Cirugía y Cirujanos

Volumen *Volume* 70 Número *Number* 4 Julio-Agosto *July-August* 2002

Artículo:

Mannheim Peritonitis Index Validation Study at the *Hospital General de Durango* (Mexico)

Mannheim Peritonitis Index Validation Study at the Hospital General de Durango *(Mexico)*

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Resumen

Para confirmar el valor predictivo del índice de peritonitis de Mannheim (MPI) en la población de afluencia al Hospital General de Durango, SSA se realizó un estudio evaluando la severidad de la peritonitis y buscando ponderar cada factor de riesgo. La sencillez del MPI lo hace ideal para hospitales con carencias, comunes en nuestro país.
Se estudiaron 176 casos del 1º de marzo al 30 de noviembre de 1999, dividiendo a los pacientes en las siguientes categorías (puntuación MPI): a) < 21 pts., 21 – 29 pts., > 29 pts.; b) ≤ 26, > 26. Se elaboró una tabla de supervivencia para comparar la sobrevida por grupo y se calculó la razón de momios analizando la presencia de cada factor de riesgo y la supervivencia.
La diferencia en sobrevida entre los tres grupos (< 21, 21 – 29 y > 29) fue estadísticamente significativa (p < 0.0001). Los pacientes con > de 26 pts. MPI tuvieron una mortalidad del 40%, mientras que aquéllos con < no llegaron al 3% de mortalidad.
Todos los factores de riesgo, excepto el origen colónico, se comportaron de manera esperada y fueron especialmente orientadores: la presencia de falla orgánica, tiempo transcurrido > 24 h., la presencia de malignidad, edad > 50 años y la presencia de peritonitis generalizada.

Palabras clave: peritonitis, sistemas de puntuación, factores predictivos.

Summary

A study to confirm the predicative value of the Mannheim peritonitis index (MPI) among patients at the *Hospital General de Durango* was undertaken to evaluate the severity of peritonitis and to make a prognosis of survival-mortality pondering each risk factor. The simplicity of MPI makes it ideal for hospitals with serious shortages such as those in Mexico).
From March 1 to November 30, 1999, 176 cases were studied. Patients were divided into groups according to the following categories (MPI points): a)< 21, 21 – 29 and >29, and b) ≤26 and >26. A life table was constructed to compare patient survival with peritonitis severity. Odds ratios that analyzed presence or absence of each adverse factor and outcome were calculated.
Survival curves of the three subgroups (<21, 21 – 29, and >29) had differences that were statistically significant ($p < 0.0001$). Patients with >26 points had a mortality rate of 40%, whereas patients with ≤26 MPI points did not reach a 3% mortality rate. All MPI adverse factors, except of colonic origin, behaved as expected, and the following were especially useful: organic failure; time elapsed ≥24 h; malignancy; age >50 years, and generalized peritonitis.

Key words: Peritonitis, Scoring systems, Outcome predictors.

Introduction

Peritonitis is still one of most important infectious problems that a surgeon has to face. Despite of the progress in antimicrobial agents and intensive care treatment, the present mortality due to diffuse superative peritonitis from 10 to 20% continues to be unacceptably high[1,2].

At the surgical department of our Institute, the *Hospital General de Durango* (HGD), 11% of patients admitted have peritonitis among their diagnoses, which corresponds to two percent of the admissions to our Emergency Service. Of all surgical procedures undertaken at our Hospital, seven percent are diagnosed with peritonitis. Considering abdominal surgery exclusively, peritonitis corresponds to 21% of diagnoses[3].

This is compatible with what has been informed by other Mexican and Latin-American institutions. In a period of 4 years (1995–1999) at the Medical and Surgical Emergency Unit of the *Hospital General de México*, 14.76% of patients operated on had abdominal sepsis[4]. A South-American report from the *Instituto de Investigación en Ciencias de la Salud* in the city of Asuncion, Paraguay (1989) indicates that 2.8% of operations at this institute correspond to peritonitis[5].

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Recibido para publicación: 07-01-2002.
Aceptado para publicación: 27-05-2002.

Reproducible scoring systems that allow a surgeon to determine the severity of the intradominal infection are essential to: 1) ratify the effectiveness of different treatment regimens, 2) scientifically compare surgical intensive care units, 3) help indicate individual risk to select patients who may require a more aggressive surgical approach and 4) be able to inform patient´s relatives with greater objectivity[6].

In the past 30 years, many prognostic scoring systems have been developed for critical patients. Despite their design for general application, some have proven specifically useful in septic patients[7]. The results of treatment for peritonitis are specially difficult to evaluate because these patients may correspond to various etiologies, treatments differ, and there is a lack of universally valid criteria and definitions[8]. Presently, one of the most accepted scores is APACHE II, which integrates various physiologic variables during the first 24 h within the intensive care unit (ICU) with age and chronic health status of the patient. This initial stratification of risk factors and a predicative equation estimate patient outcome. They are, however, both complex and time consuming[9-11].

Many second-level Mexican hospitals are required to deal with serious shortage of equipment and lack of staff. Most of our provincial general hospitals do not even have an ICU. According to present regulation; the National Health Secretariat, the presence of an UCI should be considered in hospitals with 180 or more beds; this implies that less than 10% of all general or specialized hospitals within the Health Secretariat (33/405) should have an ICU, so use the APACHE II score in these conditions is not practical[12,13].

In 1986, Wacha H et al. published the Mannheim peritonitis index (MPI) based on analysis of 17 possible risks factors in patients with peritonitis; only eight factors were truly relevant to prognosis (age, sex, organ failure, cancer, duration of peritonitis, involvement of colon, extension of spread and character of peritoneal fluid) and were finally included in the index. The score considers clinic risk factors routinely found in preoperative and transoperative registers[14]. This information is obtained during first laparotomy to establish an initial classification. Early evaluation of severity of illness using MPI allows us to estimate the probability of patient survival[8,15]. The MPI is one of the most simple scoring systems in use that allows the surgeon to easily determine outcome risk during initial surgery. The recollection of retrospective data is possible and valid, because MPI only requires information routinely found in surgical registers[15].

