LIVER NECROSIS FOLLOWING BURNS*

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ATTENTION was first directed to a liver lesion associated with cases of burns when in 1938 Wilson, MacGregor and Stewart[1] in Edinburgh, reported severe liver necrosis in 13 of 33 patients dying after burns. Each of the cases had suffered second or third degree burns involving varying-sized areas of the body surface, and had survived for from two to twelve days. In many of the cases jaundice had developed as early as the third day and had persisted until death. The livers at autopsy were found to be slightly enlarged, soft, greasy, and friable. Microscopically, focal areas of necrosis involving individual lobules were found, the necrotic areas being central and mid-zonal in distribution, with viable liver cells around the portal areas.

Within the next few months Belt[2] independently reported four cases which had come to his attention while working in the Department of Pathology at the University of Toronto. Belt's cases had suffered second and third degree burns, and death in all cases had followed on the third or fourth day after the injury, one of the patients developing jaundice. At autopsy the lesions in the liver were found to be indistinguishable from those encountered in cases of yellow fever. The livers were described as being slightly enlarged, greasy, and pale yellow in colour. Microscopically, as in the cases of Wilson, MacGregor and Stewart, there was a focal necrosis and varying degrees of fatty degeneration. In addition, Councilman bodies were a prominent feature, and intranuclear inclusions were demonstrated in all cases. This combination of focal necrosis, Councilman bodies, and intranuclear inclusions had hitherto been described in two disease entities only, yellow fever and Rift Valley fever, both virus diseases.

Within the past year evidence of impairment of liver function in patients recovering from burns has been reported by Wolff, Elkinton and Rhoads.[3] These workers found that between the third and tenth days following the burn there was elevation of blood bilirubin, impairment of glycogenesis and hippuric acid synthesis, and a lowered blood prothrombin level, all pointing to a damaged liver.

Since the publication of Belt's paper we had been on the lookout for a burn case which showed liver damage, and in April, 1941, such a one was encountered.

CLINICAL HISTORY

The patient was a 33 year old female who, in a fire at home, had received second and third degree burns of the face, neck, shoulders, arms and hands, covering in all an area of 180 square inches (approximately one-twelfth of the body surface). She was admitted at once to the Toronto General Hospital where sedatives and continuous intravenous saline and glucose were given and the burns were tanned. On the following day the urine showed 2 + albumin. On the second day slight icterus developed, which rapidly progressed to deep jaundice, and the liver became palpable. By the fourth day there was a strongly positive and biphasic van den Bergh reaction (25 units) and a non-protein nitrogen of 78 mg. per cent. The patient seemed to improve and by the eighth day the blood bilirubin had dropped to 16 units and the non-protein nitrogen to normal, while the liver was no longer palpable. On this day, however, signs of pneumonia developed, and death occurred on the tenth day following the burns.

Autopsy.—The autopsy disclosed tanned burns in the situations noted above, with granulating bases and without evidence of infection. The skin, sclerae and viscera were moderately jaundiced. The lungs showed a purulent bronchitis and patchy small areas of consolidation, which proved to be areas of staphylococcal pneumonia. Multiple small foci of suppuration were present in the lungs, kidneys and myocardium. The liver weighed 1,200 grams, being perhaps slightly smaller than normal. It was quite flabby, greasy and friable. The capsular surface, which was smooth and without wrinkling, showed a yellowish mottling, while the cut surface presented tiny dark reddish areas standing out on a bright yellow background.

Microscopically, the liver showed an intense central and mid-zonal necrosis (Fig. 1). At the periphery of the lobules there was considerable interstitial œdema and the liver cells here, although viable, exhibited loss of uniform arrangement and fatty degeneration, while the peripheral canaliculi of many of the liver cords were filled with green-brown bile casts. The picture differed from that of acute yellow atrophy in that the necrosis affected portions of the individual lobule and not the whole of several adjacent lobules. Scattered throughout the liver, numerous fading-out and some well preserved Councilman bodies were visible. These took the form of vacuolated, hyaline-like structures, made up of the altered cytoplasm of one or more cells, and in some instances situated within the cytoplasm of individual cells. Occasional rounded, brightly eosinophilic, intranuclear bodies, of the group classed by Cowdry,[4] as type A inclusions, were seen. These varied in size from 3 to 7 microns, were usually centrally situated within the nucleus, and were surrounded by a clear halo of variable thickness. There was but little polymorphonuclear leucocytic response to the necrosis.

