LESIONS OF THE INTRAHEPATIC PORTAL RADICLES IN MANSON'S SCHISTOSOMIASIS *

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The adult worms of *Schistosoma mansoni* dwell in veins of the portal system, mostly those of the colon, and oviposition takes place in the corresponding venules in the wall of the colon. The organic lesions of schistosomiasis generally can be correlated with the vascular territory inhabited by the trematode. Faust[1] has shown this to be true also in the so-called ectopic lesions.

The vascular distribution of the eggs was strikingly demonstrated in one of our heavily infested cases (no. 125, Hospital das Clínicas). This patient was a 26-year-old man showing both advanced hepatic fibrosis and pulmonary arteritis. Eggs and pseudo-tubercles were found in eighteen different regions, including the central nervous system, myocardium, thyroid gland, and both kidneys (Figs. 1, 2, 3, and 4). Eggs and egg shells were demonstrated in renal arterioles; one of them in a glomerular hilus, presumably in an afferent vessel (Fig. 4). It was concluded that ova had been shunted through pathologic vascular anastomoses in the lungs, into the greater circulation, so that they were disseminated as emboli.

While the vascular distribution of ova is a well known feature of the disease, the lesions produced in blood vessels by *S. mansoni* have received little attention. The route taken by the egg from the vascular to the intestinal lumina during colonic oviposition is largely unknown.

Kohlschütter and Koppisch[2] have described a process of vascular extrusion characterized by adhesion of the egg to the vascular wall, coverage by endothelial cells which isolate the foreign body, and finally inflammatory change around the egg resulting in compression or obliteration of the involved vessel. At the intestinal venous and capillary levels, both gross and microscopic changes have been mentioned frequently and have been summarized recently by Valladares.[3] Inflammation of the larger portal trunks and of the main stem was mentioned many years ago by Letulle.[4] Koppisch[5] found several cases of

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extrahepatic portal thrombosis in a large series of necropsies but he considered this complication to be related to portal hypertension rather than to direct inflammatory excitation by the parasite. In the experimentally infested rabbit, Koppisch[6] described an early portal endophlebitis, characteristically "polypoid" in appearance. Review of the literature revealed only two descriptions of lesions affecting the intrahepatic portal radicles; namely, mention by Hashem[7] and by Bezerra Coutinho[8] of endophlebitic proliferation of vascular endothelium in radicles containing ova or remains of ova. In the lung, the arteritis described by Shaw and Ghareeb[9] is the best known and most destructive vascular lesion of schistosomiasis. Finally, in the case mentioned (no. 125, Hospital das Clínicas), inflammation of peripheral arterioles in the vicinity of impacted ova was observed (Fig. 5). Thus, while blood-vascular lesions have been observed in practically all localizations of schistosomiasis, their importance has not been emphasized sufficiently, particularly with reference to the liver.

Material and Methods

This study is based on 27 cases of Manson's schistosomiasis from a series of 52 consecutive non-selected necropsies at the Hospital das Clínicas of the University of Bahia (1952). These cases were studied from a clinicopathologic point of view and will be reported in detail elsewhere.[10] In addition, specimens of liver for biopsy had been obtained at laparotomy from 4 cases. The data obtained from the clinical and pathologic study were pooled, the gross material was reviewed, and a complete histologic study of the livers was undertaken, including the sectioning of new tissue blocks as necessary.

In addition to the routine hematoxylin and eosin, the following stains were used as needed: the Ziehl-Neelsen method for ova of *S. mansoni,*[11] the Unna-Tänzer orcein-methylene blue stain for elastic fibrils,[12] and Masson's trichrome stain for connective tissue. A highly satisfactory stain for vascular lesions of schistosomiasis is a combination of the first two stains: elastic fibers were stained overnight in orcein as in Unna-Tänzer's method[12] and ova were then stained by a modified Ziehl-Neelsen method,[11] followed by light counterstaining with methylene blue.

General Statistical Data

The 27 necropsy cases were divided into two main groups,[10] mild and severe, on the basis of anatomical lesions. The primary criterion was the presence or absence of intestinal fibrosis, hepatic fibrosis, or

pulmonary arteritis, and the secondary criterion was the presence or absence of evidences of portal or pulmonary hypertension. In this way, 20 cases (74.1 per cent) were found to be mild and 7 cases (25.9 per cent) were classified as severe. The latter usually showed a combination of advanced organic lesions of the intestine, liver, and lung, of which the hepatic changes proved to be the most constant. Six cases (22.2 per cent) had hepatic fibrosis, 3 of which (11.1 per cent) showed a typical Symmers' pipe-stem cirrhosis. Pulmonary arteritis was found in 5 cases (18.5 per cent), in 4 of which (14.8 per cent) it was accompanied by right ventricular hypertrophy. Three cases (11.1 per cent) had advanced fibrosis and rigidity of the colon.

These data show that the Brazilian cases of schistosomiasis were more severe and more heavily infested than those studied in Puerto Rico,[5] which is almost certainly due to a higher degree of infestation of the waters to which our cases were exposed and to more frequent reinfestation.[13]

The 4 biopsy specimens represented fragments of liver tissue obtained in the course of splenectomy. These cases had been diagnosed as Manson's schistosomiasis by parasitologic examination, and clinically they were classified as of the "splenomegalic form."

Vascular Lesions Observed

Lesions of the intrahepatic portal radicles of three types were observed: (1) substitution by granuloma, (2) sclerosis and narrowing, and (3) intrahepatic thrombophlebitis. Descriptions of these lesions follow.

