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Criteria for admission to the third stage of damage control surgery in abdominal trauma at the General Hospital of Queretaro

Criterios para ingreso a la tercera etapa de cirugía de control de daños en trauma abdominal en el Hospital General de Querétaro

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Keywords:
damage control surgery, lethal triad, second stage.

Palabras clave:
cirugía de control de daños, triada letal, segunda etapa.

ABSTRACT

Introduction: damage control surgery refers to the rapid completion of surgery after controlling life-threatening bleeding and avoiding contamination, and then to correcting physiological abnormalities and definitive management to avoid the lethal triad. **Objective:** to determine the clinical and laboratory parameters and Intensive Care Unit stay most frequently used for admission to the third stage in damage control surgery. **Material and methods:** in a retrospective observational study, 30 files of patients who underwent damage control surgery at the General Hospital of Queretaro were analyzed, taking as variables pH, number of blood transfusions, coagulation times, temperature, Intensive Care Unit stay, and hemoglobin. **Results:** 80% male population, mean age of 43.5 years, Intensive Care Unit stay of 41 hours, mean hemoglobin of 12 g/dl when entering the third stage of damage control surgery, mean temperature of 36.56 degrees, pH of 7.33 and mean number of transfusions of 3.3 globular packets. **Conclusions:** with the present report, we place in an international panorama of our performance in the second stage of damage control surgery, in which we can undoubtedly improve to offer better results to our patients in the short, medium, and long term.

RESUMEN

Introducción: *la cirugía de control de daños se refiere a la culminación rápida de una cirugía después de controlar el sangrado que puede amenazar la vida y evitar la contaminación para luego llegar a la corrección de las anormalidades fisiológicas y manejo definitivo, encaminado a evitar la tríada letal.* ***Objetivo:*** *determinar los parámetros clínicos, laboratoriales y estancia en Unidad de Cuidados Intensivos (UCI) que se utilizarán con mayor frecuencia para el ingreso a la tercera etapa en la cirugía de control de daños.* ***Material y métodos:*** *estudio retrospectivo observacional. Se analizaron 30 expedientes de pacientes sometidos a cirugía de control de daños en el Hospital General de Querétaro, teniendo como variables: pH, número de transfusiones sanguíneas, tiempos de coagulación, temperatura, estancia en UCI y hemoglobina.* ***Resultados:*** *80% población masculina, edad media de 43.5 años, estancia en Unidad de Cuidados Intensivos de 41 horas, hemoglobina promedio de 12 g/dl al entrar a tercera etapa de cirugía de control de daños, temperatura media de 36.56 grados, pH de 7.33 y una media de transfusiones de 3.3 paquetes globulares.* ***Conclusiones:*** *con el presente estudio ubicamos en un panorama internacional nuestro actuar en la segunda etapa de cirugía de control de daños, en la que podemos mejorar indudablemente para así ofrecer mejores resultados a nuestros pacientes a corto, mediano y largo plazo.*

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INTRODUCTION

Damage control surgery refers to the rapid completion of surgery after control of life-threatening bleeding and avoidance of contamination, followed by correction of physiological abnormalities and definitive management, aimed at avoiding the lethal triad *(Figure 1)*.[1,2]

The term "damage control", used in the naval navy, means "the ability of a military ship to absorb damage and continue with

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the mission", hence the term damage control surgery and damage control resuscitation.[3,4]

It has been shown that combining both methods of damage control results in an approximate survival of 60 to 90% compared to using these methods separately at 58%. Feliciano also demonstrated a 90% survival in his research series.[5,6]

Traditional surgical dogma dictates that an operation be completed definitively, regardless of the patient's physiological condition. In wartime, battlefield casualties suffered exsanguinating injuries and underwent amputations for over 100 years, causing one-third of the reported 6 million trauma deaths annually.[7,8]

Pringle described hepatic hilum compression for severe liver injury, digital compression of the portal triad, and packing to stop massive hemorrhage in 1908. Halsted modified this technique by placing rubber sheets to pack and protect the hepatic parenchyma; these perihepatic packings have been reported since the 1970s and 1980s.[9,10]

Lucas and Ledgerwood, in 1976, at Detroit Hospital, reported three packings in 637 liver lesions. Feliciano reported in the 1980s 90% survival in 10 patients with severe liver lesions that were packed.[3]

Historical background

The modern concept of abbreviated laparotomy was described by Stone in 1983, and the term "damage control" was coined in 1993 by Schwab in Philadelphia. It refers to rapid initial control of hemorrhage and contamination, temporary abdominal closure, Intensive Care Unit (ICU) resuscitation, and subsequent re-laparotomy with definitive repair.[3,7] It was a term popularized by Rotondo in 1990. A modern review by Shapiro identified that this technique had been used in 1,000 patients with abdominal trauma.[11]

