine days before death, followed by a series of major seizures and coma for the last two days of life. The clinical course and pathological appearance were very similar to other forms of disseminated patchy, ischæmic encephalopathy in which multitudes of small arteries or arterioles are narrowed or occluded. The uncommon condition of severe hypertensive arteriolar sclerosis produces the same clinical and pathological course but at a slower pace, and a patient with thrombotic microangiitis, whose brain was examined in this laboratory, had an almost identical pathological picture. The clinical and pathological features of the much commoner encephalopathy of systemic lupus erythematosus are identical except for the different nature of the arterial and arteriolar lesions.[24] In our patient the narrowing of the minute vessels was caused by scleroderma; the venous hæmorrhages were considered agonal and of no significance. It is to be noted that most of the infarcts were recent and were too small and too few to account for the severity of the cerebral illness. This is the common finding in the more acute forms of disseminated ischæmic encephalopathy. It is probable that the blood supply to large regions of the brain had been reduced by the vascular occlusions to a point where the brain could still survive but no longer function. It is only where the disease was particularly intense that the blood supply was reduced to a point where infarction occurred. This patient had only mild uræmia, and the hypertension never reached levels commonly associated with hypertensive encephalopathy. Byrom[25] produced very convincing evidence that the disseminated ischæmic encephalopathy of this disease was a product of intense arteriolar spasm in reaction to extreme elevation of blood pressure. It is not unlikely that intense arterial and arteriolar spasm may develop in response to a much lower level of hypertension if the vessels are already damaged by scleroderma or systemic lupus erythematosus.

SUMMARY

A case of generalized scleroderma (progressive systemic sclerosis) is presented with autopsy findings. Clinical and pathological studies were of particular interest in reference to kidneys and brain. The kidneys were typical of scleroderma at autopsy, but the clinical course was unusual in that it resembled that of nephrotic syndrome until the final stages. Cerebral symptoms were attributed to ischæmia secondary to the sclerodermatous changes in the cerebral arteries and arterioles where the disease process was more intense than elsewhere in the body. The patient was treated with cortisone and prednisone, which may possibly have influenced the course of the disease. The literature on renal and cerebral scleroderma is reviewed.

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REITER'S SYNDROME OCCURRING IN THE FEMALE*

REPORT OF A CASE SHOWING THE COMPLETE TRIAD AND OTHER FEATURES

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REITER'S syndrome is rare in females. If the exact criteria of urethritis, conjunctivitis and arthritis are followed only four cases reported in the literature would qualify.[1-3] Other cases have been described but they had only two features of the triad, or gonorrhœa, or the protocol contained no direct mention of urethritis as distinct from vaginal discharge.[2-5] The patient reported here is interesting since she showed the three diagnostic features, and in addition, other mucocutaneous lesions.

A 33-year-old unmarried, white, school teacher came to the outpatient department of the Montreal General Hospital on September 28, 1958, complaining of profuse vaginal and urethral discharge, burning urination and painful inflamed eyes.

The patient stated that she had been subject to recurrent attacks of allergic non-purulent conjunctivitis each autumn for the past seven years. Six weeks previously she had had a single sexual exposure. This was followed ten days later by a bout of diarrhœa lasting one day. As this was receding she developed urethral irritation and vaginal discharge, both of which persisted. Two weeks later her eyes began to smart, bright lights bothered her and her eyelids stuck together in the mornings. Her companion stated that he had not had urethritis at the time of sexual contact, and subsequently he did not develop any symptoms.

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