Substitution by Granuloma (Figs. 6, 7, and 8). In the early stage of substitution by granuloma, the lumen of the portal radicle was still discernible, and it contained an egg or a more or less fragmented shell, more often than not engulfed by a multinucleated giant cell. The endothelium was swollen, proliferated, and partially desquamated. The media was partially or completely destroyed and invaded by leukocytes, epithelioid cells, macrophages, and numerous eosinophils (Figs. 6 and 7). Remains of elastic fibers could be demonstrated. As the lesion progressed, the vessel became completely occluded by a granuloma, and there was destruction of the muscular and elastic layers. Thus it was no longer possible to recognize the vascular origin of the lesion except by inference (Fig. 8). However, when the portal triads were inspected with special attention to the location of the vein, it was noticed that while some showed venous channels by-passing a granuloma, others did not seem to contain a vein at all. Serial sections

of two livers showed that the portal vein was indeed entirely substituted by a granulomatous formation.

Sclerosis and Narrowing (Figs. 9 and 10). The lesion of sclerosis and narrowing was found in portal fields showing enlargement, fibrosis, and chronic inflammation. When the portal triad was inspected, the vein at first seemed to be absent, but closer attention revealed a few compressed capillary channels surrounded by scattered cells with elongated nuclei and faintly eosinophilic cytoplasm. These structures could be identified as the remains of an almost atretic portal radicle and its fibrotic middle layer (Fig. 9). The identification was much easier with special techniques, especially with Masson's trichrome stain (Fig. 10). The lumen of the vessel was either narrowed or subdivided. The subintimal layer was greatly thickened and the fibrous tissue continued through the hiatuses between the muscle fibers into the dense connective tissue of the portal space outside of the vein. The muscle layer appeared atrophic and was separated into small fascicles. Usually there was little inflammatory change and the impression was that of a residual sclerosis rather than of an active process. While ova and granulomas might be present in the portal space affected, this was not the rule.

Intrahepatic Thrombophlebitis (Figs. 11, 12, 13, 14, and 15). Intrahepatic thrombophlebitis was the most severe vascular lesion found. It affected the larger veins in cases showing definite pipe-stem fibrosis. Since the fibrotic portal fields showed a highly irregular vascular pattern, it often was difficult to visualize the branch of the portal vein, but by special stains the lesion became evident (Figs. 11 and 12). The vein was only slightly smaller than normal, and its lumen was entirely substituted by fibrous and inflammatory tissue representing an organized thrombus. The tissue replacing the lumen might show tortuous, almost cavernous, vascular channels, which appeared to communicate with similar channels outside the vein through hiatuses in the muscle layer (Fig. 15). This tissue was infiltrated with lymphoid cells and occasional leukocytes. Numerous eggs could be demonstrated, occasionally surrounded by pseudo-tubercles. The media of the vessel was difficult to see with the hematoxylin and eosin stain, but could be visualized by special methods. Its muscle bundles were atrophied and separated by fibrous tissue and dilated capillary channels. The elastica was found to be partially destroyed and showed numerous duplications, hiatuses, and irregularities (Figs. 13 and 14). Groups of eggs could be seen between the hiatuses of the elastic layer (Fig. 14). All layers of the vessel wall appeared infiltrated with in-

flammatory cells which were found also in the surrounding portal fibrous tissue. The latter frequently presented eggs and pseudo-tubercles.

Intrahepatic thrombophlebitis could be recognized on gross examination of the cut surfaces of the liver. Systematic inspection of portal fields sometimes showed only two lumina corresponding to the hepatic artery and the bile duct, respectively, while the portal vein was replaced by a poorly defined, slightly porous, white tissue. This lesion was particularly well seen under the hand lens.

In addition to the lesions of the portal radicles, other vascular changes seen less constantly will be mentioned only briefly. These lesions appeared to be more frequent in the advanced cases and in those showing pipe-stem cirrhosis.

Capillary Telangiectasia of the Portal Fields (Fig. 15). In the most advanced manifestation of capillary telangiectasia of the portal fields, the dilated vascular channels had a distinctly "angiomatoid" aspect, paralleling the telangiectasia seen in the lung.[9] Grossly, the portal fields might be reddish and of a spongy appearance, instead of white and dense, as classically described.[5] The telangiectatic lesion was associated with the presence of ova, granulomas, and inflammatory foci.

Intrahepatic Arteriolar Sclerosis and Medial Hypertrophy (Fig. 10). Intrahepatic arteriolar sclerosis and medial hypertrophy were found in many advanced cases of schistosomiasis. Their degree and frequency appeared to be greater than in non-infested persons of similar age.

Incidence of Lesions

No vascular lesions were found in 8 necropsy cases (29.6 per cent), making 70.4 per cent the incidence of intrahepatic vascular lesions in the necropsy series. All 4 biopsies showed vascular lesions, but this figure is of little significance in view of the selected nature of the cases.

Substitution by granuloma, which represents the mildest change, was also by far the most frequent, having been found in all 19 necropsy cases showing vascular lesions. In many of these, the lesion was sporadic in distribution, and in only 9 cases (33.3 per cent) was it conspicuously frequent. It also was found in all biopsy cases. The lesion was seen both in mild and in severe cases, and with and without portal fibrosis; it did not show a significant correlation with portal hypertension. It seemed to be most conspicuous in 3 cases of rather recent, massive infestation, with numerous pseudo-tubercles of the liver.

Sclerosis and narrowing, and intrahepatic thrombophlebitis, on the other hand, usually were associated with hepatic fibrosis and with portal hypertension. Sclerosis and narrowing was found in 5 necropsy cases (18.5 per cent), 3 of which showed advanced hepatic fibrosis. The remaining 2 cases showed sporadic lesions, and were classified as mild, although one of them did show a slight degree of splenomegaly. It was found also in 3 of the 4 biopsy cases. The number of portal fields affected in the individual cases was somewhat variable, but in none was there any difficulty in finding representative lesions.

Intrahepatic thrombophlebitis was the most advanced lesion, as well as the rarest. It was found in only 3 necropsy cases (11.1 per cent), all considered severe, and showing pipe-stem cirrhosis and portal hypertension. As with the previous lesions, not all of the portal radicles were involved. In 2 of the cases only every fifth to sixth field appeared affected. In the remaining case, one third to one half of the rami were occluded.