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COMMENT

Death in this case was due to a combination of liver necrosis, which was in some manner causally related to the burning, and staphylococcal pneumonia and pyæmia. The liver lesions were indistinguishable from those presented by Belt's cases, sections from three of which we have had the opportunity of studying (Fig. 2). They were likewise indistinguishable from the lesions seen in the livers collected by the late Professor Klotz[5] from a large series of human beings and monkeys dying of yellow fever. The changes in the liver in Rift Valley fever, also a virus disease, are similar to those of yellow fever, and many features of Daubney and Hudson's[6] and also Findlay's[7] descriptions of these changes might equally well have been applied to Belt's and our burn cases. The finding of intranuclear inclusions following burns supports the concept that inclusions of this type may be produced by a multiplicity of agents, of which viruses form only one group.

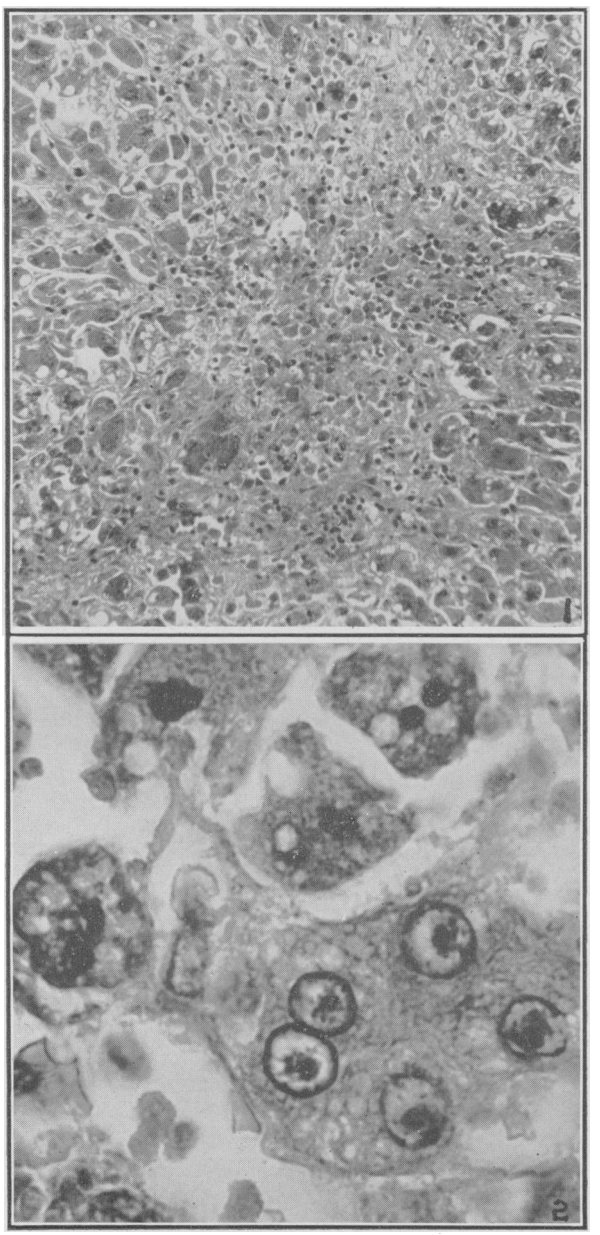

Fig. 1.—Necrosis of liver. Phloxine-methylene blue. x50. **Fig. 2.**—Five nuclei containing inclusions are seen in the right lower quadrant, with Councilman bodies above and to the left. (Belt's case 1). H. and E. x1250.

The actual cause of liver necrosis in cases of burns is obscure, but it is difficult to conceive of its being due to anything other than a circulating toxin, elaborated either in the burned tissue, or elsewhere as a result of the presence of burned tissue. Despite studies by numerous workers, however, the exact nature of this hypothetical agent is still undetermined.

SUMMARY

A case is reported in which jaundice and signs of severe liver damage developed following burns. Advanced liver necrosis was found. The lesions were indistinguishable from those encountered in the livers of previously reported cases of burns and of yellow fever, and closely resembled those described as being present in Rift Valley fever.

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