

Pyogenic liver abscess after gunshot injury: 10 years' experience at a single level 1 trauma center

O. Dandin¹ · E. J. Valle¹ · G. Pimentha¹ · C. I. Schulman¹ · U. Teomete² · K. G. Proctor¹ · N. Namias¹

Received: 22 June 2015 / Accepted: 5 September 2015 / Published online: 16 September 2015
© Royal Academy of Medicine in Ireland 2015

Abstract

Objectives Liver abscesses are approximately 50 % of all visceral abscesses, and trauma presents as a rare cause of the liver abscess. Otherwise, hepatic abscess is an uncommon complication of gunshot wound (GSW) to the liver among all trauma cases. Here we reviewed their experience in detail.

Method From January 1, 2004 to September 30, 2013, there were 2143 patients admitted to Ryder Trauma Center at Jackson Memorial Hospital/University of Miami with severe abdominal trauma: 1227 penetrating and 866 blunt. Among the patients who had penetrating trauma, 637 had GSWs and 551 had stab wounds. Thirty-nine patients had other kinds of penetrating traumas. Eleven patients were identified as having liver abscess, with 8 of them belonging to the GSW group, and 3 to the blunt injury group. The diagnosis and management of the 8 patients with a hepatic abscess after GSW to the liver were demonstrated.

Result There were seven males and one female with a mean age of 29 ± 10 years. There were one grade 2, four grade 3, two grade 4 and one grade 5 injuries. The mean abscess size was 10 ± 2 cm. The abscesses were usually caused by infection from mixed organisms. These abscesses were treated with antibiotics and drainage. No mortality and long-term morbidity were seen.

Conclusion Hepatic abscess after GSW to the liver is a rare condition, with an incidence of 1.2 %. It is usually seen in severe liver injury (grade 3 and above), but our patients were all treated successfully, with no mortality.

Keywords Trauma · Liver · Infection · Risk factors · Complications

Introduction

Pyogenic liver abscess is a rare and lethal illness with the incidence of 2,3/100,000 population per year [1]. In Western countries, pyogenic liver abscess is seen in 80 % of the patients with liver abscess. The other reasons are parasitic, mixed (bacterial superinfection of parasitic abscess) and uncommonly fungal infections [2]. Three scenarios are mentioned for liver abscess formation: agents may invade the liver by way of biliary tract and blood system or by direct extension, especially by way of gallbladder bed [2, 3]. They can occur most frequently in the presence of biliary tract infections. Also hepatobiliary surgery, radiological hepatobiliary procedures, non-biliary intraabdominal infections (appendicitis or sigmoiditis), diabetes, malignancy, denutrition and immunosuppression are the other risk factors. However, liver abscess uncommonly develops after trauma [1, 2, 4, 5]. In recent years, new advanced diagnostic and treatment options have provided management of liver abscess.

In general, liver abscesses are located in the right lobe and are solitary. Right upper abdominal pain, chills and fever are the most frequently occurring symptoms. *Klebsiella* and *Escherichia coli* are the most common bacterial agents. Enterococcus and streptococcus are the other agents [6–10]. The mortality of pyogenic liver abscess usually

✉ O. Dandin
dandinozgur@gmail.com

¹ Division of Trauma and Surgical Critical Care, Dewitt–Daughtry Family Department of Surgery, University of Miami Miller School of Medicine, Miami, FL, USA

² Department of Radiology, University of Miami Miller School of Medicine, Miami, FL, USA

depends on comorbidities and although the rate gradually reduces, it varies between 2.5 and 12.3, 6 % [3, 6, 11].

Although liver abscesses due to trauma were presented in prior studies [12, 13], these series include few cases and detailed clinical presentation of liver abscess after gunshot wound (GSW) was not described.

Materials and methods

This was a retrospective chart review with IRB approval. From January 1, 2004 to September 30, 2013, 637 patients with GSWs to the abdomen were identified from a registry of all patients admitted to the Ryder Trauma Center at Jackson Memorial Hospital/University of Miami Miller School of Medicine. A total of 8 patients was identified with a liver abscess after sustaining a GSW to the liver.