Since initial publication in 1986, 29 articles that validate or use this scoring index can be consulted at the MEDLINE data base, all European studies, all except one, published in Old World Journals[6,14-41]. The simplicity of MPI makes it ideal for hospitals with serious shortages, such as those in Mexico. Because the abovementioned articles were favorable and due to the absence of U.S. studies using MPI, the need for a validation study of our work place was justified.

A study to confirm the predicative value of MPI among patients with intraoperative diagnosis of peritonitis at the surgical department of the HGD was undertaken, to evaluate severity of peritonitis and to make a prognosis of survival-mortality, considering the risk factors analyzed in this index.

Patients and Methods

At the HGD, a prospective, descriptive, transversal and observational study was undertaken. Patients included were all male and female patients, 16 years of age or older, seen at the Surgical Service of the HGD with diagnoses of peritonitis confirmed during surgery regardless of etiology. The patients operated on and diagnosed at other hospitals, or without surgical confirmation of peritonitis were excluded. Surgical and medical treatment was determined by the surgeon. The one-tail sample size, calculated with a precision of 0.05 and expected a mortality of 20%, corresponded to 174 individuals.

Once diagnosis of peritonitis had been determined by operative findings registered in the postoperative report, the patient was accepted into the study. Using data recollection sheets, risk factors found in MPI were classified according to values indicated in Table I and individual variable scores were added to establish initial MPI score. In addition to personal data such as name, age, sex, etc., the following intrahospital information was registered: file number; dates of admission and discharge from the hospital; days hospitalized; date of surgery and information related to illness (surgical findings, medical treatment and evolution of illness). Patient evolution was followed, indicating presence of complications and discharge due to improvement or death. Time elapsed from initial diagnosis to moment of event (death or discharge from hospital) was determined. Out-patient follow-up was continued for 30 days to establish perioperative morbidity and mortality. The minimum possible score was zero, if no adverse factor were present, and maximum was 47 if presence of all were confirmed.

Patients were divided in three groups according to the following categories (MPI points) fewer than 21; from 21 to 29, and more than 29. These categories and useful clinical reference at 26 MPI points were considered as done in the study published by Billing et al.[15] A life table, using the actuarial method, was constructed to compare patient survival with peritonitis severity according to MPI score. To determine significance of possible differences among three categories (< 21 points, between 21 and 29, and >29 points), Logrank method was used (significance of 0.05 and two degrees of freedom according to numbers of groups compared K = three groups-1 = 2).

Considering the following null hypothesis "There will be no difference in mortality in patients with peritonitis with scores of >21, from 21 to 29, and >29 points in MPI"; if $\chi^2_{cal} \geq \chi^2_{tab}$ of 5.99, null hypothesis is rejected and it is inferred that survival curves of the three categories are significantly different.

To validate each risk factor, patients who survived were separated from non-survivors, studying each parameter of MPI. A contingency table was constructed to analyze presence or absence of adverse factor and result (death vs survival) to calculate odds ratio (OR) observed.

Results

From March 1, 1999 to November 30 of the same year, 184 patients with peritonitis confirmed during surgical intervention were admitted to the Surgical Service of HGD. Of these patients, 176 meet with the inclusion criteria to be admitted to the study, excluding eight. From this total, two were eliminated, one who was transferred to another hospital and the other who requested voluntary discharge. We terminated the study on November 30, 1999 having completed the calculated sample.

Study Group General Data

Of the sample of 174 patients, 84 were female (48%) and 90 were male (52%). Group mean age was 34.6 years with a median of 27 years and a range from 16 to 93 years.

Mean age of survivors was 32.7 years of age (SD ± 16.64); among non-survivors, mean age was 63 years (SD ± 18.94) (p <0.0001).

Of the 174 patients that were operated on 11 died (global mortality 6%). Eighty two percent of survivors (133/163) evolved without complications and were discharged from the hospital. Subsequently, three patients reentered the hospital: one with secondary pleural effusion treated with endopleural tube to evacuate hemopneumothorax due to trauma and two for intestinal occlusion (one responded favorably to conservative treatment and the other was intervened). Eighteen percent (30/163) of survivors initially operated on presented some complications during their stay in the hospital: 18 patients had wound infection, three of these had wound dehiscence; three patients had intraabdominal abscesses that were drained; one had postincisional hernia; three had intestinal occlusion; one had gastrointestinal hemorrhage; one, hyperthermia; three required reintervention; one had lung thromboembolism and three had respiratory insufficiency and required assisted mechanical ventilation.

The patients spent a mean of 4.8 days in the hospital, a range 0 to 26 days. Ten patients required intensive care (6%) and one (0.57%) coronary care after presenting ventricular extrasystole. Mean length of stay of survivors was 4.72 days (SD ± 4.56) and non survivors, 6.09 days (SD ± 5.15)

Origin of peritonitis was from 11 different anatomic sites and was due to various causes (Table II). Sixteen patients with appendicitis had ruptured appendix. Of 34 patients with abdominal trauma 15 were due to stab wounds (three with thoraco-abdominal lesion), 12 had gunshot wounds, and seven, closed trauma. Among patients with cholecystitis, one had alithiasic cholecystitis, two cholascos, two hydropyocholecystitis (both with necrosis), and eight pyocholecystitis (one ulcered and the others ruptured). Of pathology of gynecologic origin, we found ruptured ectopic pregnancy in one patient, ruptured ovarian follicle in three, seven with ovarian cysts (three with hemorrhage, one ruptured, one twisted, and the other presented a pelvic abscess), in one patient salpingitis, and in another uterine perforation. Of complicated hernias one was incarcerated umbilical hernia, two postincisional hernias (one strangled, the other incarcerated) one strangled inguinal hernia, an incarcerated crural hernia, a strangled inguinal hernia with necrosis and perforation of the small bowel and two diaphragmatic hernias with intestinal occlusion. Of patients with gastric pathology, four had perforated ulcers and the other, stomach neoplasm. Patients with small bowel pathology included three with intestinal occlusion due to peritoneal bands (one with internal hernia, the other with perforated intestine), one with ileitis (perfo-