Comment

The frequency of intrahepatic vascular lesions found in this study raises the question as to why they have not been emphasized by previous workers. One of the presumptive reasons for the lack of previous attention is the fact that the lesions described here leave the vessels either substituted by granulomatous tissue, or markedly atrophied and inconspicuous, so that it is necessary to search systematically for the portal radicles in order to discover these alterations which, furthermore, may be inapparent unless certain special stains are utilized.

A consistent theory about their pathogenesis cannot be offered at this time. The frequency of vascular lesions in other localizations of Manson's schistosomiasis has been discussed. It must be emphasized that the pulmonary arterial lesions are destructive and feature a rupture of the elastic membranes.[9,14] It seems significant that in the present study lesions of the elastic layer of the portal veins also have been found. However, the speed with which eggs become impacted and the vascular anatomy of the organs are different, and, while the lesions appear analogous, their pathogenesis may vary. It is conceivable that the embryo within the egg shell produces substances endowed with a histolytic effect in order to facilitate its extrusion and motion toward the site of elimination, but such substances have not been identified chemically to date. It seems of interest, however, that an acid-fast-staining substance was recently demonstrated in the eggs.[11] Oliver González[15] and others believe that the pathogenetic action of the schistosome is more intimately associated with the egg than with

other forms of the parasite. Lópes de Faría[16] has attributed great importance to the finding of hyaline thrombi and believes that an "allergic" mechanism is at work in the production of schistosomal pulmonary endarteritis. He also suggested that "substances produced by the ovum" are the cause of the endarteritis. Whether the mechanism of action of the eggs is predominantly toxic, lytic, of an allergic-immunologic nature, or mainly "mechanical," is a matter of speculation.

Of the lesions described here, both substitution by granuloma and intrahepatic thrombophlebitis appear to be due to an active inflammatory process associated with the presence of eggs. Fibrosis and narrowing of the portal venules is a late manifestation associated with portal fibrosis. It may represent either the scar of a previous phlebitic lesion or the result of fibrosis spreading from the outside to the inside of a vein in a previously inflamed portal field. Substitution by granuloma occurs early in the course of infestation, and probably does not lead to embarrassment of the portal circulation unless a large number of venules are involved. Fibrosis and narrowing are frequently accompanied by portal hypertension. Intrahepatic thrombophlebitis is found only in cases with Symmers' pipe-stem cirrhosis and is associated with portal hypertension. Anatomically, this lesion is analogous to pulmonary arteritis. Just as the latter represents the severest involvement of the lung, intrahepatic thrombophlebitis may signify the most severe lesion affecting the liver.

It must be emphasized that the thrombophlebitis, as described in this paper, is not the ordinary recanalization of a thrombus, but a true phlebitis, since all vessel layers are altered by inflammation. It is an intrahepatic process, not to be confused with extrahepatic portal thrombosis. Koppisch[5] has described the latter alteration in 3 of his 147 cases. In the series of 27 cases,[10] one showed thrombosis of the splenic vein which, however, did not coincide with intrahepatic thrombophlebitis. Furthermore, while extrahepatic portal thrombosis may complicate any hepatic disease producing portal hypertension, intrahepatic thrombophlebitis has been observed only in schistosomiasis.

There is little doubt that any form of hepatic fibrosis will eventually lead to intrahepatic vascular blocking and to portal hypertension,[17] but there are vast variations among different diseases of the liver in the degree of vascular participation. Portal hypertension is a predominant manifestation of severe Manson's schistosomiasis in all endemic areas.[5,18,19] The incidence and degree of splenomegaly seem to be higher than in other forms of hepatic fibrosis, and splenic Gandy-Gamna bodies, as well as intrasplenic hemorrhages, are particularly

frequent in this condition.[20] Phlebosclerosis also is frequent,[5] and thrombosis of the portal vein is not uncommon.[5] Esophageal varices are a prominent part of the clinical picture,[21] and recently it has been emphasized that they may exist in otherwise asymptomatic patients.[22] Portal hypertension of long duration, in the absence of liver cell failure, and resembling the Banti syndrome, is one of the characteristic forms of the disease.[19]

Attempts have been made to correlate the frequency of splenomegaly with toxic factors produced by the adult parasite.[20] However, this report shows that in Manson's schistosomiasis, the intrahepatic portal radicles are affected early in the course of the disease, even before fibrosis is fully developed, and that the later stages of the disease are accompanied by narrowing lesions of the portal veins.

It must be emphasized that these findings show only anatomical narrowing and do not permit judgment as to what degree of functional impairment should be attributed to it. Also, the ratio of obstructed to unobstructed vessels was not established. Injections of the hepatic vascular bed in the manner of McIndoe[23] may be helpful in clarifying these and other questions.

In spite of the reservations outlined, it is my distinct impression that the lesions of the intrahepatic portal vessels found in schistosomiasis are one of the causes of the high incidence of portal hypertension in this disease. In addition, the identification of these lesions establishes a parallel with other vascular lesions of schistosomiasis previously described in the intestine and in the lung, thus suggesting a somewhat more unified picture of their pathogenesis.

Summary and Conclusions

In a study of the intrahepatic portal radicles of 27 cases of Manson's schistosomiasis, hitherto undescribed vascular lesions were found in 19 (70.4 per cent).

The lesions were of three types: substitution by granuloma of a portal radicle; fibrosis and narrowing, a more advanced lesion found in fibrotic portal fields; and intrahepatic thrombophlebitis, an obstructive lesion of larger venous ramifications, which may be analogous to schistosomal pulmonary arteritis. Intrahepatic thrombophlebitis was found only in advanced cases showing pipe-stem cirrhosis.