Fever was defined as a temperature higher than 37.5 °C. Tachycardia was defined as a heart rate >100 beats per minute. All other vital signs and laboratory values, which were obtained on the same day of diagnosis showed significant deviation from the patient's baseline values.

The diagnosis of liver abscess was based on evidence from imaging studies [ultrasound or computed tomography (CT)] and microbiology (blood or aspirate culture results). Liver injuries were graded according to the system proposed by Moore et al. (Table 1) [14].

The size of the abscess was defined by its greatest diameter. Clinical findings included abdominal pain, fever

with chills, nausea/vomiting, malnutrition, respiratory symptoms, hypotension and diarrhea.

Basic demographics, physical exam findings, mechanisms and severity of injury, and the extent of liver and associated injuries were all recorded. The development, diagnosis, location of the hepatic abscess, management and complications were reviewed on a standardised data sheet. Age, gender, body mass index, laboratory values, injury severity score (ISS), grade of liver injury, size of abscess, time from injury to abscess formation, length of hospital stay, length of drainage, and transfusion amount, were also collected, calculated and expressed as mean \pm standard deviation.

Results

From January 1, 2004 to September 30, 2013, there were 2093 patients admitted with severe abdominal trauma: 1227 with penetrating and 866 with blunt. In the penetrating group, there were 637 GSW, and 590 stab/other, of which 769 were operative. There was a total of 866 patients with blunt abdominal trauma, of which 172 required an operation. A total of 11 developed liver abscess. Eight patients had liver abscess after GSW and three patients had a liver abscess after blunt trauma. Only 2 patients of these 11 were managed non-operatively after their injury; one in the GSW group, and one in the blunt group. The incidence of liver abscess after GSW was 1.2 %. No liver abscess has developed after the other penetrating injuries including stab wounds. Liver abscess occurred in 0.7 and 0.3 % of penetrating and blunt trauma patients, respectively (Table 2).

Demographic characteristics, presentation and initial procedure

Eight patients (seven males, one female) were identified, with a mean age of 29 ± 10 years. BMI was 27 ± 3 , ISS was 27 ± 12 , and an average of 17 ± 24 units of PRBCs was transfused. There was one grade 2 liver injury, four grade 3 injuries, two grade 4 injuries, and one grade 5 injury. The primary liver injury was managed operatively in seven patients and non-operatively in one remaining patient. Hepatorrhaphy was performed in seven patients, packing in two, hepatic resection in one patient, and finally, hepatic artery angiogram with embolization was performed in one patient (Table 3).

Symptoms and exam findings

Abdominal pain and fever with chills were the most commonly encountered findings at presentation of the

Table 1 Liver injury scale

Grade	Injury type	Injury description
I	Hematoma	Subcapsular, <10 % surface area
	Laceration	Capsular tear, <1 cm parenchymal depth
II	Hematoma	Subcapsular, 10–50 % surface area, Intraparenchymal, <10 cm diameter
	Laceration	1–3 cm parenchymal depth, <10 cm length
III	Hematoma	Subcapsular, >50 % surface area or expanding. Ruptured subcapsular or parenchymal haematoma. Intraparenchymal haematoma >10 cm or expanding
	Laceration	>3 cm parenchymal depth
IV	Laceration	Parenchymal disruption involving 25–75 % of hepatic lobe or 1–3 Couinaud's segments in a single lobe
V	Laceration	Parenchymal disruption involving >75 % of hepatic lobe or >3 Couinaud's segments within a single lobe
	Vascular	Juxtahepatic venous injuries, i.e., retrohepatic vena cava/central major hepatic veins
VI	Vascular	Hepatic avulsion

hepatic abscess. Physical examination demonstrated tenderness and tachycardia most commonly followed by hepatomegaly, pallor and guarding/rigidity (Table 4).