Table I. Mannheim peritonitis index score assigned to each risk factor

Study variable	Adverse factor	Points	Favorable factor	Points
Age	>50 years	5	–50 years	0
Sex	Female	5	Male	0
Organic failure	Present	7	Absence	0
Malignancy	Present	4	Absence	0
Evolution time	≥24 H	.4	< 24 Hrs	0
Origin	Non-colonic	4	Colonic	0
Extension of peritonitis	Generalized	6	Localized	0
Character of peritoneal fluid	Fecal 12 pts.	Purulent 6 pts.	Clear 0 pts	

rated), and the other with Meckel's diverticulum with inflammatory process. Of four patients with colonic pathology, all of sigmoid segment, two had volvulus, one stenosis, and the other, perforation.

With regard to spread of peritonitis, 60 patients were found with generalized peritonitis and 114, localized peritonitis; 68% of survivors (111/163) had localized peritonitis and 32% (52/163), generalized peritonitis. Among patients who died, 27% (3/11) had localized peritonitis and 73% (8/11), generalized peritonitis.

Mannheim Criteria Data

Group mean MPI score was 14 points. Among surviving patients, mean score was 13 points and among non-survivors, mean was 29 points ($p < 0.0001$).

In Figure 1, we can observe the study group life table. χ^2_{cal} was 152.875 and χ^2_{tab}, with α of 0.05 was 5.99. χ^2_{cal} is $>\chi^2_{tab}$; thus, null hypothesis is rejected, which means, survival curves of the three subgroups (<21, 21–29 and >29) have differences that are statistically significant.

Tables III and IV, succinctly, break down information of each risk factor according to the following categories: a) MPI scores <21, 21–29 and >29, and b) MPI scores of ≤26 and >26.

Mean length of stay in the group of <21 MPI was 3.71 days (SD ± 3), in the group of 21–29 MPI, 8.57 days (SD ± 6.02), and of in group of >29 MPI, 9.43 days (SD ± 8.04). Considering the reference cut-off point of 26 MPI, it was 4.40 days (SD ± 3.98) in group of ≤26 MPI and 8.59 days (SD ± 7.56) in group of >26 MPI.

In descending order, results of odds ratio (OR) for each risk factor were presence of malignancy 16.20, age over 50 years 12.86, generalized peritonitis 5.69, fecal peritoneal fluid 5.33, female gender 1.31, purulent peritoneal fluid 0.95, clear peritoneal fluid 0.75, and non-colonic origin, 0.33. It was not possible to construct the contingency table because there were no deaths without organic failure or with time elapsed of <24 h; thus, OR could not be calculated for these risk factors.

Table II. Anatomic origin or etiology of peritonitis in study group of Mannheim peritonitis index validation study at the *Hospital General de Durango*.

Diagnoses	Number of patients	%
Appendicitis	84	48.28
Abdominal trauma	34	19.54
Cholecystopathy	13	7.47
Uterus and adnexa pathology	13	7.47
Complicated hernias	8	4.60
Gastric pathology	5	2.87
Small bowel pathology	5	2.87
Idiopathic	4	2.30
Colonic pathology	4	2.30
Pancreatitis	3	1.72
Carcinomatosis	1	0.57

Discussion

A glance at the life table (cfr of Figure 1) shows a difference in prognosis of the three established intervals. There is absence of deaths in patients with scores <21 MPI points, and survival of patients with interval of 21–29 points was superior to those with 29 points, confirming the predicative value of MPI among patients with surgically diagnosed peritonitis at the HGD. Analyzing significance using the logrank method, χ^2_{cal} of 152.875 surpassed critical value of 5.99 (χ^2_{tab}), which must be equaled or surpassed, rejected the null hypothesis; considering α of 0.05, and even increasing α to 0.001, null hypothesis is still rejected. This implies that survival differences observed among three intervals (<21, 21–29, and >29 MPI points) are statistically significant ($p = <0.001$).

Mean MPI score of all patients with peritonitis studied at the HGD was 14 points. Among patients who died, mean was 29 points compared to 13 points among survivors. This difference is statistically significant ($p = <0.0001$). When consulting LILACS data base, we found a Mexican study of patients with abdominal sepsis; mean MPI was 21.5 points among survivors (range 11–32) and 30.69 points among non-survivors (range 15–39)[47]. Other foreign studies show mean MPI scores between 19 and 34 points (range 0 to 47 points)[6,15,18,21,39,42-45]. MPI among survivors and non-survivors reported is 25 and 31 MPI points, respectively[46].

In our study, overall mortality rate was 6.32%; other studies report global mortality rates from 3.9% to 54%[6,14,15,18,20,21,23,26,32,34,42,43,45,47,53].

In concordance with the life table, when MPI score increased, mortality increased, which coincides with other publications.[49] In general, patients admitted at the HGD had lower MPI scores than other studies; this may be because secondary peritonitis of any etiology were included (e.g., chemical, traumatic, infectious, etc), whereas other studies only considered patients with abdominal sepsis, whose global score was higher. In addition as will be discussed later, group mean age corresponded to a young population, which reflects upon MPI score.