Intrahepatic vascular lesions arise early in Manson's schistosomiasis, and are frequent in the severe cases. They may be one of the causes of the frequency and severity of portal hypertension in this disease.

The vascular lesions of Manson's schistosomiasis in the liver parallel the vascular lesions in other organs, most notably in the lung, and therefore suggest a similar pathogenesis.

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REFERENCES

1. Faust, E. C. An inquiry into the ectopic lesions in schistosomiasis. *Am. J. Trop. Med.*, 1948, **28**, 175–199.
2. Kohlschütter, E., and Koppisch, E. On the mode of extrusion of Schistosome ova from blood vessels into the tissues. *Schweiz. Ztschr. f. Path. u. Bakt.*, 1941, 4, 357–369.
3. Valladares, C. Determinações intestinais na doença de Manson-Pirajá da Silva. Thesis: Fundação Gonzalo Moniz, Bahia, Brasil, 1953.
4. Letulle, M. Bilharziose intestinale. *Arch. de Parasitol.*, 1905, 9, 329–439.
5. Koppisch, E. Studies on schistosomiasis mansoni in Puerto Rico. VI. Morbid anatomy of the disease as found in Puerto Ricans. *Puerto Rico J. Pub. Health & Trop. Med.*, 1940–41, **16**, 395–455.
6. Koppisch, E. Studies on schistosomiasis mansoni in Puerto Rico. IV. The pathological anatomy of experimental schistosomiasis mansoni in the rabbit and albino rat. *Puerto Rico J. Pub. Health & Trop. Med.*, 1937, **13**, 1–54.
7. Hashem, M. The aetiology and pathogenesis of the endemic form of hepatosplenomegaly "Egyptian splenomegaly." *J. Royal Egyptian M. A.*, 1947–48, 30, 48–79.
8. Bezerra Coutinho, A. Aspectos histológicos das localizações viscerais da esquistosomose de Manson. *An. Fac. de med. do Recife*, 1934, **1**, 16–21.
9. Shaw, A. F. B., and Ghareeb, A. A. The pathogenesis of pulmonary schistosomiasis in Egypt with special reference to Ayerza's disease. *J. Path. & Bact.*, 1938, 46, 401–424.
10. Lichtenberg, F.; Valladares, C., and Studart, J. Análise anatomoclínica de 27 casos de esquistossomose mansoni. To be published.
11. Lichtenberg, F., and Lindenberg, M. An alcohol acid-fast substance in eggs of *Schistosoma mansoni*. *Am. J. Trop. Med. & Hyg.*, 1954, 3, 1066–1076.
12. Lillie, R. D. Histopathologic Technic. The Blakiston Co., Philadelphia, 1948, pp. 198–199.
13. Coutinho, J. O. Contribuição ao estudo da esquistossomose mansônica no estado da Bahia-Brasil. *Arq. de hig. e saúde pub.*, 1951, **16**, 3–42.
14. Andréa, J. Localizações pulmonares da doença de Manson-Pirajá da Silva. Thesis: Era Nova, Bahia, Brasil, 1949.
15. Oliver González, J. Personal communication.
16. Lópes de Faría, J. Histopatología da endarterite pulmonar esquistossomótica. Thesis: Empresa Gráfica da "Revista das Tribunais" Ltda., Sao Paulo, Brasil, 1952. *See also:* Cor pulmonale in Manson's schistosomiasis. I. Frequency in necropsy material; pulmonary vascular changes caused by schistosome ova. *Am. J. Path.*, 1954, 30, 167–193.
17. Himsworth, H. P. The Liver and Its Diseases. Harvard University Press, Cambridge, Mass., 1948, p. 121.

18. Day, H. B. Bilharzial cirrhosis. (Egyptian splenomegaly.) *J. Trop. Med.*, 1933, **36**, 17–23.

19. Rodríguez da Silva, J. Estudo clinico da esquistossomose mansoni (doença de Manson-Pirajá da Silva). Thesis: Grafica Laemmert Ltda., Río de Janeiro, Brasil, 1949.

20. Meira, J. A. Esquistosomiase mansoni hepato-esplénica. Thesis: Tipografía Edanec Ltda., Sao Paulo, Brasil, 1951.

21. Muñoz-MacCormick, C. E. The injection treatment of esophageal varices due to Manson's schistosomiasis. Preliminary report. *Laryngoscope*, 1948, **58**, 992–1012.

22. Palmer, E. D., and Jahnke, E. J., Jr. Observations on portal hypertension among schistosomiasis patients with relatively insignificant complaints. *Am. J. Trop. Med. & Hyg.*, 1954, **3**, 139–146.

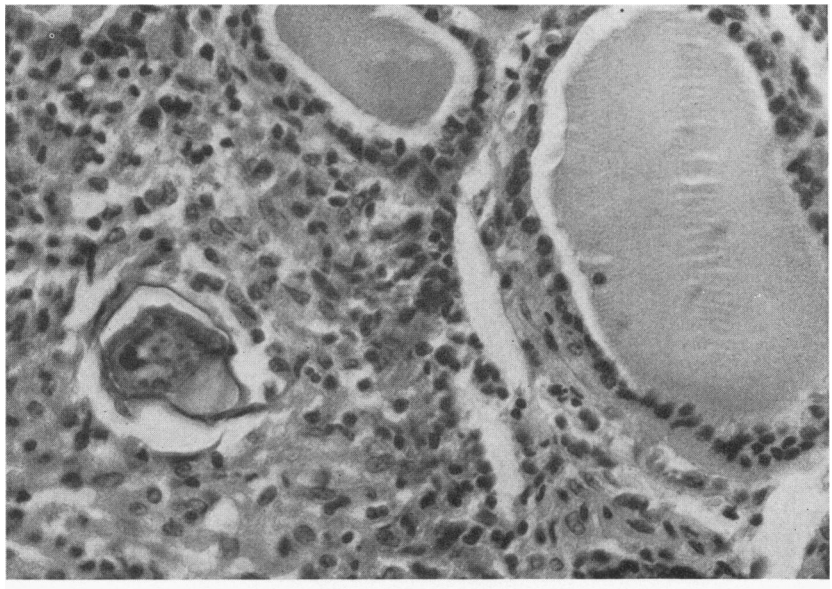
23. McIndoe, A. H. Vascular lesions of portal cirrhosis. *Arch. Path.*, 1928, **5**, 23–42.

