Effect of Blockade of Transdiaphragmatic Absorption of Bacteria on Survival in Peritonitis: An Experimental Study in Rats

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Abstract

An experimental rat model of established peritonitis was used to test the effect of intraperitoneal injection of platelet rich plasma (PRP) on blood and peritoneal fluid culture positivity and survival rates. Thirty animals divided into two groups of 15 each were studied. The first group served as control while animals in the second group received intraperitoneal injection of PRP. The use of PRP in established peritonitis was of no significant benefit. (Indian J Gastroenterol 1992; 11: 168-9).

Keywords: Platelet rich plasma

Introduction

Bacterial peritonitis due to spillage of intestinal contents into the peritoneal cavity is a frequent and dangerous surgical condition. If untreated, septicemic shock, intraperitoneal abscesses and adhesions may develop, leading to significant morbidity and mortality. Most deaths occur in the early stages of untreated infection, i.e., in the initial 12-72 hours among humans and in 24 hours among experimental rats. The high mortality is believed to be largely due to absorption of bacteria and their endotoxins from the peritoneum.

Intraperitoneal administration of platelet rich plasma (PRP) immediately after experimental induction (by colonic perforation) of peritonitis has been shown to improve survival rate and to reduce positive aerobic and anaerobic blood culture rates in animals. Platelet's block absorption of bacteria from the peritoneal cavity into the systemic circulation, by plugging gaps between the peritoneal mesothelial cells. Since, in clinical practice, treatment is usually started after peritonitis is already established, we undertook this experimental study to evaluate the effect of intraperitoneal injection of PRP on blood culture positivity and survival rates in established peritonitis.

Material and Methods

Thirty adult Wistar rats of either sex weighing between 175 and 220 g each were studied. Peritonitis was induced in all animals by intraperitoneal injection of 4 mL of a suspension containing Escherichia coli (dose 10⁷ organisms/mL), Bacteroides fragilis (10⁷) and Clostridium perfringens (10⁷). These bacterial concentrations were selected to mimic those in human feces, since fecal contamination is the commonest cause of peritonitis.

The animals were divided into two groups. Those in the test group (n = 15) received two doses of 3-4 mL of PRP each intraperitoneally, first 6-8 h after the induction of peritonitis and another 6-8 h later. The 6-8 h interval was chosen because most patients with peritonitis reach the hospital after this period. PRP was prepared from fresh clotted O:R-negative human blood by centrifugation at 1500 rpm for 10 min at 22°C; the platelet count achieved was 400,000-500,000/mL. The control group (n = 15) received 3-4 mL of sterile saline intraperitoneally at similar intervals.

Cardiac blood obtained at 6, 24 and 72 h after the induction of peritonitis was directly plated on MacConkey agar and blood agar plates. Animals surviving on the 7th day were killed. Autopsy was carried out in all the animals to look for the presence of pus in the peritoneal cavity or macroscopic changes in the viscera. Peritoneal fluid was obtained at autopsy and cultured.

Survival and blood culture positivity rates in the two groups were compared using the χ² test and a level of 0.05.

Results

Ten control and seven test group animals died in the first 24 h (p = ns); no deaths occurred after the first 24 h. The blood culture positivity rates at 6 (10/15 vs 12/15), 24 (2/8 vs 2/5) and 72 (2/8 vs 2/5) after induction of peritonitis were similar in the two groups. Similarly, peritoneal fluid culture positivity rates in the control and study groups were similar (12/15 vs 10/15, p = ns).

Autopsy

Animals dying within 24 h showed the presence of 4-5 mL of peritoneal fluid, dilated bowel and congested viscera. These features were less marked in the test animals than in the control animals. At day 7, all test
animals showed the presence of 2-3 mL of purulent fluid in interloop areas, fibrin flakes and fibrous adhesions over the bowel and visceral congestion, whereas the control animals showed minimal fluid, fibrin flakes and visceral congestion.

Discussion

Animal studies have shown that platelets can prevent bacteremia by blocking the absorption of bacteria through the gaps between mesothelial cells on the peritoneal surface of the diaphragm. It is not known if a similar process occurs in humans too.

Dumont et al. demonstrated increase in survival and negative blood culture rates when absorption of bacteria was blocked, immediately after their introduction into the peritoneal cavity, by the use of PRP. Since such a situation would be very rare in clinical practice, we tried the use of PRP in established peritonitis.

Our results demonstrate that in established peritonitis there is no significant change in 24-h mortality (67% vs 47%) with PRP injection. Similarly, no significant difference was observed in the number of negative blood and peritoneal fluid cultures.

As PRP localizes the septic process to the peritoneal cavity, presence of peritoneal collection / fibrinous adhesions and visceral congestion was noted more frequently in the study group than in the control group. It has been reported that fibrin deposits reduce early mortality but increase late morbidity and mortality since they act as a barrier against neutrophilic antibacterial activity and systemic antibiotics. Their effect on long term morbidity and mortality could not be evaluated in our study as the animals were killed on the sevenths day.

We conclude that the use of PRP in established peritonitis is of no significant benefit. As our study had a small sample size, it is proposed that a larger sample should be studied.

References


Conference Report

The Mid-Term National Symposium of the Indian Society of Gastroenterology (ISG) was held on June 27-28, 1992 at Nizam’s Institute of Medical Sciences, Hyderabad (NIMS) under the leadership of Dr V K Dixit, Organizing Secretary. It included i) four symposia viz (a) Peptic ulcer disease, (b) Gall stone disease, (c) controversies in portal hypertension, and (d) Viral hepatitis, ii) nine guest lectures and iii) a panel discussion on ‘Controversies in management of duodenal ulcers’.

The symposium was inaugurated by Sri K Rosaiah, Honorable Minister for Medical and Health, Andhra Pradesh in a simple ceremony. Dr J P Gupta, President, ISG released the souvenir on the occasion. Dr D Raja Reddy, Director, NIMS presided over the function.

The scientific session started with a lecture by Prof N Madangopalan on the ‘Historical aspects of Gastroenterology in India’. It included a gem of collections, quotes, photographs, etc, particularly to the youngest in this field. The excellent talks on ‘Subacute hepatic failure’ and Hepatitis C & E by Prof B N Tandon and Dr Toshio Shikata (Japan) were universally appreciated.

The lectures on ‘Corrosive Acid Injury’ (Prof S K Mehta, Chandigarh), ‘Abdominal tuberculosis - recent trends’ (Prof P Raja Sambandham, Madras), ‘Irritable bowel syndrome’ (Prof P J P Gupta, Varanasi), ‘Non ulcer dyspepsia’ (Prof S L Broor, New Delhi) and ‘Current status of shunt surgery in Non-cirrhotic portal hypertension’ (Dr Aradar Choudhary, New Delhi) were very informative.

The local faculty included Prof C M Habibullah, Prof Bajrang Pratap, Prof Rupender Prasad, Dr Selhu Babu, Dr Manu Tandon, Prof G Satyanarayana, Dr R A Sastry, Dr Nageswar Reddy, Prof P N Rao, Dr Vidya Sagar, Dr Ajit Kumar, Dr Y R Nagajuma Kumar and Dr D V Sreenivas.

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