In our results, 26 MPI points was a useful reference, as informed in other series. Patients with >26 points had mortality rate >40%, whereas patients having a score ≤26 MPI points did not reach a 3% mortality rate (OR 26.775)[25].

MPI is an important index for predicting patient outcome in peritonitis[54]. Various publications use more than one score

system to consider and determine which gives the best results[6,20,25,27,28,30,31,33,34,37-39,42,43,46,47,55]. Some concluded that APACHE II score, as well as MPI, correctly determine severity of intraabdominal infection and are strongly and independently associated with prognosis, but MPI has the advantage of simplicity and easy application[6]. The combination of APACHE II System and MPI provide the best scoring system appropriate for clinic and epidemiologic use[26].

Table III. Comparison of behavior of each risk factor of Mannheim peritonitis index in three intervals studied

Risk factor	<21 points 79% (137/174)		21–29 points 13% (23/174)				>29 points 8% (14/174)			
	Patients	%	Patients	%	Dead (6/23)	% (26%)	Patients	%	Dead (5/14)	% (36%)
Age >50 years	10	7.30	12	52.17	3	25	14	100	5	35.71
Age ≤50 years	127	92.70	11	47.83	3	27.27	0	0	0	0
Female	66	48.18	10	43.48	3	30	8	57.14	3	37.50
Male	71	51.82	13	56.52	3	23.08	6	42.86	2	33.33
Presence of organic failure	6	4.38	12	52.17	6	50	12	85.71	5	41.67
Absence of organic failure	131	95.62	11	47.83	0	0	2	14.29	0	0
Presence of malignancy	0	0	0	0	0	0	2	14.29	1	50
Absence of malignancy	137	100	23	100	6	26.09	12	85.71	4	33.33
Time ≥24 h	51	37.23	21	91.30	6	28.57	14	100	5	35.71
Time <24 h	86	62.77	2	8.70	0	0	0	0	0	0
Non-colonic origin	129	94.16	19	82.61	4	21.05	14	100	5	35.71
Colonic origin	8	5.84	4	17.39	2	50	0	0	0	0
Generalized peritonitis	31	22.63	19	82.61	4	21.05	10	71.43	4	40
Localized peritonitis	106	77.37	4	17.39	2	50	4	28.57	1	25
Clear peritoneal fluid	107	78.10	11	47.83	5	45.45	3	21.43	2	66.67
Purulent peritoneal fluid	29	21.17	11	47.83	1	9.09	9	64.29	2	22.22
Fecal peritoneal fluid	1	0.73	1	4.35	0	0	2	14.29	1	50

Table IV. Comparison of behavior of each risk factor of Mannheim peritonitis index in two intervals studied

Risk factor	≤26 points 90% (157/174)				>26 points 10% (17/174)			
	Patients	%	Dead (4/157)	% (2.55%)	Patients	%	Dead (7/17)	% (41%)
Age >50 years	21	13.38	2	9.52	15	88.24	6	40
Age ≤50 years	136	86.62	2	1.47	2	11.76	1	50
Female	75	47.77	2	2.67	9	52.94	4	44.44
Male	82	52.23	2	2.44	8	47.06	3	37.50
Presence of organic failure	15	9.55	4	26.67	15	88.24	7	46.67
Absence of organic failure	142	90.45	0	0	2	11.76	0	0
Presence of malignancy	0	0	0	0	2	11.76	1	50
Absence of malignancy	157	100	4	2.55	15	88.24	6	40
Time ≥24 h	69	43.95	4	5.80	17	100	7	41.18
Time <24 h	88	56.05	0	0	0	0	0	0
Non colonic origin	146	92.99	3	2.05	16	94.12	6	37.50
Colonic origin	11	7.01	1	9.09	1	5.88	1	100
Generalized peritonitis	48	30.57	3	6.25	12	70.59	5	41.67
Localized peritonitis	109	69.43	1	0.92	5	29.41	2	40
Clear peritoneal fluid	117	74.52	4	3.42	4	23.53	3	75
Purulent peritoneal fluid	38	24.20	0	0	11	64.71	3	27.27
Fecal peritoneal fluid	2	1.27	0	0	2	11.76	1	50

In the Mexican study previously cited, it was concluded that the only prognostic factor with statistic value was MPI score in patients with abdominal sepsis; however, combined with APACHE II, prognosis was more thorough, realistic, and significant[47]. Others differ, concluding that sensitivity and specificity with MPI is greater than that calculated with APACHE II[37].

When comparing risk factors of each variable indicating the presence or absence of adverse factor among survivors and non-survivors, (cfr. Figures 2 and 3), a mirror image is expected, as occurred. This means that adverse factor is low and favorable factor is high in survivors, and the contrary in non-survivors. This was clearly observed in the variables organic failure, evolution time, spread of peritonitis, age, and

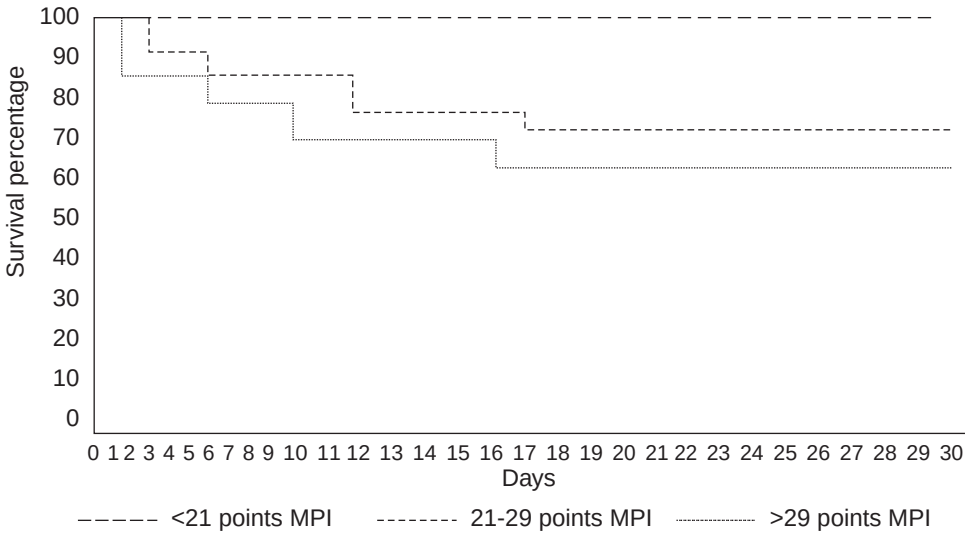

Figure 1. Life table of patients with peritonitis, operated on at the Hospital General de Durango, comparing Mannheim peritonitis index scores