LEGENDS FOR FIGURES

FIG. 1. Schistosome egg in thyroid gland, surrounded by a pseudo-tubercle. Hematoxylin and eosin stain. × 400.

FIG. 2. Egg in white substance of cerebellum, near a capillary containing a hyaline thrombus; the surrounding glial fibers have a reticulated appearance. Hematoxylin and eosin stain. × 400.

FIG. 3. Egg in the myocardium. The nuclei of the embryo are seen through the tangentially sectioned egg shell. The surrounding cells are mainly lymphocytes. To the left of the egg in the area of inflammation there is a partially lined space suggesting a capillary. Hematoxylin and eosin stain. × 400.



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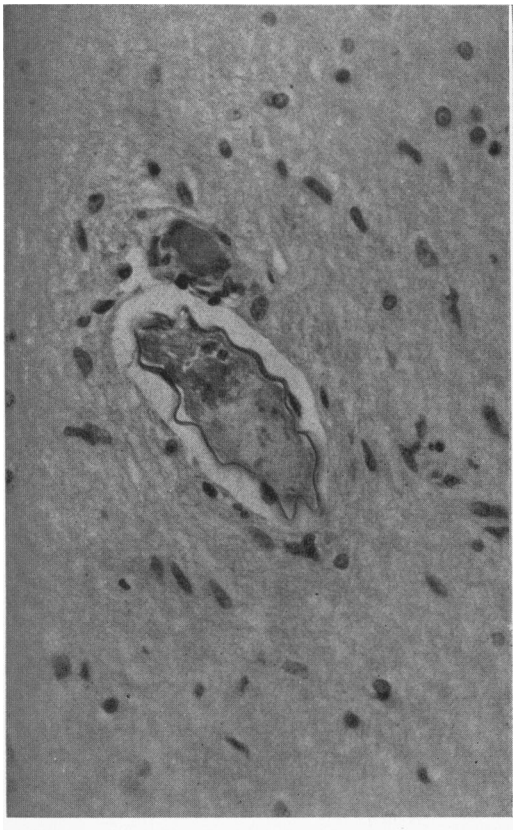

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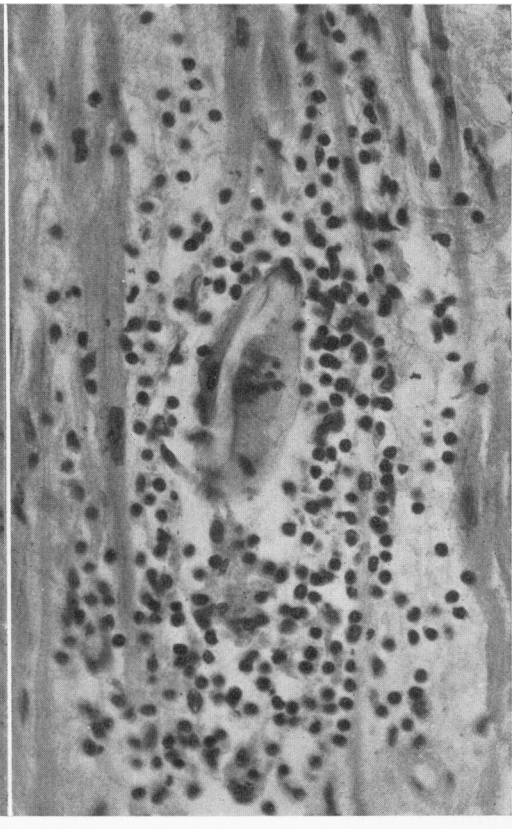

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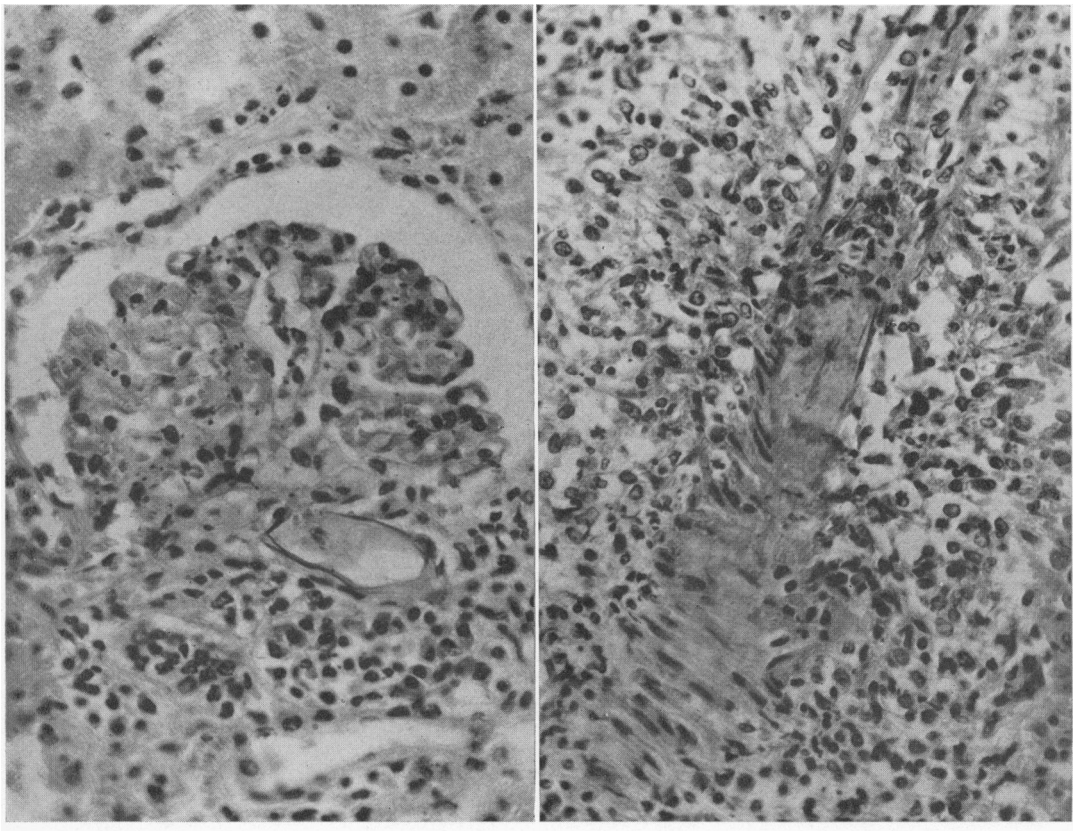