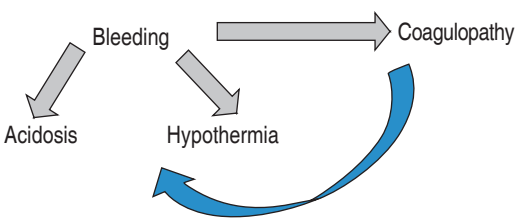

Figure 1: *Vicious circle of the lethal triad.*

The 1970s and 1980s also saw the widespread use of ICUs based on clinical resuscitation managing acute respiratory distress syndrome (ARDS), systemic inflammatory response syndrome (SIRS), and multiple organ failure (MOF) avoiding early death during post-surgery, an alternative sought by trauma surgeons in the 1980s identifying the lethal triad, and the definitive definition of damage control surgery as it is known today. The management of patients with an injury severity score of at least 30 points with orthodox surgery carried a mortality of over 70%. It was with damage control surgery (DCS) in the 1990s that mortality was reduced to 58-67%, to 33% in 2001, and 10-27% by 2006.[7,10]

Baker, in 1974 created a method to describe the patient with multiple injuries and evaluate the emergency, called ISS (Injury Severity Score). The score is the sum of the highest scores of the three most affected body regions, obtaining a critical index. Tornetta highlights the importance of the ISS score as a prognostic factor for mortality; a score of 0 to 18 represents less than 5% mortality. From 19 to 30, the mortality is 30%, and more than 30 points 45%.[1]

Pathophysiology

The presence of coagulopathy, hypothermia, and metabolic acidosis, the "deadly triad", was first described by Burch in 1992.[12] In a severely injured patient, it carries a mortality risk of up to 90%.[7] Uncontrolled bleeding and iatrogenic intravenous therapy result in the development of the lethal triad, leading to a vicious cycle that rapidly triggers death.[13]

The presence of coagulopathy is associated with a 28-46% mortality.[12] Intense and rapid resuscitation with intravenous fluids leads to dilution of coagulation factors, which affects the coagulation cascade and may cause consumption of coagulation factors, triggering disseminated intravascular coagulation (DIC). Intravascular hydrostatic pressure increases, displacing fluid, platelets,

and blood into the extravascular space, aggravating DIC. An imbalance between thromboxane and prostacyclin leads to dysfunction of the fibrinolytic system and platelet dysfunction.[14]

Hypothermia is a body temperature of 35 ºC or less for more than four hours[15] and can result in hypotension, cardiac arrhythmias, and hematologic, respiratory, renal, and endocrine disturbances. It is secondary to fluid loss from trauma, intense resuscitation with intravenous fluids and total body exposure as defined by the ATLS detailed review. It also occurs in surgery with extensive incisions that cause evaporation of intraperitoneal fluid from exposed surfaces.[16,17]

Hypothermia is a phenomenon observed in more than two-thirds of trauma patients. Its effect on mortality was first observed in cases with ruptured abdominal aortic aneurysm, being 91-100% in patients with a temperature below 32 ºC and 40-60% in those with a temperature of 32-35 ºC.[7] Mortality increases by 10% if the temperature falls below 34º C.[18] The main effects of hypothermia are:

1. Decreased cardiac output.
2. Decreased heart rate.
3. Increased peripheral vascular resistance.
4. Arrhythmias such as sinus bradycardia.
5. Decreased glomerular filtration rate.
6. Decreased Na+ absorption in renal tubules.
7. Depression of the central nervous system (CNS).
8. Decreased fibrinolytic activity (coagulopathy).

Acidosis is an indicator of tissue hypoxia produced by ischemia and necrosis.[14] Acidosis is metabolic and occurs due to lactate production and anaerobic metabolism. If the acid-base defect is not corrected in at least 48 hours, mortality is as high as 86-100%.[19] By driving anaerobic metabolism and the synthesis of lactic acid and other cytotoxic substances, increased lactate correlates with injury severity, and even base deficit can be used as a marker of injury severity and a predictor of transfusion requirements. Aortic clamping, vasopressors, massive transfusions, impaired myocardial performance, and resuscitation with large volumes of crystalloids may exacerbate metabolic acidosis in the shock state. If lactic metabolic acidosis is not corrected, it has a 45-67% mortality rate.[20]

The organic effects are listed below:

1. Decreased myocardial contractility.
2. Decreased inotropism.
3. Decreased response to catecholamines.
4. Ventricular arrhythmias.
5. Increased intracranial pressure (ICP).
6. Prolongation of partial thromboplastin time (PTT).
7. Decreased activity of factor V of coagulation.

Indications for damage control surgery

A. Physiological critical factors: demonstrated hypothermia, demonstrated acidosis, with base deficit > 8, coagulopathy demonstrated by PT (prothrombin time) lengthening, thrombocytopenia, massive transfusion requirements (more than ten globular units), time to repair exceeding 90 minutes, hemodynamic instability, with frank data of tissue hypoperfusion.[21]
B. Complex associated injuries to the primary trauma: high energy blunt trauma with thoracic involvement, multiple penetrating chest injuries, severe abdominal trauma, with major vascular trauma in the same patient.[22]
C. Other considerations: lesions that can be repaired more effectively, such as with angiographic embolization, elderly patients, or those with other comorbidities.[23-25]

Damage control surgery approach strategy

The initial management of trauma patients is based on the principles of the ATLS course. A systematic patient assessment focused on treating life-threatening injuries leads to patients who present with surgically correctable injuries being taken to the operating room immediately.[26] The selection of patients who would benefit from damage control surgery is based on a large constellation of injuries as well as

the physical condition of the patient, the best candidates being those with extensive injuries requiring long operative time, hemodynamic instability, and significant exsanguinating injuries.[27,28]

The strategy of the method for damage control surgery is divided into several stages. Mainly three stages are described; some authors describe four and even five stages, taking the convalescence and the definitive reconstruction of the abdominal wall,[29] as this last stage of the strategy.