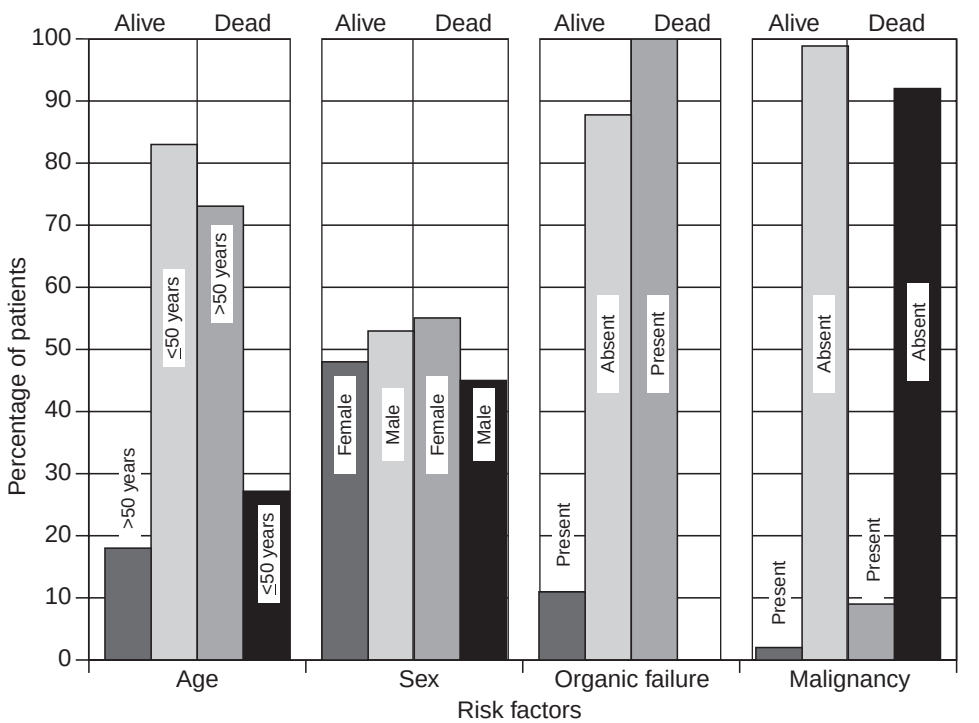

Figure 2. Comparison between survivors and non-survivors of Mannheim peritonitis index risk factors among patients with peritonitis operated on at the Hospital General de Durango.

character of peritoneal fluid. This took place, but was less evident, in presence of malignancy, while the opposite occurred with risk factor, colonic origin.

When considering each risk factor, constructing a contingency table in which presence or absence of adverse factor and result (death or survival) are considered, OR value obtained allows us to weigh, in descending order of significance, each risk factor as follows: presence of malignity; age 50 years; generalized peritonitis; presence of fecal peritoneal fluid, and female gender. Contrary to what was expected, in this study colonic origin was not an adverse factor.

Even though mortality rate in presence of malignity was 50% (1/2), the result was not conclusive due to the small number of patients with malignancy in this series.

Mean age of patients was 34.6 years old (range 16–93 years), and mean age of survivors was 32.7 years (SD ± 16.64); among non-survivors, mean age was 63 years (SD ± 18.94) considerably younger than other studies using MPI in which mean age was 49–66 years old (range 2 to 93 years). Although our study group excluded pediatric patients, our mean age was younger than other series that included children[6,16,26,28,42,43,53]. This can be due to difference in population pyramids, Mexico corresponding to a young population. In the study by Rodríguez GH et al. in Mexico City, mean age of survivors was 39.4 years (range of 19–75 years) and 47.5 years among patients who died (range of 19–85 years), similar to our results[47]. In European studies, average age was 60 years among non-survivors (37–81 years) and 56 years among survivors (21–81 years)[26].

Other studies confirmed age as a decisive factor related with mortality; one study showed that patients with <70 years of age had a mortality rate of 17.2% compared to 37.7% mortality in patients <70 years[6]. Other publications indicate that deaths occurred in patients with mean age of 80 years (63–93 years)[43]. Further studies must be undertaken to establish critical values related with age and intervals into which this variable may be subdivided, for possible modifications of MPI, sacrificing simplicity to obtain better accuracy.