Laboratory findings

Seven patients had elevated white cell counts and all had decreased serum albumin, haemoglobin, and hematocrit levels. Also serum prealbumin levels were decreased in five patients; however, these data were not available for the remaining three patients. Liver enzymes were elevated in five patients. Alkaline phosphatase levels were elevated in two patients. Only one had elevated serum urea and creatinine levels secondary to acute renal failure. Two patients were found to have elevated serum total bilirubin levels. Platelet counts were elevated in four patients. Prothrombin time was the only coagulation value was found to be elevated which occurred in six of the patients. Finally, serum fasting glucose levels were elevated in six patients (Table 5).

Imaging and treatment

All of the liver abscesses were primarily diagnosed by CT. The abscesses were all solitary and located in the right lobe of the liver in all eight patients, with one demonstrating subhepatic extension. The mean abscess size was

10 ± 2 cm. The liver abscesses were formed and diagnosed within a median of 16 ± 18 days after injury. Depending on their size and location each abscess was managed by a combination of CT-guided percutaneous drainage, antibiotics, and laparotomy. Intravenous antibiotic therapy was administered to all patients. The most commonly used antibiotic was piperacillin-tazobactam. The other antibiotics used included vancomycin, carbapenems, and fluoroquinolones in combination with metronidazole (Table 6).

Microbiology

Liver abscess aspirate culture studies were performed in six patients of whom only five reports identified a bacterial species. One abscess was caused by *Staphylococcus aureus* and the others were attributed to mixed organisms, including *Enterococcus faecium*, *Enterobacter cloacae*, *Escherichia coli*, *Streptococcus viridans*, *Enterococcus faecalis*, *Morganella morganii*, *Enterococcus cloacea*, and *Acinetobacter lwoffii*. Table 6 summarizes the bacterial composition of the abscesses.

Complications and outcomes

There were several other injuries associated with the liver GSWs, including 7 lung contusions, 6 pneumothoraces, 5

Table 2 Number of liver abscesses after abdominal trauma

	Operative	Non-operative	Total	Rate (%)
Blunt abdominal trauma	172 (2)	694 (1)	866 (3)	0.3
Penetrating abdominal trauma	769	458	1227 (8)	0.7
GSW	473 (7)	164 (1)	637 (8)	1.2
Stab wounds	277	274	551 (0)	0.0
Other	19	20	39	0.0
Total abdominal trauma	941	1152	2093 (11)	0.5

() number of patients with liver abscess

Table 3 Characteristics of eight patients with liver abscess as a complication of the gunshot hepatic trauma

Patient	Gender	Age	BMI	ISS	Management of primary liver injury	Transfusion amount (unit)	Grade of liver injury
1	F	40	25.35	25	Operative (hepatorrhaphy)	11	3
2	M	46	28.29	43	Operative (hepatorrhaphy + packing)	67	3
3	M	16	28.1	13	Operative (hepatorrhaphy)	1	2
4	M	30	25.1	26	Operative (hepatorrhaphy)	4	4
5	M	26	32.8	9	Operative (hepatorrhaphy)	4	3
6	M	24	28.29	43	Operative (hepatorrhaphy)	3	3
7	M	33	29.35	29	Operative (hepatorrhaphy +resection + packing)	39	5
8	M	18	25.35	25	Non-operative (hepatic artery angiogram with embolization)	6	4

BMI body mass index, ISS injury severity score

Table 4 Summary of symptomatology, physical examination findings and associated injuries

Symptoms	<i>n</i> (%)	Sign on examination	<i>n</i> (%)	Associated injuries	<i>n</i> (%)
Fever with chills (>37.5 °C)	8 (100)	Tachycardia	8 (100)	Lung contusion	7 (87.5)
Abdominal pain	5 (62.5)	Tenderness	6 (75)	Pneumothorax	6 (75)
Respiratory symptoms	4 (50)	Guarding/rigidity	2 (25)	Diaphragm injury and hemothorax	5 (62.5)
Nausea/vomiting, hypotension, diarrhea, malnutrition	1 (12.5)	Sign of toxemia, hepatomegaly, pallor	1 (12.5)	Rib fractures	3 (37.5)
				Spinal cord, small bowel and gallbladder injury	2 (25)
				Pneumomediastinum; kidney, stomach, pancreas, colon and vascular injury; femur, pelvis and upper extremity fractures	1 (12.5)

