

Slobodan Savic,¹ M.D., Ph.D.; Radmila Stevanovic,² M.D., M.A.; Djordje Alempijevic,¹ M.D., Ph.D.; Stojan Petkovic,³ M.D., Ph.D.; and Ivanka Baralic,¹ M.D., Ph.D.

Medicolegal Aspects of Post-traumatic Gastroduodenal Ulcers: A Retrospective Study*

ABSTRACT: Retrospective examination of 5-year autopsy material showed the presence of posttraumatic gastroduodenal ulcers (PGDU) in 17.7% of decedents deemed to be at risk. They were more common in males (77%) and in patients aged over 50. In the majority of cases (76%) the survival period was <12 days; in 16.5% it was < 48 h. PGDU developed most commonly in victims of polytrauma and isolated craniocerebral injury, with ISS values ≥ 16 ; patients with spinal cord injuries were at greatest risk. Most frequently affected was the stomach, exhibiting numerous, usually superficial lesions, while solitary acute and exacerbated chronic peptic ulcers were more common in the duodenum. Complications of PGDU developed in 40% of cases, mostly in the form of hemorrhage; in 20% of cases PGDU have contributed to death. Medicolegal aspects of PGDU are, most frequently, concerned with the causal relationship between trauma, PGDU, and fatal outcome, as well as the potential for allegations of medical negligence.

KEYWORDS: forensic science, forensic pathology, stress ulcers, gastroduodenal ulcers, posttraumatic complication, gastrointestinal hemorrhage, medicolegal aspects

Acute gastrointestinal ulcers can develop on the background of alcohol abuse, use of drugs such as antinflammatory substances, extensive surgery, severe diseases (e.g., those accompanied with shock and increased intracranial pressure, acute pancreatitis, peritonitis, sepsis, uraemia, hepatic failure, etc.), as well as various types of trauma, including burns and hypothermia. Posttraumatic gastrointestinal erosions and ulcers represent a significant, sometimes life-threatening and possibly fatal complication of injuries; their medicolegal implications are of great importance in forensic pathology practice.

The terminology encountered in medical literature, which deals with this type of gastrointestinal pathology, includes "acute erosive gastritis," "acute hemorrhagic gastritis," "acute gastric mucosal lesions (AGML)," "stress gastroduodenitis," "stress ulcers," "stress-related mucosal disease," Curling ulcers (in burns), Cushing ulcers (in craniocerebral trauma), "leopard spots," and Wischnewsky spots (in hypothermia). Our research was limited to victims of trauma so we adopted the term "posttraumatic gastroduodenal ulcers" (PGDU). Furthermore, almost all lesions diagnosed at post-mortem (with the exception of two cases) were confined to the stomach and/or duodenum.

As early as 1772, John Hunter presented a case of acute gastro-duodenal ulcers found at autopsy of a patient with cerebral trauma (1); to this date, several papers on this subject have been published in the medical literature. However, the available research deals predominantly with pathophysiological and clinical aspects of PGDU, addressing the pathogenesis of their development and appropriate prophylactic and therapeutic measures (2–7). Larger studies based

on autopsy material and focused on medicolegal aspects of PGDU have not, however, been recently published in the English language (8–11). Therefore, we aim to assess various characteristics of PGDU, based on postmortem data, and their implications in medicolegal expertise dealing with complications of various types of trauma.

Material and Methods

We reviewed all autopsies carried out at the Institute of Forensic Medicine in Belgrade in a 5-year period from 1996 to 2000. Out of the total of 5197 postmortems there were 887 trauma cases with a risk of developing PGDU, as identified in the literature. The majority of these patients died after receiving treatment at the Emergency Department of the Clinical Center of Serbia, Belgrade. PGDU were diagnosed at autopsy in 157 out of the 887 decedents (group A); in 730 cases, PGDU did not develop despite the presence of risk factors (control group or group B).

Data were obtained from autopsy protocols, available medical records, police reports, and interviews with next of kin. The results were analyzed by means of usual statistical methods including standard deviation (SD) and χ^2 -test.