In other studies, patients with generalized peritonitis corresponded to 30–66%; in our study, generalized peritonitis corresponded to 34%[6,24,43,49]. As expected, extension of peritoneal inflammatory process was related with mortality rate. Among survivors, local peritonitis was found more frequently than generalized peritonitis (68% vs 32%), while in non-survivors, the relationship between localized peritonitis and generalized peritonitis was inverted (27% vs 73%). Mean MPI score among 114 patients with localized peritonitis was 11.85 MPI, with 3% mortality rate (3/114), whereas, 60 patients with generalized peritonitis had mean score of 19.68, with mortality of 13% (8/60). Reported mortality rate among patients with local peritonitis was 8.8% and with generalized peritonitis, 14 to 28%[6,18,21]. Mean MPI score reported in localized peritonitis 19 (range 0 to 35) and in generalized peritonitis, 26 to 27 points (range 11 to 43)[18,21,22]. Survival outcome in this study was high, but MPI

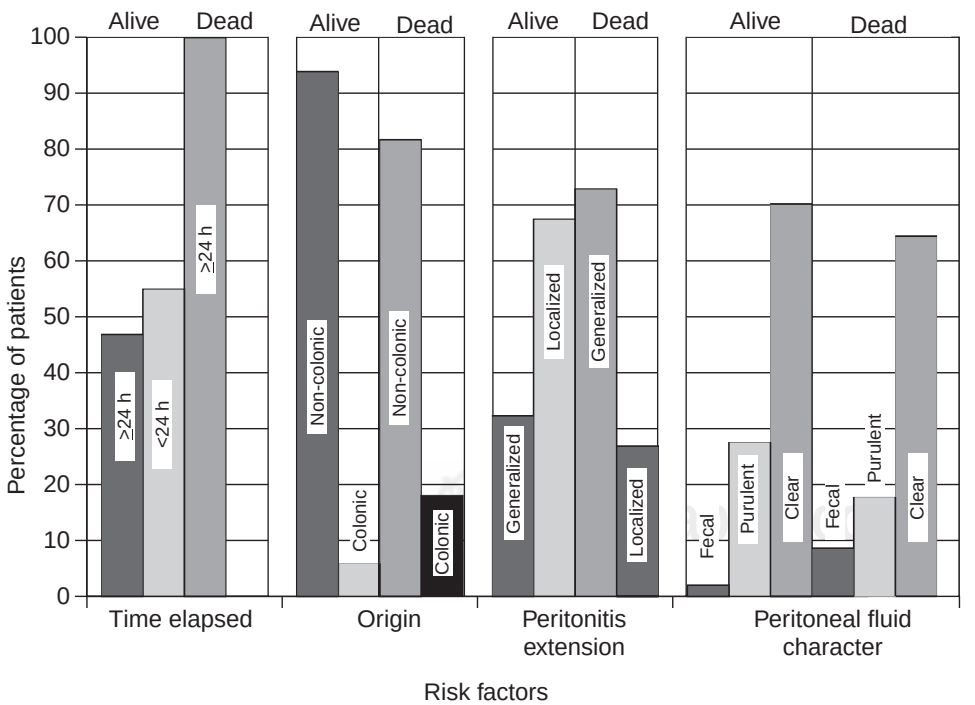

Figure 3. Comparison between survivors and non-survivors of Mannheim peritonitis index risk factors among patients with peritonitis operated on at the Hospital General de Durango.

score, both in localized and generalized peritonitis, were lower than international average MPI scores reported. This may be due to the youth of the study group. If we add an additional five points score that corresponds to an age over 50 years, MPI score obtained by our group would be closer to MPI scores reported in the literature.

Considering survival related with character of peritoneal fluid, we found the following gradient: clear fluid had mortality rate of 5.8% (7/121), purulent fluid had mortality rate of 6.3% (3/49), and fecal fluid had mortality of 25% (1/4).

The influence of variable gender in this study coincided with that reported previously, but was of little importance. Approximately 48% of patients were female and 52% male, with mortality rate of 7% (6/84) and 6% (5/90), respectively. Gender composition cited in other publications showed similar percentages, varying from 43 to 52% females and 48 to 57% male[6,28,43,49], except for one study, in which females corresponded to 37% and males to 63%[53].

As mentioned previously, it was not possible to calculate OR for variables organic failure nor evolution time of >24 h because all deaths presented these adverse factors. Another study confirmed that cause of death was multiple organ failure (MOF) in all cases of abdominal sepsis, which supported our findings related with this risk factor.[42].

Non-colonic origin is also considered an adverse factor, not confirmed in this study in which 6% of deceased (9/162) had non-colonic origin while mortality rate of 15.38% (2/12) was found patients with colonic origin.

As to influence of anatomic origin or etiology of peritonitis on prognosis independently of patient MPI score, the following was observed: While some etiologies, such as appendicitis, gynecologic pathology, abdominal trauma, and carcinosis had no deaths even though they had high MPI scores, other etiologies such as pancreatitis, small bowel pathology, complicated hernias, colonic pathology, gastric diseases, and cholecystopathies had mortalities of 67% (2/3), 40% (2/5), 38% (3/8), 25% (1/4), 20% (1/5) and 15% (2/13), respectively. Anatomic origin of bacterial contamination and microbiologic findings are not the main predictors of patient outcome[56]. Other studies have shown that factors related to host overshadowed type and source of infection in evolution of patients with intraabdominal infection[6]. The small number of patients of each etiology studied in this series did not allow conclusive results with regard to influence of the specific origin in prognosis.

We can conclude that MPI is a useful method to determine study group outcome in patients with peritonitis, surgically evaluated, at the HGD. All MPI adverse factors, except colonic origin, behaved as expected, and the following were especially useful: presence of the organic failure; time elapsed >24 h; presence of malignity; age >50 years, and generalized extension of peritonitis. MPI, together with surgeon clinic judgment of each case, may be another possible use of this score, aiding the surgeon in making the alwais difficult decision of reintervening a patient.