Stage I

This stage goes from pre-hospital management, also called stage 0, until the patient is admitted to the operating room and the decision is made to perform damage control surgery, and this is concluded.[30] The abdominal injuries that most frequently require management with damage control surgery are severe hepatic injuries up to 83%, being more common grade III injury[31] and splenic and renal injuries. For injuries that can be repaired, the Pringle maneuver is indicated for up to 60 minutes without representing parenchymal ischemia that affects liver function.[32] The primary method for complex abdominal liver injuries is packing.[33,34] Packing the liver is performed using a laparotomy and placing compresses at the site of origin of the hemorrhage when the retro hepatic vena cava is injured; packing is performed anteriorly by compressing the vena cava completely. Other parenchymal injuries require anterior and posterior packing; the goal is to buffer the bleeding site without suppressing the blood flow of the hepatic parenchyma. Plastic sheets can be placed over the parenchyma and then packed with compresses to avoid removing clots when the packing is removed.[35,36] Packing is the most commonly used method in the management of significant liver injuries; the indications for packing are the treatment of the liver injury due to the extent of other intra-abdominal injuries, the presence of coagulopathy related to deep shock, or the "irreparable" nature of the liver injury.[26,37,38] Judicious use of packing in highly selected patients provides 60-90% survival. Feliciano demonstrated 90% survival in the 1980s in liver packing.[39] Hepatic hemorrhage may persist in case of misapplication of compresses around the liver or due to irregularities in the wound (as occur in blunt trauma). In addition, packing is associated with some complications, such as the development of biliary fistulas, biliomas, and hepatic abscesses.[16,20]

Once the hemorrhage and peritoneal contamination have been controlled, the abdomen should be temporarily closed. For this, field clamps, mesh, plastic bags, the "Bogotá bag", aponeurosis closure, plastic or silicone sheets and vacuum packing, and Velcro-glued sheets, which provide a tension-free and impermeable cover of the abdominal contents to prevent fluid loss and evisceration, can be used.[16]

Stage II

The second stage, also called resuscitation, goes from the conclusion of the surgical event and the patient's admission to the ICU for physiological stabilization until the decision to perform the definitive laparotomy. Today this stage is also known as damage control resuscitation.[20]

The first measure of physiological correction should be the recovery of body temperature in an insulated room with constant temperature, warm solutions, and warm or thermal covers that maintain the heat.[40]

Acidosis must then be corrected, achieved by improving oxygen demand and ensuring tissue perfusion, the determinants of tissue perfusion being cardiac output, hemoglobin, and arterial blood oxygen saturation. Therefore, resuscitation with blood products is ideal, avoiding hyperchloremic acidosis in this type of patient, which increases mortality. Central venous pressure monitoring is the best parameter to assess whether resuscitation is adequate.[39-41]

Adequate resuscitation up to this point helps in the correction of coagulopathy, accompanied by resuscitation with fresh frozen plasmas, cryoprecipitates, and coagulation factors such as factor VII, in

which improvement in coagulopathy has been found for cases undergoing damage control surgery.[39,41]

Stage III

This stage, also known as definitive surgery, must be performed when the patient is out of the lethal triad and without risk of suffering it again, and with stable physiological vital signs, the patient may enter the operating room again for the definitive repair of the lesions and the definitive closure of the abdominal wall, preferably between 24 and 36 hours. This stage does not have a standard time to be performed; however, it is recommended to stay within 72 hours.[39,41] It has been reported that mortality increases by performing it in the first hours after the first surgery. During the definitive procedure, the revision is completed in search of lesions that could have gone unnoticed in the first surgery, the packing is removed, and bleeding sites and the definitive closure of the aponeurosis are controlled.[42]

Stage IV

The concept of delayed closure of the abdominal wall is credited to Stone and collaborators, in 1981, who carried out a study among 167 patients, with an approximate mortality of 85% in those patients whose abdomen was closed under tension, compared to 22% only in those in whom delayed closure was decided.[3] Some modern reviews already describe stage IV resuscitation after definitive surgery and describe stage V as definitive closure; this depends on the literature reviewed and the use in each hospital center.[43]

Complications

The main complications depend on the injury site and the type of repair performed, or systemic complications derived from hemorrhage, massive resuscitation, or local or intra-abdominal infections, up to compartment syndrome.[44] A more significant number of complications and worse postoperative prognosis have been described in morbidly obese patients with body mass index (BMI) > 40.[45]