Results and Discussion

Incidence of PGDU

At autopsy PGDU were diagnosed in 157 (17.7%) out of the total of 887 victims deemed to be at risk from the development of such lesions. In an earlier study, carried out 23 years previously at our Institute on a similar sized sample (867 injured individuals), PGDU were detected in a significantly smaller proportion of victims—35 or 4% (9). This discrepancy is explicable by the advance of medical science and improvement in treatment of severely injured patients, leading to a prolonged survival period, which carries an inherent risk for the development of various complications,

¹Institute of Forensic Medicine, Medical School, University of Belgrade, Serbia.

²Institute of Pathology, Medical School, University of Belgrade, Serbia.

³Institute of Forensic Medicine, Clinical Centre of Vojvodina, Novi Sad, Serbia.

*Presented at the 60th Annual Meeting of the American Academy of Forensic Sciences, Feb. 18–23, 2008, in Washington, DC.

Received 19 Sept. 2008; and in revised form 26 Nov. 2008; accepted 06 Dec. 2008.

including PGDU. According to Zeltzman et al., the reported incidence rates of hemorrhagic stress ulceration in the surgical intensive care unit patient population vary between 25% and 75% (12). Popovic claims that at least 75% of critically ill patients develop mucosal lesions, as a direct consequence of stress, within the first 24 h following admission to intensive care unit (5). On the other hand, Simons et al., by analyzing 33,637 major trauma patients treated in a regionalized trauma system from 1985 to 1991, found that clinically obvious stress ulceration developed in 57 patients (0.17%) despite adequate prophylaxis. They concluded that clinically significant posttraumatic stress ulceration is uncommon, but can occur even with appropriate prevention (13). The difference between Simons' and our series is explicable by the nature of the study sample; the former only registered cases of clinically obvious PGDU in patients with serious injuries, while our results were based on postmortem diagnosis (irrespective of clinical manifestations) in persons who sustained fatal and therefore, as a rule, more serious trauma.

Sex and Age of Decedents

Our study sample showed a marked preponderance of male individuals both in group A and in the control group B—77.1% and 74.2% respectively. There was no statistically significant difference in the distribution of sexes between the two groups, which suggests that the male gender carries no specific risk for the development of PGDU, other than the fact that men appear to be more likely to suffer serious trauma than women. This is in accordance with results reported previously in the literature (9,14).

The age of patients developing PGDU varied between 5 and 88 years, with the mean age of 50.72 (SD = 21.53); the majority were in their seventies (Table 1). In the control group the youngest decedent was 6 months and the oldest 93 years of age, with the mean age of 48.91 (SD = 20.47); the majority were in their fifties and seventies. There was no statistically significant difference in the age distribution between groups A and B; there was, however, a somewhat higher percentage of patients aged over 50 in group A (52.3%), by comparison to group B (47.8%). This is in accordance with the results of our earlier research (9). Simons et al. concluded that age over 55 represents an independent risk factor for the development of PGDU (13).

There are various factors which could explain the increased incidence of PGDU in older individuals. The aging process diminishes the ability of the body to adapt to changes in the environment, as well as the efficiency of various protective mechanisms. According to some authors, the development of generalized atherosclerotic

and other degenerative changes affecting, among others, the digestive tract, may also play a part.

Onset of PGDU

The length of survival postinjury ranged between 24 h and 25 days, with an average of 8.8 days (SD = 6.47) in group A, and 7.7 days (SD = 5.86) in group B; the difference between the two groups was not statistically significant. In the majority of victims developing PGDU (76%), the survival time was 12 days or less; this was mostly due to the severity of initial trauma.

It should be pointed out that 16.5% of all PGDU were detected in victims who survived <48 h following trauma. Fennerty claims that 75–100% of critically ill patients demonstrate evidence of stress-related mucosal damage within 24 h of admission to the intensive care unit (2); this is confirmed further by Popovic (5). Such early development of PGDU underlines the necessity for immediate preventive measures.

Injuries in Cases with PGDU

The manner of death was similar in both groups and was mostly accidental (81%). The majority of decedents were injured in traffic accidents (66%), most commonly as pedestrians (66 or 42% of all 157 cases with PGDU).

Table 2 shows the absolute numbers and percentages of various types of trauma in the group with PGDU, the control group, and the sample as a whole. We found a statistically significant difference in the prevalence of isolated craniocerebral and cervical spinal cord injury between groups A and B.

The majority of victims developing PGDU sustained multiple injuries, a finding in accordance with the general prevalence of polytrauma in the seriously injured, particularly in vehicular accidents. Concurrent injury of two or more major organs or organ systems, with frequent development of posttraumatic complications, contributes to the cumulative effect of various risk factors for the development of PGDU.