References

1. Maddaus MA, Ahrenholz D, Simmons RL. Biología de la peritonitis. Surg Clin North Am 1988;2:471-485.
2. Wittmann DH, Walker AP, Condon RE. Peritonitis e infección intraabdominal. In: Schwartz SI, Shires GT, Spencer FC, Husser WC, Editors. Principios de cirugía. 6th ed. México: McGraw-Hill, Inc.; 1994.Pp.1493-1528.
3. Clinical files, Hospital records, *Hospital General de Durango, SSD.*
4. Rodea-Rosas H, Athié-Gutiérrez C, Guízar-Bermúdez C, Zaldívar-Ramírez R, Flores-Martínez J. Experiencia en el tratamiento integral de 602 pacientes con sepsis abdominal. Cir Gen 1999;21:131-35.
5. Caballero RF, Acosta R, Ortíz Villalba JO. Peritonitis agudas. In: Ferreira ML, coordinator. Primeras jornadas anuales de médicos, internos, y residentes. s.l, México: Instituto de Investigación en Ciencias de la Salud;1989.Pp.567-585.
6. Pacelli F, Battista DG, Alfieri S, Piccioni E, Sgadari A, Gui D, et al. Prognosis in intra-abdominal infections. Arch Surg 1996;131(6): 641-645.
7. Barriere SL, Lowry SF. An overview of mortality risk prediction in sepsis. Crit Care Med 1995;23(2):376-393.
8. Koperna T, Schulz F. Prognosis and treatment of peritonitis. Do we need new scoring systems? Arch Surg 1996;131:180-186.
9. Knaus WA, Wagner DP, Draper EA, Zimmerman JE, Berger M, Bastos PG, et al. The APACHE III prognostic system. Risk prediction of hospital mortality for critically ill hospitalized adults. Chest 1991;100(6):1619-1636.
10. Sawyer RG, Rosenlof LK, Adams RB, May AK, Spengler MD, Pruett TL. Peritonitis into the 1990s: changing pathogens and changing strategies in the critically ill. Am Surg 1992 Feb;58(2):82-87.
11. Lemeshow S, Teres D, Spitz Avrunin J, Pastides H. A comparison of methods to predict mortality of intensive care unit patients. Crit Care Med 1987;15(8):715-722.
12. Velásquez-Díaz G, coordinator. Lineamientos y criterios de estructura progresiva para la integración de recursos humanos en hospitales generales de 30 a 180 camas censables. s.l.: Dirección de Desarrollo de los Servicios de Salud. Dirección General de Regulación de los Servicios de Salud. Subsecretaría de Regulación y Fomento Sanitario. México: Secretaría de Salud;2000:Pp.77-83.
13. Dirección General de Estadística e Informática. Directorio de Unidades 1999. México, D.F.: Secretaría de Salud; 2000.
14. Linder MM, Wacha H, Feldmann U, Wesch G. Streifensand RA, Gundlach E. Der Mannheimer peritonitis-index. Ein instrument zur intraoperativen prognose der peritonitis. Chirurg 1987;58(2):84–92.
15. Billing A, Fröhlich D, Schildberg FW. Prediction of outcome using the Mannheim peritonitis index in 2003 patients. Br J Surg 1994;81: 209-213.
16. Zeitoun G, Laurent A, Rouffet F, Hay J, Fingerhut A, Paquet J, Peillon C, Research TF. Randomized clinical trial: multicentre, randomized clinical trial of primary *versus* secondary sigmoid resection in generalized peritonitis complicating sigmoid diverticulitis. Br J Surg 2000 Oct;87(10):1366-1374.
17. Hut'an M, Poticny V, Balaz P. Modern trends in the treatment of diffuse peritonitis. Rozhl Chir 2000 Apr;79(4):171-174.
18. Seiler CA, Brugger L, Forssmann U, Baer HU, Buchler MW. Conservative surgical treatment of diffuse peritonitis. Surgery 2000 Feb;127(2):178-184.
19. Yilmazlar T, Toker S, Zorluoglu A. Non-traumatic colorectal perforations. Int Surg 1999 Apr-Jun;84(2):155-158.