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FIG. 4. Egg shell impacted in a vessel of the glomerular hilus, presumably an afferent arteriole, surrounded by inflammatory cells. The black granules are produced by formalin pigment. Hematoxylin and eosin stain. × 400.

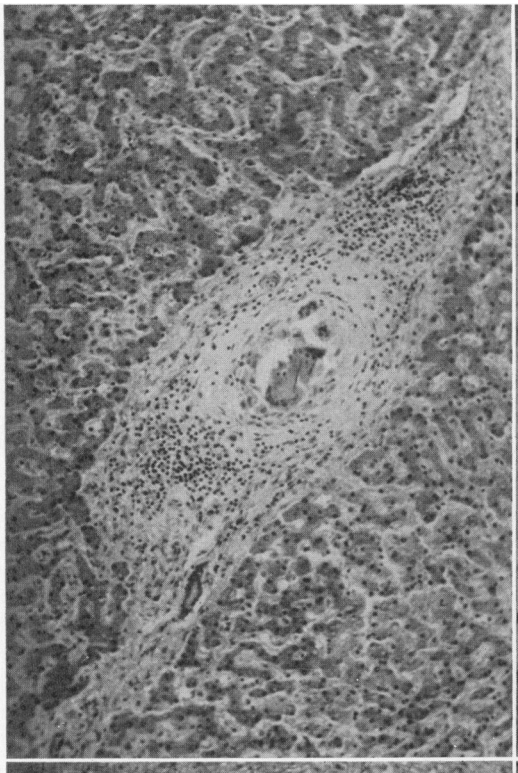
FIG. 5. Arteriole of renal cortex with impacted egg shell sectioned tangentially. Fibrinoid material is present below and to the right. The vessel is surrounded by a dense halo of inflammatory cells. Hematoxylin and eosin stain. × 400.

FIG. 6. Portal space showing initial phase of substitution by granuloma. Arteriole and bile capillary are below at the left. The lumen of the portal vein is occupied by an egg shell engulfed by giant cells. The proliferating endothelium resembles epithelioid cells. The muscle layer is not recognizable. There is marked round cell infiltration. Hematoxylin and eosin stain. × 120.

FIG. 7. The small arteriole (lower left) shows its elastica interna. Remains of elastic fibers are seen concentrically around the inflamed vein. Proliferation of endothelium around the egg is not yet sufficient to obliterate the vessel completely. Orcein-methylene blue stain. × 400.

FIG. 8. Complete substitution by granuloma. Arteriole and bile duct are in the upper left. The venule is substituted by a well developed pseudo-tubercle with centrally placed egg. Hematoxylin and eosin stain. × 80.

FIG. 9. Chronically inflamed and enlarged portal field showing sclerosis and narrowing of portal vein. Several small bile ducts and arterioles can be observed, but the main ramus of the portal vein is not immediately visible. The slit-like opening extending to the left from the center represents the remains of its lumen. About it there are occasional groups of elongated, dark staining cells representing the remains of the muscle layer. Masson's trichrome stain. × 80.



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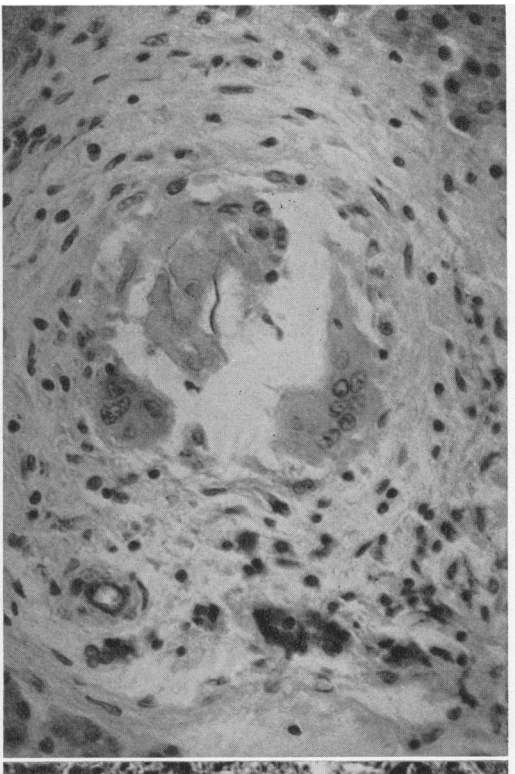