Table 5 Summary of laboratory findings

	Reference range	No. of patients	Mean	±SD	% of the patients outside reference range
White cell count ($\times 10^3/\mu\text{L}$)	3.8–10.8	8	18.6	±6.5	87.5
Haemoglobin (g/dL)	Male: 13.8–17.2 Female: 12–15.6	8	9.2	±1.5	100
Hematocrit (%)	Male: 41–50 Female: 35–46	8	27.6	±3.7	100
Platelet ($\times 10^9/\text{L}$)	150–450	8	455	±116	50
Urea (mg/dL)	7–30	8	21.7	±35.9	12.5
Creatinine (mg/dl)	0.7–1.4 (<1.2)	8	1.9	±2.2	12.5
Albumin (g/dL)	3.5–5	8	2.2	±0.7	100
AST (U/L)	Males: 0–37 Females: 0–31	8	258	±258.6	62.5
ALT (U/L)	Males 0–35 Females 0–20	8	278	±284	62.5
ALP (U/L)	40–120	8	118	±94	25
Total bilirubin (mg/dL)	<1.3 (0.1–1.3)	8	1.2	±1.5	25
INR	<1.5	8	1.1	±0.16	0
PT (seconds)	9–12.5	8	13.7	±2.1	75
Glucose (mg/dL)	70–99	8	123	±40	75
Prealbumin (mg/dL)	18–45	5	13.6	±4.6	100

hemothoraces, 5 diaphragmatic injuries and 3 rib fractures. Eight patients developed complications that were directly related to sepsis, liver abscess or their associated injuries (Table 4). Acute renal failure due to sepsis was demonstrated in one patient, liver abscess recurrence was seen in three patients. Liver necrosis was seen in one and intraabdominal abscess (not associated with the liver abscess) in four patients. Persistent bile fistulas were investigated by endoscopic retrograde cholangiopancreatography in two with one of those noted to have an infected hematoma, frank pus and devitalized liver par-

enchyma. Sphincterotomy and endoscopic biliary stenting were performed in both of these patients. The other complications were pyothorax, respiratory failure, open abdomen, diabetes mellitus, malnutrition, abdominal compartment syndrome and acidosis. Of note, one patient had bullet fragments in the liver and another had bullet plus bone fragments in the site of abscess.

The mean length of drainage was 69 ± 98 days. The mean length of hospital stay was 51 ± 44 days. With a mean follow-up period of 73 months, there was no long-term morbidity or mortality (Table 6).

Table 6 Characteristics of liver abscess

Patient	Days from injury to abscess formation	Site of abscess	Amount of abscess (cc)	Treatment of abscess	Abscess culture	Length of drainage (days)	Length of hospital stay (days)	Mortality
1	8	Right lobe of liver	400	CT-guided percutaneous drainage + antibiotic		62	16	No
2	44	Right lobe of liver	1000	CT-guided percutaneous drainage + antibiotic	<i>Enterococcus faecium</i> , <i>Enterobacter cloacae</i>	9	150	No
3	4	Right lobe of liver	125	CT guided percutaneous drainage (two times)	<i>S. aureus</i>	25	18	No
4	5	Right lobe of liver	200	CT guided percutaneous drainage + antibiotic		21	30	No
5	10	Right lobe and all Subhepatic region	200	Laparotomy + antibiotic	<i>Escherichia coli</i> , <i>Streptococcus viridians</i> , <i>Enterococcus faecalis</i>	43	44	No
6	5	Right lobe of liver	200	CT guided percutaneous drainage + antibiotic	No growth	33	49	No
7	47	Right lobe of liver	500	CT guided percutaneous drainage (two times) + laparotomy + antibiotic	<i>Escherichia coli</i> , <i>Morganella morgani</i>	307	68	No
8	5	Right lobe of liver	600	Laparotomy + antibiotic	<i>S. aureus</i> , <i>Enterococcus cloacae</i> , <i>Acinetobacter Iwoffii</i>	53	32	No

