CHEMICAL CHANGES IN THE BLOOD OF THE DOG IN EXPERIMENTAL BILE PERITONITIS

By ALLEN M. ZIEGLER, M.D., and THOMAS G. ORR, M.D.

(From the University of Kansas School of Medicine, Kansas City, Kansas)

(Received for publication, March 24, 1931)

General bile peritonitis in the human being is usually fatal if permitted to continue for a sufficient period of time without treatment. Horrall (1) gives the mortality rate as 50 per cent or more. The same author has found that bile from the gall bladder injected into the peritoneal cavity of dogs in quantity as large as 5 cc. per kilo of body weight will cause peritonitis and death within 24 hours. Wangensteen (2) found that bile peritonitis produced death in both dogs and rabbits within 24 hours.

In such a rapidly fatal condition it seemed probable that a study of the blood chemistry would show definite and constant changes. Definite changes have been noted in the chemistry of the blood of the dog in general peritonitis produced by ligating the appendix (3).

We report below changes noted in the non-protein nitrogen, urea nitrogen, chlorides and CO_2 combining power of dogs with bile peritonitis produced by draining bile from the gall bladder directly into the peritoneal cavity.

Methods

Dogs weighing 11 to 18 kilos were used. In every instance the operation was done under complete ether anesthesia with aseptic technic. The contents of the gall bladder were drained into the peritoneal cavity by cutting a slit in the fundus or by clipping off a small portion of the fundus. The common duct was not ligated. The animals were kept in metabolism cages and permitted to drink water as desired. Blood for chemical studies was drawn from the jugular vein before operation and after operation at least once each day until the death or recovery of the animal.

EXPERIMENTAL OBSERVATIONS

Drainage of bile directly from the gall bladder into the peritoneal cavity did not always produce death. Several of our dogs recovered

TABLE I

Bile Drained into Abdominal Cavity

Dog No.	Days after operation	Blood				Remarks
		Amount per 100 cc.			CO_2 combining power	
		Total non-protein nitrogen	Urea nitrogen	Chlorides		
1	0	27.7	10.5	480	48.3	Weight 11 kg., 600 gm.
	1	35.7	25.9	420	46.6	
	2	28.0	18.9	400	47.5	
	3	45.6	12.6	400	52.0	
	4	42.2	12.2	420	50.2	
	5	86.3	39.9	330	—	Died 5th day. No culture
2	0	40.0	21.7	520	41.9	Weight 14 kg.
	1	27.3	10.5	500	38.1	
	2	27.0	9.1	520	38.1	
	3	25.0	10.5	510	34.3	Appeared well
	4	28.9	7.0	540	28.7	Began feeding
	5	48.2	27.3	510	50.2	
	6	27.7	13.3	520	38.1	
	7	29.7	12.0	580	36.2	
	36	54.0	32.2	460	47.5	Reoperated upon 36 days later. Drained gall bladder into abdominal cavity
	37	126.0	80.5	400	20.9	Died within 48 hrs. Culture negative
3	0	31.4	13.3	450	34.3	Weight not recorded
	1	86.3	37.8	380	33.4	
	p.m.	137.0	92.4	350	—	Died within 36 hrs. Culture negative
4	0	21.8	7.4	450	38.1	Weight 18 kg., 400 gm.
	1	85.4	45.2	180	12.9	Died within 24 hrs. Culture positive
5	0	31.2	10.7	430	32.4	Weight not recorded
	1	30.0	13.5	350	40.9	
	2	98.0	73.9	600	38.1	
	p.m.	110.0	85.4	410	—	Died within 48 hrs. No culture
6	0	46.0	26.6	560	32.8	Weight 12 kg., 200 gm.
	1	57.0	24.3	630	27.3	
	p.m.	65.0	30.3	415	—	Died within 36 hrs. Culture negative

TABLE I—*Concluded*

Dog No.	Days after operation	Blood				Remarks
		Amount per 100 cc.			CO_2 combining power	
		Total non-protein nitrogen	Urea nitrogen	Chlorides		
7	0	27.5	12.6	510	36.1	Weight 10 kg., 200 gm.
	1	35.3	12.6	480	40.3	
	2	50.0	19.6	460	29.1	
	3	50.8	12.8	430	32.8	
	4	46.0	20.0	470	39.3	
	5	50.8	20.0	440	32.8	
	6	48.2	17.7	450	40.3	
	7	41.3	17.7	430	31.0	Dog killed. Abdomen full of bile-stained fluid. Began to eat on 7th day following operation and appeared improved. Culture negative. This animal probably would have recovered

completely without evidence of illness. Autopsy of these animals showed the gall bladder healed with no evidence of bile peritonitis. Dogs developing a bile peritonitis which caused death invariably showed definite changes in the blood chemistry. There was an increase in the non-protein nitrogen and urea nitrogen and a moderate decrease in the whole blood chlorides. The carbon dioxide combining power did not show any constant change. However, in three of the animals there was a marked decrease in the carbon dioxide combining power of the blood just before death.

Dog 2 recovered completely from the first drainage of the gall bladder into the peritoneal cavity. On the 36th day the abdomen was reopened and the gall bladder again drained. Death resulted within 48 hours. Dog 7 was apparently recovering when killed with chloroform on the 7th postoperative day. Definite changes were noted in the blood chemistry.

At autopsy all dogs showed the peritoneal cavity distended with bile-stained liquid. Culture from the ascitic fluid of dogs dead with bile peritonitis showed positive cultures in one case and negative cul-

tures in four cases. In two cases no culture was taken. Anaerobic cultures were not made in this series.

DISCUSSION

After drainage of bile directly into the peritoneal cavity dogs died in from 1 to 5 days. It is evident that dog bile is quite toxic. Our animals lived somewhat longer than those of Wangensteen and Horrall, probably because the common duct was not ligated and the rate of flow of bile into the peritoneal cavity varied in different animals. We agree with these authors that sufficient bile, either injected or drained into the abdominal cavity of dogs, will invariably cause death. Apparently infection is not a contributing factor. We consider the positive cultures found in our animals a contamination.

The changes in the blood chemistry are similar to those found in experimental peritonitis in dogs. The increase in the non-protein and urea nitrogen in the blood is probably due to increased tissue destruction. The fall in chlorides is best explained by vomiting.

CONCLUSIONS

1. Changes in the chemistry of the blood of dogs with experimental bile peritonitis are here reported.
2. In all animals that died, a bile ascites was found.
3. Dogs dying of bile peritonitis showed a constant increase in the blood non-protein and urea nitrogen, and a fall in the chlorides.

LITERATURE

1. Horrall, O. H., *Arch. Int. Med.*, 1929, **43**, 114.
2. Wangensteen, O. H., *Ann. Surg.*, 1926, **84**, 691.
3. Orr, T. G., and Haden, R. L., *J. Exp. Med.*, 1928, **48**, 339.