A review by Rotondo identified an overall mortality of up to 50% and morbidity of 40% in 961 patients undergoing damage control surgery. These reports point to the improved survival of patients with abbreviated surgery compared to a conventional procedure.[46]

Increased mortality was identified in other serious injuries associated with abdominal trauma.[47]

Adequate resuscitation in the first 15 minutes is a risk factor that predicts survival in cases undergoing damage control surgery.[10]

The presence of the lethal triad was associated with increased mortality regardless of the type of trauma.[48]

The abdominal compartment syndrome described by Richardsson in 1976 results from a persistent increase in intra-abdominal pressure (IAP) and can be of two types: primary (caused by abdominal injuries) and secondary (without intraperitoneal injuries). Abdominal trauma is the most frequent cause of primary abdominal compartment syndrome (ACS), mainly if a damage control laparotomy is performed. Factors predisposing these patients to increased IAP are abdominal packing, bleeding from coagulopathy, bowel edema from massive fluid resuscitation, increased bowel volume from mesenteric vascular injury, closure of the aponeurosis and skin under tension, and extensive contamination resulting in abdominal ileus and distention.[49] The incidence of ACS in severe trauma is 14-33% of trauma patients admitted to the ICU.[7,10]

Once ACS has developed, the associated mortality ranges from 63-72%.[7]

"The success of damage control surgery depends on the disciplined approach, which includes surgeon decisions, quick control, and determination."

MATERIAL AND METHODS

A descriptive, retrospective, and observational study that covered the period from January 01, 2015, to June 01, 2018, was done. The group of patients included all those admitted to the General Hospital of Queretaro for intensive care for damage control surgery; the clinical

records of patients were analyzed by collecting the information in data collection tables, and the number of patients who met the following inclusion criteria was determined: patients with clinical records and who had entered intensive care to continue with the third stage of damage control surgery and had completed the surgery.

After determining the number of patients who met the inclusion criteria, the records were carefully reviewed to determine which patients did not meet the criteria 100% by excluding the following:

1. Patients with incomplete registration.
2. Other eventual diseases, such as diabetes and arterial hypertension, affecting all injured patients.
3. Patients under 16 and over 60 years of age.

The total number of variables were identified and analyzed: pH, which is a measure of acidity or alkalinity of a solution, being a numerical quantitative variable; blood transfusions, which are the number of erythrocyte concentrates administered, being a numerical quantitative variable; clotting times, which refers to the time in which clotting is obtained in blood drawn from patients, and is a numerical quantitative; temperature is a measure of heat within a body expressed in degrees, and it is a numerical quantitative variable; Intensive Care Unit stay, which refers to the period in which a patient remains in the Intensive Care Unit, and it is a numerical quantitative variable; hemoglobin, amount of hemoprotein found in the body expressed in g/dl, and it is a numerical quantitative variable.

The information was compiled in data collection tables, and the results were then entered into a database in MS Excel and analyzed using the IBM SSPS 20.0 statistical program to obtain the variables to be considered, representing the results of the research using graphs and correlation tables, in addition to their description in the text.

Descriptive measures of central tendency, such as mode, mean, and median, were performed. Statistical analysis of the results in percentages and averages was performed to determine the frequency.

Descriptive statistics were performed to facilitate the information's management, organization, and analysis.

RESULTS

In the present investigation, 30 patients were analyzed (data collected from clinical records) during the period from January 01, 2015, to June 01, 2018, who underwent damage control surgery at the General Hospital of Queretaro. The following variables were analyzed: pH, number of transfusions, temperature, Intensive Care Unit stay, coagulation time, and hemoglobin. Likewise, data prior to admission to the Intensive Care Unit and before surgery were analyzed, such as hemoglobin levels, temperature, pH, and coagulation times, which yielded the following results throughout the research.

Of the 30 patients analyzed, we found that the gender distribution was 80% male and 20% female.

For the age distribution of the patients who underwent damage control surgery, the mean age was 43.5 years, with a mode of 48 years, as shown in *Figure 2*.

The results show the time in hours the patients were in the Intensive Care Unit, reporting a mean of 41 hours, a mode of 48 hours, and a median of 40 hours.

For hemoglobin levels in patients undergoing damage control surgery, two measurements were taken from the records, one prior to admission to the Intensive Care Unit with a mean of 9.1 mg/dl and mode of 10 mg/dl, and another measurement prior to the second surgical time or admission to the third stage of damage control surgery, finding a mode of 11.8 mg/dl and a mean of 12 mg/dl as shown in *Figure 3*.

Figure 4 shows the coagulation times measured according to the INR (international normalized ratio), of which two measurements were taken, prior to admission to the Intensive Care Unit, with a mean of 1.42 and a mode of 1.3, and prior to the third stage of damage control surgery, with a mean of 1.31 and a mode of 1.3.

For temperature, two measurements were taken prior to admission to the Intensive Care Unit, which gave a mean of 35.8 degrees Celsius and a mode of 36 degrees Celsius, and another measurement prior to admission to the third stage of damage control surgery with a mode of 36.8 degrees Celsius, a mean of 36.5 degrees Celsius and a median of 36.6 degrees Celsius, as shown in *Figure 5*.