Discussion

In last 10 years, 11 patients who had liver abscess after liver trauma (penetrating and blunt) at our trauma center were identified. Eight of them were following GSW and three of them were after blunt abdominal trauma. This series focuses hepatic abscess following GSW to the liver. Liver abscess after gunshot injury management has been discussed in detail infrequently, mostly in sporadic reports. A summary of several studies, including case reports on liver abscess after GSWs is listed in Table 7 [15–20]. According to this summary, the incidence of liver abscess after GSW was 3 %, with a mortality of 22 %. In our study the incidence of liver abscess after GSW was 1.2 %, with no mortality.

Foreign bodies and blast effect must be considered as the reasons of infection occurrence following GSW [21, 22]. The velocity is the most important factor determining the severity of GSW. Properties of involved organ must also be examined. The extent of the damage varies depending on the quantity of collagen and elastin in the tissue. The common affected organs from blast effect are brain, liver, kidney, and spleen. However, the least affected organs are lungs, vascular structures and muscle [23–27].

Unfortunately, in our cases, the caliber and velocity of the bullet projectiles were unknown. Therefore, we cannot make any particular conclusions regarding development of liver abscess in the frame of a certain bullet caliber/velocity combination.

Intermediate targets (IT) (bone, clothing, and metals, etc.) can determine the morphology of a wound entrance and exit site with route of the bullet. Also the trajectory of the bullet can be contaminated with pieces from the IT. Textile fibers from the patient’s clothes are the most frequent contaminants and they are strong infection sources [28]. This phenomenon was observed in one patient from our review in which both bullet and bone fragments were demonstrated within the liver on CT imaging. Bacterial contamination from a bullet itself has also been reported [29].

In their study, Noyes et al. demonstrated more than two intraabdominal organ injuries or high injury scores increase the risk of developing intraabdominal abscess [30]. This was observed in our review as well. All patients had several other injures as well as a high ISS. There is a relationship between severity of liver injury and the increased abscess rate. In recent series, it was shown that while the patients with minor blunt hepatic trauma (grades 1 and 2) had no liver abscess following non-operative management, liver abscess developed in patients with at least grade 3 liver injury [5, 12]. Noyes et al. also showed

Table 7 Literature review of liver abscess after hepatic gunshot injury

Author	Year	No. of patients	Liver injury grade	No. of abscesses	Management of primary liver injury	Diagnosis modality for liver abscess	Management of abscess	Culture	Mortality
Navsaria [20]	2009	63	-	3	Non-operative	CT	US-guided percutaneous drain [1], antibiotics [2]	<i>E. coli</i> [1]	0
Losanoff [15]	2007	1	2	1	Non-operative	CT	CT-guided percutaneous drain	<i>P. aeruginosa</i>	0
Dunham [16]	1996	1	3	1	Non-operative	Laparotomy	Laparotomy	<i>C. perfringens</i>	1
Mays [17]	1971	1	-	1	Operative	Radioactive gold scintiscan	T tube reopened	-	0
Elkin [18]	1943	238	-	2	Operative	Laparotomy	Laparotomy	-	1
Lin [19]	1995	1	-	1	Operative	CT	Laparotomy	<i>S. aureus</i> [1], Group D <i>Enterococcus</i> [1]	0

in their study that for developing an intraabdominal abscess compared to patients with grades 1,2, or 3 liver injury, a sixfold increased risk was found in the patients with grade 4 or 5 injury [30]. Our review corroborates these results as only one patient suffered liver injury that was less than grade 3.