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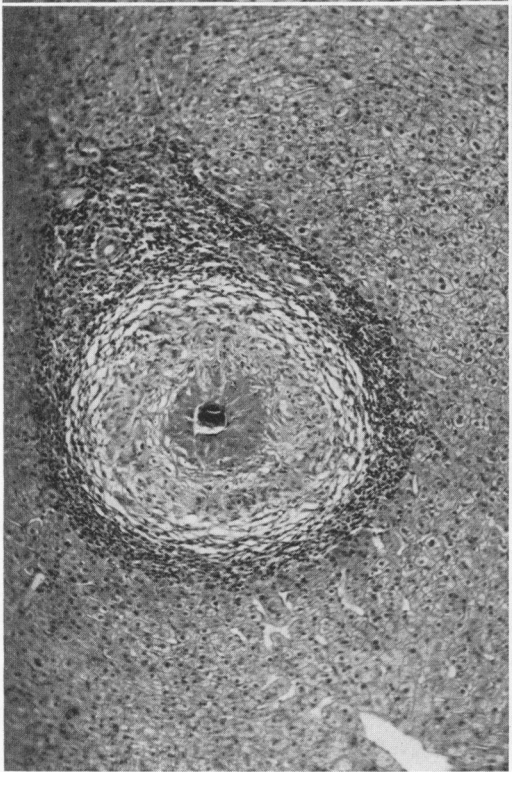

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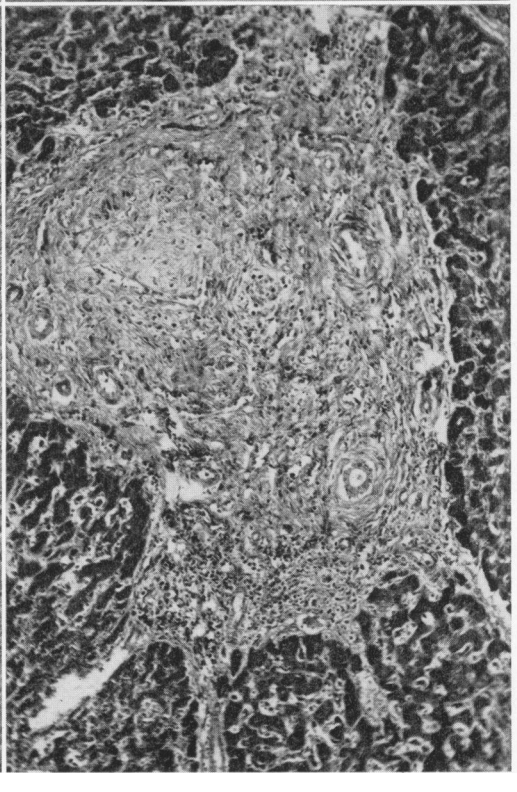

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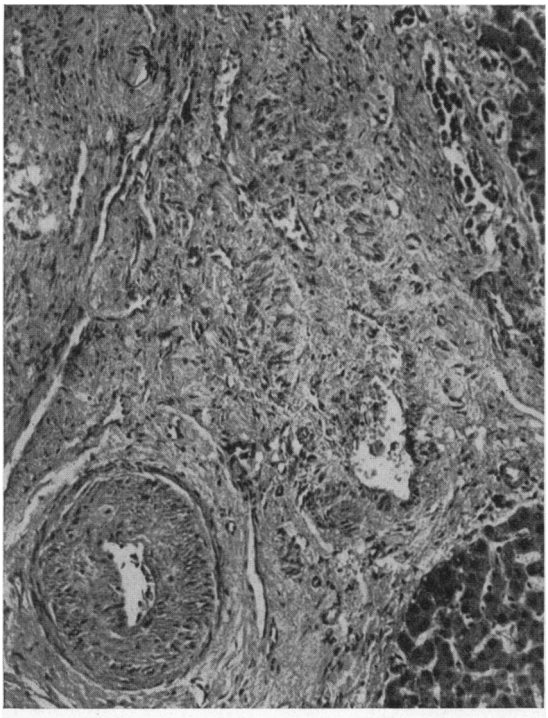

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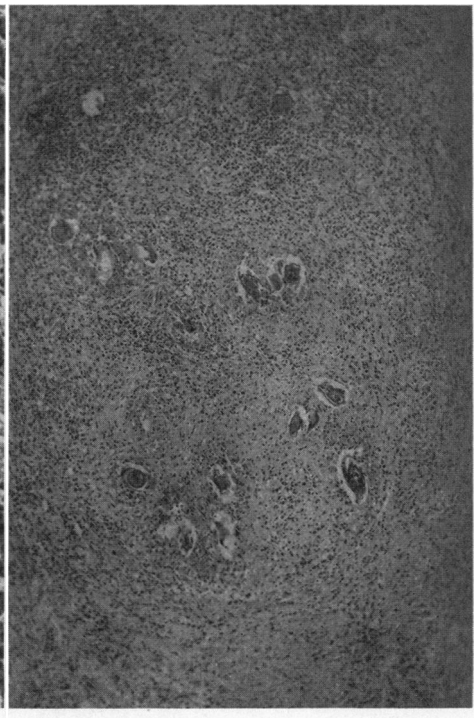

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FIG. 10. Part of an enlarged portal field. The artery shows marked intimal sclerosis and medial hypertrophy. The fibrous tissue is dense and contains numerous branching capillaries. The former outlines of the vein are marked by scattered atrophic muscle bundles, roughly elliptic (to the right of the center). Subdivision of the lumen and connection with adventitial capillaries may be noted; also absence of inflammatory infiltration. Masson's trichrome stain. × 120.