In *Figure 6*, we can find the pH with which the patients arrived at the Intensive Care Unit with a mode of 7.2 and a mean of 7.1, and the values obtained prior to admission to the third stage of damage control surgery with a mode of 7.3 and a mean of 7.33.

For the number of blood transfusions, a mean of two units of erythrocyte concentrates and one unit of plasma was observed, with a mode of 3.3 units of erythrocyte concentrates and 2.1 units of plasma, as shown in *Figure 7*.

DISCUSSION

The international literature mentions a high incidence in subjects under 30 years of age, mainly males,[7,50,51] data that partially coincide with the study results in the population group studied, with a mean age of 43.5 years and a mode of age of 48. The most affected age group was observed between 40 and 50 years. Regarding gender, male patients were more affected, with 24 cases representing 80%, and the female gender with six cases corresponding to 20%. This population is highly productive; it should be noted that patients under 16 years of age were eliminated from this population because they are not patients who are routinely admitted to our unit and patients over 60 years of age due to the high frequency of comorbidities.

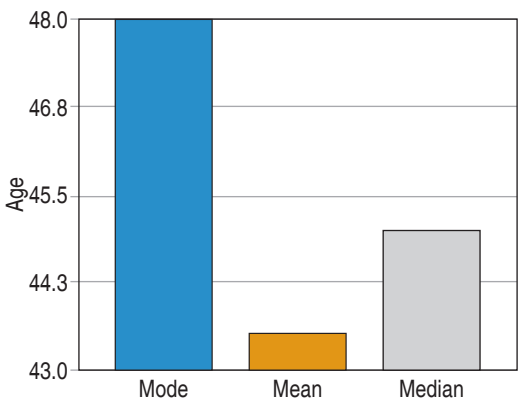

Figure 2: *Age distribution of patients undergoing damage control surgery at the General Hospital of Queretaro.*

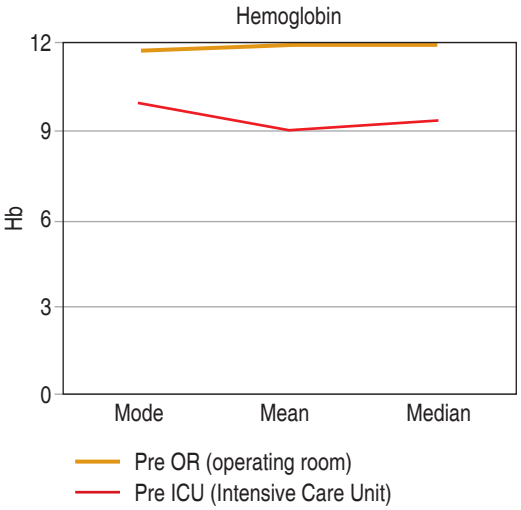

Figure 3: *Hemoglobin levels in patients before admission to the Intensive Care Unit and before the second surgical time, undergoing damage control surgery at the General Hospital of Queretaro.*

Stage III of damage control surgery, also known as definitive surgery, i.e., where the unpacking and final repair is carried out if necessary, which should be performed when the patient is out of the lethal triad and without risk of suffering it again, as well as to perform the definitive closure of the abdominal wall, preferably between 24 and 36 hours[50] this stage does not have a standard time to be performed. However, it is recommended to stay within 72 hours for its realization.[52-54] Concerning this variable is where a mean of 41 hours is obtained to enter the third stage of damage control surgery and a mode of 48 hours, entering international ranges and recommended as the authors mentioned above, compared with Latin American publications is below

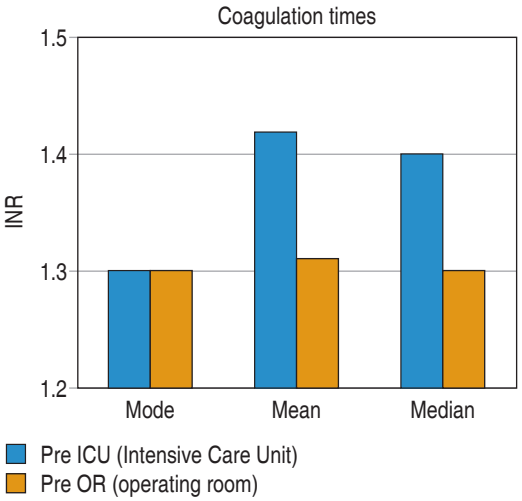

Figure 4: *Coagulation times in patients before admission to the Intensive Care Unit and before the second surgical time, undergoing damage control surgery at the General Hospital of Queretaro.*
INR = international normalized ratio.

the average time of 72 hours (measure that is standardized for entry to the third stage of damage control surgery)[3,7] but when comparing with Canadian and European publications where the length of stay in the Intensive Care Unit is reduced from 12-24 hours, a considerable gap of hours is reflected for the recovery of the patient from the lethal triad.