Generally, the symptoms of pyogenic liver abscesses are nonspecific and the diagnosis needs an extremely clinical suspicion. In this current study the most significant clinical signs of liver abscess were high fever with chills, upper abdominal pain, respiratory symptoms, tachycardia and localized right upper quadrant tenderness (Table 4).

Liver enzymes were abnormal in five of the patients and two patients had mild hyperbilirubinemia. There was no clinically significant hyperbilirubinemia. These results are consistent with a recent large case series. These case series also presented that the reason of hypoalbuminemia was commonly underlying septic process. This condition causes unsuccessful initial treatment and infections in critically injured patients. Nonetheless, all patients had decreased albumin and prealbumin levels [31].

In our case series, liver abscess recurrence was seen in three patients. Our management had a 38 % failure rate (3/8). Hsieh [32] described 14.3 % (3/21) recurrence rate in 21 patients with liver abscess after 674 blunt hepatic injuries. In another study [33] the overall rate of recurrent pyogenic liver abscess found 8.6 % (45/525). Both blunt and penetrating traumas increase the rate of recurrence.

Misselbeck et al. [34] found that the complications due to hepatic angioembolization (HAE) (bile leak, liver abscess, necrosis, liver failure, gallbladder necrosis) in trauma patients were frequent but the mortality was rare. In our review, one case had HAE. This patient had hepatic necrosis and an intrahepatic hematoma, however, despite this, the patient did survive.

An increased risk of liver-related complications (bilomas, bile fistulas, hepatic or subphrenic abscess, etc.) are also seen due to perihepatic packing. However, removal of the packing within 32 to 72 h after placement, reduction in recurrence of bleeding without increased complication rates mentioned above was noted by Caruso et al. [35]. We had two patients managed with perihepatic packing. Although the packs were removed in 72 h, liver abscesses still formed.

According to a recent prospective series, multiple liver abscesses are associated with arterial circulation or biliary system. They are frequently subdiaphragmatic and located in the right lobe. Otherwise, solitary abscesses mostly involve the portal circulation and they are usually idiopathic and traumatic [36]. Each of the abscesses in our review was solitary compatible with the results of this prospective case series. Moreover, they were all in the right side of the liver.

To provide resolution, drainage is required for large abscesses (>5 cm in size). Percutaneous drainage is the first option in most cases. However, under conditions of multiloculation, rupture, related intraabdominal or biliary pathology, open surgical drainage is preferred [36]. Each abscess encountered in our review was greater than 5 cm. Percutaneous drainage was used when possible, however, there were two cases where percutaneous drainage seemed ineffective and thus, open surgical drainage was performed. These cases were complicated by either biliary injuries or collections that were inaccessible by percutaneous methods.

Abscesses, less than 5 cm in diameter, can be treated with 4–6 weeks of antibiotic therapy referring to the sensitivity of the microorganism [37]. Also prophylactic antibiotics should be used in patients who have at least a grade 3 liver injury [5]. Otherwise, targeted antibiotic therapy is advised in addition to percutaneous drainage [36, 38, 39]. These principles were not applied in our review secondary to the complicated presentations of each abscess; however, they can serve as guidelines when encountering future hepatic abscesses status after liver GSW. Although many have made recommendations, there is no prospective evidence to suggest that the antibiotic treatment of these intraabdominal abscesses should be different from any other abscesses. A recent STOP-IT clinical trial demonstrated that approximately 4 days' antibiotic therapy was similar to approximately 8 days' therapy (longer course of antibiotics) for obtaining enough source control of complicated intraabdominal infection [40].

Escherichia coli is one of the most frequent microorganisms causing liver abscess [6, 36, 41]. Also, *E. coli* was only isolated in two of our patients. Interestingly, *Acinetobacter lwoffii* was found in one patient and there have been reports citing *Acinetobacter lwoffii* [42] as a predominant bacterium in the formation of liver abscess secondary to foreign bodies. Additionally, in another case report, *Morganella morganii* was isolated as a rare cause of liver abscess [43]. The prognosis of the patients can be effected by multidrug-resistant organisms [44]. Additionally, gas containing liver abscess caused by *Clostridium perfringens* can be severe and lethal [45].