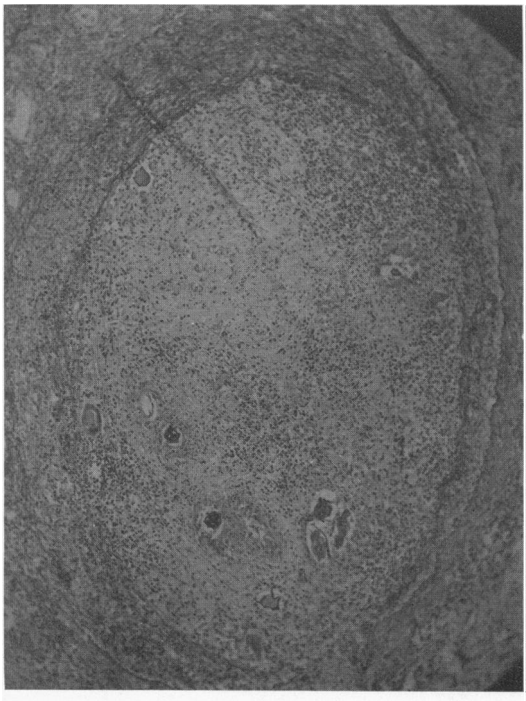
FIG. 11. Low-power view of large portal field in a case of advanced pipe-stem cirrhosis. This area is adjacent to the field seen in Figure 12 after elastica and acid-fast staining. It represents a partially obliterated portal vein with intrahepatic thrombophlebitis. The outline of the vessel is defined with difficulty. Hematoxylin and eosin stain. × 35.

FIG. 12. Low-power view of obliterated and inflamed vein showing intrahepatic thrombophlebitis. There are irregularities and interruptions of elastica, and dense inflammatory infiltration of all layers. Orcein-Ziehl-Neelsen's and methylene blue stains. × 35.

FIG. 13. Greater magnification of a field similar to that seen in Figure 12, showing irregularities of elastic layer. Orcein-Ziehl-Neelsen's and methylene blue stains. × 80.

FIG. 14. Detail of eggs close to the elastic fibers, which appear irregular and partially interrupted. The inflammatory elements are mostly histioid, lymphoid, and plasma cells. One egg shell is engulfed by a multinucleated giant cell. Orcein-Ziehl-Neelsen's and methylene blue stains. × 365.

FIG. 15. Telangiectasia of newly formed capillaries of a large portal field near the hilus of the liver, resembling "angiomatoids" of lung. A few fat cells are present and the inflammatory infiltration is dense and mainly lymphoid. Hematoxylin and eosin stain. × 90.



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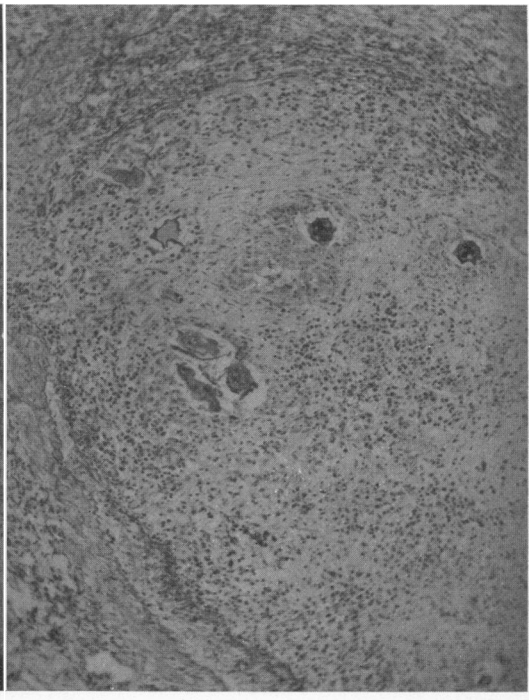

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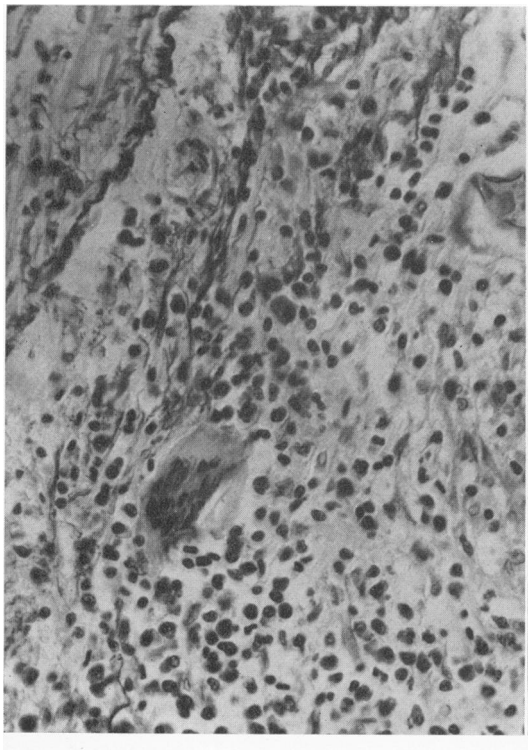
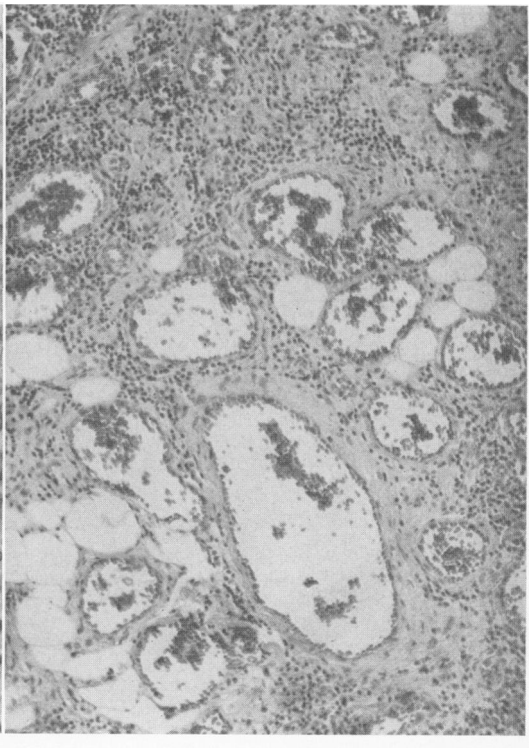


14



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