Coagulopathy is related to a mortality of 28-46%.[1,2] Concerning this point, we found that the patients presented a recovery for the INR (international normalized ratio) with a mean of 1.4 on admission to the Intensive Care Unit and recovery to a mean of 1.3; the same event was observed with the temperature which had a correction since on admission to the Intensive Care Unit it had a mean of 35.8 degrees centigrade and was modified to achieve a mean of 36.5 degrees centigrade. The pH correction benefitted favorably, achieving an average of 7.33 to 7.1, the value with which they entered the Intensive Care Unit. With this, the correction of the lethal triad is seen as the main objective of the second stage of damage control surgery, since with this, we avoid reaching mortality of 90% once this is established and without the possibility of recovery of these parameters.[7]

Concerning the number of transfusions required in the Intensive Care Unit, we have a mean of two red blood cell packs and a mode of 3.3 red blood cell packs to contribute to the correction of the lethal triad, which is not significant because the number of units administered since admission to the emergency department was not counted.

CONCLUSIONS

The action in damage control surgery, specifically in the second stage, consists of the management of the lethal triad in the Intensive Care Unit, where the interventions are aimed at preparing the patient for a definitive but safe intervention for the patient. It is concluded that the indispensable requirements for correcting acidosis, coagulopathy, and hypothermia are met, thus undoubtedly improving the morbimortality of these patients in the short, medium, and long term. Nevertheless, it is also worth mentioning that the stay in this unit is considerably shorter, thus avoiding the possibility of infections both associated

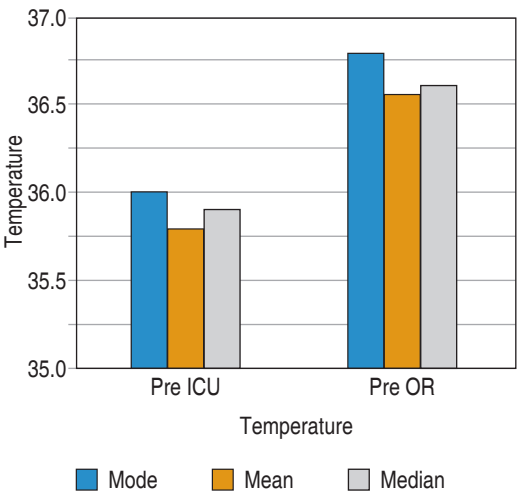

Figure 5: *Temperature in patients before admission to the Intensive Care Unit and before the second surgical time, undergoing damage control surgery at the General Hospital of Queretaro.*

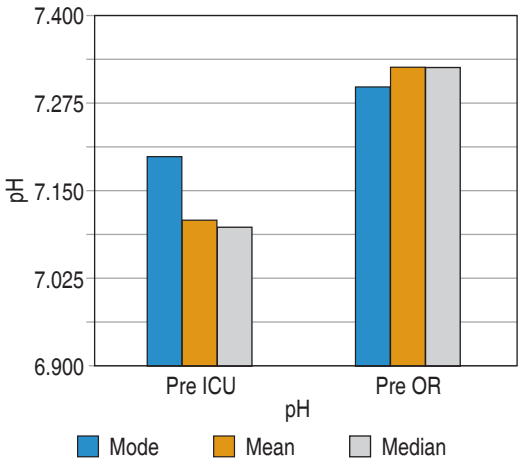

Figure 6: *pH levels in patients before admission to the Intensive Care Unit and before the second surgical time, undergoing damage control surgery at the General Hospital of Queretaro.*

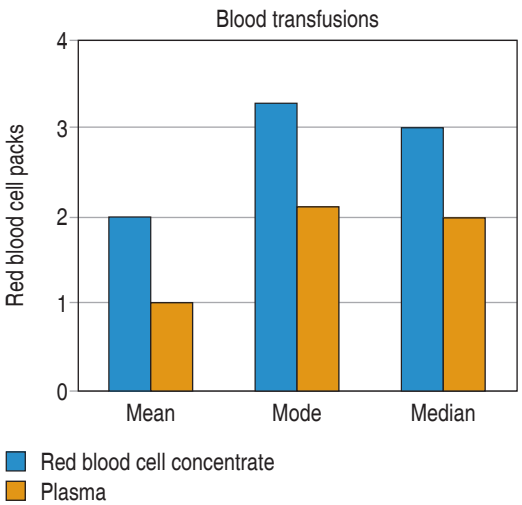

Figure 7: *Number of red blood cell packs transfused to patients undergoing damage control surgery at the General Hospital of Queretaro.*

with mechanical ventilation that these patients require and intra-abdominal and the characteristics of the tissues at the time of definitive surgery, being this study an indicator to alert in possible improvements in our performance in damage control surgery for better results to our patients. This is to optimize times in care and strategies for correcting the lethal triad in trauma patients.