Conclusion

This is the largest series of liver abscesses following hepatic GSW. In our review of 8 patients with this pathology, several modalities were used in the treatment of the injury itself and its resultant complications. Although there is no gold standard for treatment, our review demonstrates that antibiotics, percutaneous drainage, and open surgical

drainage can all play a role in achieving successful outcomes.

Acknowledgments The authors appreciate the efforts of Kenneth G. Proctor, PhD, who edited the manuscript.

Compliance with ethical standards

All authors declare that they have no conflict of interest. All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. Informed consent was not required because it is a retrospective study.

References

- Kaplan GG, Gregson DB, Laupland KB (2004) Population-based study of the epidemiology of and the risk factors for pyogenic liver abscess. *Clin Gastroenterol Hepatol* 2(11):1032–1038
- Lardiere-Deguelte S et al (2015) Hepatic abscess: diagnosis and management. *J Visc Surg*
- Mohsen AH et al (2002) Liver abscess in adults: ten years experience in a UK centre. *QJM* 95(12):797–802
- Huang CJ et al (1996) Pyogenic hepatic abscess. Changing trends over 42 years. *Ann Surg* 223(5):600–607 (**discussion 607–9**)
- Hsieh CH et al (2003) Liver abscess after non-operative management of blunt liver injury. *Langenbecks Arch Surg* 387(9–10):343–347
- Rahimian J et al (2004) Pyogenic liver abscess: recent trends in etiology and mortality. *Clin Infect Dis* 39(11):1654–1659
- Seeto RK, Rockey DC (1996) Pyogenic liver abscess. Changes in etiology, management, and outcome. *Medicine (Baltimore)* 75(2):99–113
- Bissada AA, Bateman J (1991) Pyogenic liver abscess: a 7-year experience in a large community hospital. *Hepatogastroenterology* 38(4):317–320
- Chen SC et al (2007) Comparison of *Escherichia coli* and *Klebsiella pneumoniae* liver abscesses. *Am J Med Sci* 334(2):97–105
- Sifri CD, Madoff L (2010) Infections of the liver and biliary system. In: Mandell GL, Bennett J, Dolin R (eds) *Principles and practice of infectious diseases*. Churchill Livingstone, Philadelphia, pp 1035–1044
- Wong WM et al (2002) Pyogenic liver abscess: retrospective analysis of 80 cases over a 10-year period. *J Gastroenterol Hepatol* 17(9):1001–1007
- Bender JS, Geller ER, Wilson RF (1989) Intra-abdominal sepsis following liver trauma. *J Trauma* 29(8):1140–1144 (**discussion 1144–1145**)
- Fabian TC et al (1991) Factors affecting morbidity following hepatic trauma. A prospective analysis of 482 injuries. *Ann Surg* 213(6):540–547 (**discussion 548**)
- Moore EE et al (1995) Organ injury scaling: spleen and liver (1994 revision). *J Trauma* 38(3):323–324
- Losanoff JE, Millis JM (2007) Giant necrotizing abscess of a liver transplant after gunshot injury. *Transplantation* 84(10):1373–1374
- Dunham CM, Coates S (1996) Clostridial septic shock following an isolated, hepatic gunshot wound. *Injury* 27(4):291–293
- Mays ET (1971) Complex penetrating hepatic wounds. *Ann Surg* 173(3):421–428