20. Bergamaschi R, Marvik R, Johnsen G, Thoresen JE, Ystgaard B, Myrvold HE. Open *vs* laparoscopic repair of perforated peptic ulcer. Surg Endosc 1999 Jul;13(7):679-682.
21. Brugger LE, Seiler CA, Mittler M, Balsiger B, Feodorovici M, Baer HU, Buchler MW. New approaches to the surgical treatment of diffuse peritonitis. Zentralbl Chir 1999;124(3):181-186.
22. Seiler CA, Brugger L, Maurer CA, Renzulli P, Buchler MW. Peritonitis in diverticulitis: the Bern concept. Zentralbl Chir 1998;123(12): 1394-1394.
23. Liverani A, Correnti SF, Paganelli MT, Antonini G, Mercati U. Mannhein index in the prognosis and treatment of acute peritonitis. Minerva Chir 1998 May;53(5):385-389.
24. Moesgaard F, Jensen LS, Christiansen PM, Thorlacius-Ussing O, Nielsen KT, Rasmussen NR, Bardram L, Nielsen HJ. The effect of ranitidine on postoperative infectious complications following emergency colorectal surgery: a randomized, placebo-controlled, double-blind trial. Inflamm Res 1998 Jan;47(1):12-17.
25. Bosscha K, Reijnders K, Hulstaert PF, Algra A, van der Werken C. Prognostic scoring systems to predict outcome in peritonitis and intra-abdominal sepsis. Br J Surg 1997 Nov;84(11):1532-1534.
26. Ohmann C, Hau T. Prognostic indices in peritonitis. Hepatogastroenterology 1997 Jul-Aug;44(16):937-946.
27. Grunau G, Heemken R, Hau T. Predictors of outcome in patients with postoperative intra-abdominal infection. Eur J Surg 1996 Aug;162(8):619-625.
28. Gotzinger P, Gebhard B, Wamser P, Sautner T, Huemer G, Fugger R. Revision of diffuse peritonitis: planned *versus* on demand. Langenbecks Arch Chir 1996;381(6):343-347.
29. Ermolov AS, Bagdat'ev VE, Chudotvortseva EV, Rozhnov AV. Evaluation of the Mannheim peritonitis index. Vestn Khir Im I I Grek 1996;155(3):22-23.
30. Burger JA, Schoffel U, Sach M, Jacobs E, Kownatzki E, von Specht BU, Farthmann EH. Effects of peritonitis exudates on chemotaxis and phagocytosis of human neutrophils. Eur J Surg 1995 Sep;161(9):647-653.
31. Schoeffel U, Jacobs E, Ruf G, Mierswa F, von Specht BU, Farthmann EH. Intraperitoneal micro-organisms and the severity of peritonitis. Eur J Surg 1995 Jul;161(7):501-508.
32. Demmel N, Maag K, Osterholzer G. The value of clinical parameters for determining the prognosis of peritonitis-validation of the Mannheim Peritonitis Index. Langenbecks Arch Chir 1994;379(3):152-158.
33. Demmel N, Muth G, Maag K, Osterholzer G. Prognostic scores in peritonitis: the Mannheim Peritonitis Index or APACHE II? Langenbecks Arch Chir 1994;379(6):347-352.
34. Nespoli A, Ravizzini C, Trivella M, Segala M. The choice of surgical procedure for peritonitis due to colonic perforation. Arch Surg 1993 Jul;128(7):814-818.
35. Ohmann C, Wittmann DH, Wacha H. Prospective evaluation of prognostic scoring systems in peritonitis. Peritonitis Study Group. Eur J Surg 1993 May;159(5):267-74.
36. Altaca G, Sayek I, Onat D, Cakmakci M, Kamiloglu S. Risk factors in perforated peptic ulcer disease: comparison of a new score system with the Mannheim Peritonitis Index. Eur J Surg 1992 Apr;158(4): 217-221.
37. Rogy M, Fugger R, Schemper M, Koss G, Schulz F. The value of 2 distinct prognosis scores in patients with peritonitis. The Mannheim peritonitis index *versus* the Apache II score. Chirurg 1990 Apr;61(4):297-300.
38. Krenzien J, Lorenz W. Scoring systems for severe intra-abdominal infections. Zentralbl Chir 1990;115(17):1065-1079.
39. Eypasch E, Spangenberger W, Bouillon B, Moser KH, Vestweber KH, Troidl H. Score systems, their importance for the intensive care patient. Langenbecks Arch Chir Suppl II Verh Dtsch Ges Chir 1989;813-817.
40. Fugger R, Rogy M, Herbst F, Schemper M, Schulz F. Validation study of the Mannheim Peritonitis Index. Chirurg 1988 Sep;59(9):598-601.
41. Schirrmacher E, Seifert J. The Mannheim peritonitis index. Its reliability for the assessment of prognosis in peritonitis patients. Fortschr Med 1988 Jul 30;106(22):454-456.
42. Sautner T, Götzinger, Redl-Wenzl EM, Dittrich K, Felfernig M, Sporn P, et al. Does reoperation for abdominal sepsis enhance the inflammatory host response? Arch Surg 1997;132:250-255.
43. Navez B, Tassetti V, Scohy JJ, Mutter D, Guiot P, Evrard S, et al. Laparoscopic management of acute peritonitis. Br J Surg 1997;85: 32-36.
44. Rodloff AC, Kujath P, Lunstedt B, Gaus W. Vergleichende untersuchung zum kosten-/effektivitatsverhaltnis einer initialen therapie mit imipenem/cilastin bei der sekundaren peritonitis. Chirurg 1998;69(10): 1093-1100.
45. Kriwanek S, Gschwntler M, Beckerhinn P, Armbruster C, Roka R. Langzeitergebnisse nach Hartmann-operationen. Chirurg 1999;70(1): 49-53.
46. Bruch HP, Woltmann A, Eckmann C. Chirurgisches management bei peritonitis und sepsis. Zentralbl Chir 1999;124(3):176-180.
47. Rodríguez-Gojón GH, Pérez-García R, Poblano-Morales M, Rodríguez-Brambilia V, Chavero-Garcia V. Factores pronósticos asociados a mortalidad en pacientes con sepsis abdominal tratados en la unidad de terapia intensiva. Cir Cir 1999;67(6):205-207.
48. Demmel N, Osterholzer G, Gunther B. Differentiated treatment strategy for peritonitis: single stage closure with drainage or open with programmed reinterventrion/lavage? Zentralbl Chir 1993;1187(7): 395-400.
49. Kriwanek S, Armbruster C, Beckerhinn P, Dittrich K. Prognostic factors for survival in colonic perforation. Int J Colorectal Dis 1994;9(3):158-162.
50. Winkeltau GJ, Bertram P, Braun J, Schumpelick V. Differentiated surgical therapy of diffuse peritonitis–a prospective study. Leber Magen Darm 1996;26(3):144-148.
51. Winkeltau GJ, Tons C, Bertram P, Schumpelick V. Stage-oriented antibiotic therapy of peritonitis. Langenbecks Arch Chir 1997;382(4 Suppl 1):S22-25.
52. Tassetti V, Valvano L, Navez B, Mutter D, Scohy JJ, Evrard S, Marescaux J. Perforated peptic ulcer and laparoscopic treatment. Minerva Chir 1998;53(10):777-780.
53. Yilmazlar T, Toker S, Zorluo glu A. Non traumatic colorectal perforations. Int Surg 1999;84(2):155-158.
54. van Laarhoven CJ, Hoofwijk AG, van Vroonhoven TJ. The Mannheim peritonitis index: a valuable method in predicting the outcome of severe peritonitis? Neth J Surg 1988;40(6):168.
55. Copes WS, Champion HR, Sacco WJ, Lawnick MM, Keast SL, Bain LW. The injury severity score revisited. J Trauma 1988;28(1):69-77.
56. Farthmann EH, Schoffel U. Epidemiology and pathophysiology of intraabdominal infections (IAI). Infection 1998;26(5):329-334.