REFERENCES

1. Barbieri P, Pratesi P, Navarro G. The philosophy of "damage control" in trauma and critically ill patients. 2007; 65: 203-212.
2. Soto GS, Oettinger WR, Brousse MJ, Sanchez CG. Current approach to trauma. Cuadernos de Cirugía. 2003; 17: 95-102.
3. Ball CG. Damage control resuscitation: history, theory, and technique. Can J Surg. 2014; 57: 55-60.
4. Gonclaves R, Saad RJ. Thoracic damage control surgery. Rev Col Bras Cir. 2016; 43: 374-381.
5. Espinoza R. Damage control surgery for abdominal trauma. Its fundamentals, when, and how? Rev Chil Surgery. 2004; 56: 204-209.
6. Kushimoto S, Miyauchi M, Yokota H, Kawai M. Damage control surgery and open abdominal management: recent advances and our approach. J Nippon Med Sch. 2009; 76: 280-290.
7. Camacho AJF, Mascareño JS. Damage control surgery?: a review. Gac Med Mex. 2013; 149: 61-72.
8. Gómez HMM, Morales WMM, González OJM, López CZC. Damage control surgery. Rev Cubana Cir. 2006; 45.
9. Dormoi G, Ávila AA. Closed hepatic trauma: case report of damage control surgery. Revista Médico Científica. 2003; 16: 90-93.
10. Kapan M, Onder A, Oguz A, et al. The effective risk factors on mortality in patients undergoing damage control surgery. Eur Rev Med Pharmacol Sci. 2013; 17: 1681-1687.
11. Chaudhry R, Tiwari GL, Singh Y. Damage control surgery for abdominal trauma. Med J Armed Forces India. 2006; 62: 259-262.
12. French RL. Control of haemorrhage and damage control surgery. Surgery. Elsevier Ltd. 2016; 1-7.
13. Curry N, Davis PW. What's new in resuscitation strategies for the patient with multiple trauma? Injury. 2012; 43: 1021-1028.
14. Holcomb JB, Tilley BC, Baraniuk S, et al. Transfusion of plasma, platelets, and red blood cells in a 1:1:1 vs a 1:1:2 ratio and mortality in patients with severe trauma the PROPPR randomized clinical trial. JAMA. 2015; 313: 471-82.
15. Wu D, Zhou X, Ye L, Gan J, Zhang M. Emergency department crowding and the performance of damage control resuscitation in major trauma patients with hemorrhagic shock. Acad Emerg Med. 2015; 22: 915-921.
16. Sharrock AE, Midwinter M. Damage control-trauma care in the first hour and beyond: a clinical review of relevant developments in the field of trauma care. Ann R Coll Surg Engl. 2013; 95: 177-183.
17. Midwinter MJ, Woolley T. Resuscitation and coagulation in the severely injured trauma patient. Philos Trans R Soc B Biol Sci. 2011; 366: 192-203.
18. Ravi PR, Puri B. Fluid resuscitation in haemorrhagic shock in combat casualties. Disaster Mil Med. 2017; 3: 2.
19. Imran JB, Tsai S, Timaran CH, Valentine RJ, Modrall JG. Damage control endografting for the unstable or unfit patient. Ann Vasc Surg. 2017; 42: 150-155.
20. Tien H, Beckett A, Garraway N, Talbot M, Pannell D, Alabbasi T. Advances in damage control resuscitation