18. Elkin CD, Ward WC (1943) Gunshot wounds of the abdomen: a survey of 238 cases. *Ann Surg* 118(5):780–787
19. Lin SS et al (1995) Low-velocity gunshot wounds to the spine with an associated transperitoneal injury. *J Spinal Disord* 8(2):136–144
20. Navsaria PH et al (2009) Selective nonoperative management of liver gunshot injuries. *Ann Surg* 249(4):653–656
21. Karademir K, Gunhan M, Can C (2006) Effects of blast injury on kidneys in abdominal gunshot wounds. *Urology* 68(6):1160–1163
22. Symonds RP, Mackay C, Morley P (1985) The late effect of grenade fragments. *J R Army Med Corps* 131(2):68–69
23. Amato JJ et al (1970) Vascular injuries. An experimental study of high and low velocity missile wounds. *Arch Surg* 101(2):167–174
24. Amato JJ et al (1974) Temporary cavitation in high-velocity pulmonary missile injury. *Ann Thorac Surg* 18(6):565–570
25. Oehmichen M, Meissner C, Konig HG (2000) Brain injury after gunshot wounding: morphometric analysis of cell destruction caused by temporary cavitation. *J Neurotrauma* 17(2):155–162
26. Lindsey D (1980) The idolatry of velocity, or lies, damn lies, and ballistics. *J Trauma* 20(12):1068–1069
27. Santucci RA, Chang YJ (2004) Ballistics for physicians: myths about wound ballistics and gunshot injuries. *J Urol* 171(4):1408–1414
28. Vennemann B et al (2008) Textile fibres along the bullet path—experimental study on a skin-gelatine composite model. *Int J Legal Med* 122(3):213–218
29. Tian HM et al (1982) Primary bacterial contamination of wound track. *Acta Chir Scand Suppl* 508:265–269
30. Noyes LD, Doyle DJ, McSwain NE Jr (1988) Septic complications associated with the use of peritoneal drains in liver trauma. *J Trauma* 28(3):337–346
31. Pang TC et al (2011) Pyogenic liver abscess: an audit of 10 years' experience. *World J Gastroenterol* 17(12):1622–1630
32. Hsieh CH (2002) Comparison of hepatic abscess after operative and nonoperative management of isolated blunt liver trauma. *Int Surg* 87(3):178–184
33. Cheng HC et al (2008) Long-term outcome of pyogenic liver abscess: factors related with abscess recurrence. *J Clin Gastroenterol* 42(10):1110–1115
34. Misselbeck TS et al (2009) Hepatic angioembolization in trauma patients: indications and complications. *J Trauma* 67(4):769–773
35. Caruso DM et al (1999) Perihepatic packing of major liver injuries: complications and mortality. *Arch Surg* 134(9):958–962 (**discussion 962–963**)
36. Mangukiya DO et al (2012) A prospective series case study of pyogenic liver abscess: recent trends in etiology and management. *Indian J Surg* 74(5):385–390
37. Lok KH et al (2008) Pyogenic liver abscess: clinical profile, microbiological characteristics, and management in a Hong Kong hospital. *J Microbiol Immunol Infect* 41(6):483–490
38. Mohan S et al (2006) Liver abscess: a clinicopathological analysis of 82 cases. *Int Surg* 91(4):228–233
39. Ch Yu S (1997) Pyogenic liver abscess: treatment with needle aspiration. *Clin Radiol* 52(12):912–916
40. Sawyer RG et al (2015) Trial of short-course antimicrobial therapy for intraabdominal infection. *N Engl J Med* 372(21):1996–2005
41. Cerwenka H et al (2005) Treatment of patients with pyogenic liver abscess. *Chemotherapy* 51(6):366–369
42. Ku SC et al (2000) Clinical and microbiological characteristics of bacteremia caused by *Acinetobacter lwoffii*. *Eur J Clin Microbiol Infect Dis* 19(7):501–505
43. Tsai WC, Chang LK (2002) *Morganella morganii* causing solitary liver abscess complicated by pyopericardium and left pleural effusion in a nondiabetic patient. *J Microbiol Immunol Infect* 35(3):191–194
44. Kumar P et al (2013) Multidrug resistant citrobacter: an unusual cause of liver abscess. *BMJ Case Rep*
45. Oshima S et al (2013) Two cases of liver abscess caused by *Clostridium perfringens* after transcatheter arterial chemoembolization. *Gan To Kagaku Ryoho* 40(12):1795–1797