and surgery: Implications on the organization of future military field forces. Can J Surg. 2015; 58: S91-S97.
21. Waibel BH, Rotondo MM. Damage control surgery: its evolution over the last 20 years. Rev Col Bras Cir. 2012; 39: 314-321.
22. Simmons JW, Powell MF. Acute traumatic coagulopathy: pathophysiology and resuscitation. Br J Anesth. 2016; 117: 31-43.
23. Kanat BH, Bozan MB, Emir S, Bali I, Sozen S, Dal B, et al. Damage Control Surgery. Current Problems of Emergency Abdominal Surgery. 2016 September 21.
24. Kirkpatrick AW, Tien H, LaPorta AT, Lavell K, Keillor J, Wright Beatty HE, et al. The marriage of surgical simulation and telementoring for damage-control surgical training of operational first responders: a pilot study. J Trauma Acute Care Surg. 2015; 79: 741-747.
25. Roberts DJ, Zygun DA, Kirkpatrick AW, et al. A protocol for a scoping and qualitative study to identify and evaluate indications for damage control surgery and damage control interventions in civilian trauma patients. BMJ Open. 2014; 4: e005634.
26. Roberts DJ, Jenne CN, Ball CG, Tiruta C, Léger C, Xiao Z, et al. Efficacy and safety of active negative pressure peritoneal therapy for reducing the systemic inflammatory response after damage control laparotomy (the Intra-peritoneal Vacuum Trial): study protocol for a randomized controlled trial. Trials. 2013; 14: 141.
27. Ordoñez CA, Pino LF, Badiel M, Sánchez AI, Loaiza J, Ballestas L, et al. Safety of performing a delayed anastomosis during damage control laparotomy in patients with destructive colon injuries. J Trauma. 2011; 71: 1512-1517; discussion 1517-1518.
28. Becher RD, Peitzman AB, Sperry JL, Gallaher JR, Neff LP, Sun Y, et al. Damage control operations in non-trauma patients: defining criteria for the staged rapid source control laparotomy in emergency general surgery. World J Emerg Surg. 2016; 11: 10.
29. Matsumoto H, Mashiko K, Sakamoto Y, Kutsukata N, Hara Y, Yokota H. A new look at criteria for damage control surgery. J Nippon Med Sch. 2010; 77: 13-20.
30. Enriquez-Domínguez L, Díaz-Rosales JDD, Herrera-Ramírez F, Castillo-Moreno JR. Damage control surgery. Initial experience in the General Hospital of Ciudad Juarez, Mexico, during the period from April 2008 to December 2010. Méd UIS. 2011; 24.
31. Choron RL, Hazelton JP, Hunter K, et al. Intra-abdominal packing with laparotomy pads and QuikClotTM during damage control laparotomy: a safety analysis. Injury. 2017; 48: 158-164.
32. Smith JW, Garrison RN, Matheson PJ, Franklin GA, Harbrecht BG, Richardson JD. Direct peritoneal resuscitation accelerates primary abdominal wall closure after damage control surgery. J Am Coll Surg. 2010; 210: 658-664, 664-667.
33. Zhang LY, Yao YZ, Jiang DP, et al. Surgical treatment strategy for multiple injury patients in ICU. Chin J Traumatol. 2011; 14: 42-45.
34. Liu YH, Chao CS, Chang YP, Chin HK. Hemostatic resuscitation for massive hemorrhage with warm fresh whole blood in a patient with severe blunt trauma. Asian J Surg. 2014; 37: 205-207.
35. Manterola C, Flores P, Otzen T. Floating stoma: An alternative strategy in damage control surgery. J Visc Surg. 2016; 153: 419-424.
36. Voiglio EJ, Dubuisson V, Massalou D, et al. Abbreviated laparotomy or damage control laparotomy: why, when and how to do it? J Visc Surg. 2016; 153: 13-24.
37. Laguzzi MC, Monge G, Ferla D, Sciuto PM. Damage control surgery rationale and results. Review of cases in a hospital in Uruguay over 4 years. Anales de la Facultad de Medicina. 2015; 2: 45-52.
38. Schnüriger B, Inaba K, Barmparas G, Rhee P, Putty B, Branco BC, et al. A new survivable damage control model including hypothermia, hemodilution, and liver injury. J Surg Res. 2011; 169: 99-105.
39. Ruiz C, Andresen M. Resuscitation with fluids and blood products in trauma. Rev Med Chile. 2014; 142: 758-766.
40. Sagraves SG, Toschlog EA, Rotondo MF. Damage control surgery--the intensivist's role. J Intensive Care Med. 2006; 21: 5-16.
41. Malagón AJV, Dolores J, Mendoza V, Pérez AJV, Dolores J, Villanueva Y, et al. Damage control surgery. Cir Gen. 2010; 32: 37-42.
42. Smith BP, Adams RC, Doraiswamy VA, et al. Review of abdominal damage control and open abdomens: focus on gastrointestinal complications. J Gastrointestin Liver Dis. 2010; 19: 425-435.
43. Weber DG, Bendinelli C, Balogh ZJ. Damage control surgery for abdominal emergencies. Br J Surg. 2014; 101: e109-e118.
44. Schreiber MA. The beginning of the end for damage control surgery. Br J Surg. 2012; 99: 10-11.
45. Marquez J, Blanco G, López D. Damage control surgery. Emergencias. 2012; 24: 219-224.
46. Liao LM, Fu CY, Wang SY, Liao CH, Kang SC, Ouyang CH, et al. Risk factors for late death of patients with abdominal trauma after damage control laparotomy for hemostasis. World J Emerg Surg. 2014; 9: 1.
47. González Balverde M, Ramírez Lizardo EJ, Cardona Muñoz EG, Totsuka Sutto SE, García Benavides L. Prognostic value of the lethal triad among patients with multiple trauma. Rev Med Chil. 2013; 141: 1420-1426.
48. Godat L, Kobayashi L, Costantini T, et al. Abdominal damage control surgery and reconstruction: world society of emergency surgery position paper. World J Emerg Surg. 2013; 8: 53.
49. Ghosh S, Banerjee G, Banerjee S, Chakrabarti DK. A logical approach to trauma-Damage control surgery. Indian J Surg. 2004; 66: 336-340.
50. Cirocchi R, Montedori A, Farinella E, Bonacini I, Tagliabue L, Abraha I. Damage control surgery for abdominal trauma. Cochrane Database Syst Rev. 2013; 2013(3): CD007438.
51. Mattox K. Introduction, background, and future projections of damage control surgery. Surg Clin North Am. 1997; 77: 753-759.
52. Schreiber MA. Damage control surgery. Crit Care Clin. 2004; 20: 101-118.
53. Germanos S, Gourgiotis S, Villias C, Bertucci M, Dimopoulos N, Salemis N. Damage control surgery in

the abdomen: An approach for the management of severe injured patients. Int J Surg. 2008; 6: 246-252.
54. Wang SY, Liao CH, Fu CY, Kang SC, Ouyang CH, Kuo IM, et al. An outcome prediction model for exsanguinating patients with blunt abdominal trauma after damage control laparotomy: a retrospective study. BMC Surg. 2014; 14: 24.

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