Apart from 45 cases (28.7%) with isolated craniocerebral trauma, there were 56 further decedents where severe head injury occurred in combination with other serious injuries. Therefore, serious and/or fatal head injury was present in 101 (64.3%) of patients in group A. Out of the 70 decedents (44.6%), craniocerebral trauma was cited as the sole cause of death, while in the remaining 31 cases, head injury was diagnosed as fatal in concurrence with other types of trauma and/or complications of trauma.

Our results confirm the importance of craniocerebral trauma in the etiology and pathogenesis of posttraumatic ulcers, also known as Cushing's ulcers (15). According to Alain and Wang, Cushing's ulcer is one of the severe complications of traumatic brain injury, with a mortality rate exceeding 50% (16). Hatton et al. claim that neurosurgical patients are at risk from stress-induced gastric

TABLE 1—Age of injured individuals.

Age (years)	Group A		Group B	
	N	%	N	%
0–10	6	3.8	27	3.7
11–20	11	7.0	50	6.9
21–30	16	10.2	82	11.2
31–40	15	9.5	86	11.8
41–50	27	17.2	136	18.6
51–60	16	10.2	98	13.4
61–70	34	21.7	128	17.5
71–80	24	15.3	101	13.8
81–90	8	5.1	21	2.9
91–100	—	—	1	0.2
Total	157	100	730	100

TABLE 2—Type of injury.

Type of Injury	Group A		Group B		Total	
	N	%	N	%	N	%
Mechanical polytrauma	95	60.1	538	73.7	633	71.4
Isolated craniocerebral injury	45	28.7	157	21.5	202	22.8
Burns & scalds	9	5.7	24	3.3	33	3.7
Cervical spinal cord injury	4	2.5	4	0.5	8	0.9
Other	4	2.5	7	1.0	11	1.2
Total	157	100	730	100	887	100

erosions; they found a significant correlation between the occurrence of PGDU, GCS <8 and length of ventilatory support (17).

Twenty patients (12.7%) with polytrauma suffered potentially fatal chest injuries; the consequent respiratory insufficiency and inadequate ventilation appear to have a significant contributory role in the development of posttraumatic ulcers (15).

The relationship between burns and the development of gastroduodenal ulcers was first described by Curling; such ulcers are, to this date, known as Curling's ulcers. In 5.7% of our cases with PGDU there was evidence of extensive burns or scalds (involving more than 25–30% of body surface) which, together with associated complications, were responsible for death. Chernov and Miziev stated that patients with burns have the highest risk of dying as the result of hemorrhage from gastric and/or duodenal ulcers (18).

There were very few cases of hypothermia in our sample; all exhibited scanty mucosal lesions in the stomach, known as Wischnewski spots (19,20). These cases were excluded from the study, since recent research failed to demonstrate histological evidence of erosions or ulcers in the region of Wischnewski spots (20).

Table 3 shows the rate of PGDU in certain types of trauma; patients with cervical spinal cord injuries appear to be at greatest risk, followed by those suffering with burns and craniocerebral trauma; this is in accordance with data reported in the literature (15,21). There is a statistically significant difference in the occurrence of ulcerations in cases with cervical spinal cord trauma when compared with polytrauma ($\chi^2 = 3.83$, $p < 0.05$). There is no statistically significant difference between patients with burns and isolated craniocerebral injuries. PGDU developed in 15% of decedents with polytrauma, which is in keeping with earlier reports of posttraumatic ulcerations occurring in 17–20% of such cases (22).

Although, in our sample, the absolute number of cases with isolated cervical spinal cord injury was significantly smaller than those with polytrauma and craniocerebral trauma, the results indicate, nevertheless, that this type of injury carries a high risk for the development of PGDU. This is in accordance with other reports in the literature (7); some authors even identify cervical spinal cord injury as an independent risk factor in the development of posttraumatic ulcers (13). According to earlier research, gastrointestinal ulceration or hemorrhage occur in 2–20% of victims of cervical spinal cord injury; some authors highlight the joint effects of vagal stimulation and sympathetic inhibition in the pathogenesis of these ulcers, while others support a multifactorial approach to causes of gastrointestinal bleeding, rather than a mechanism depending on cervical spinal cord injury alone (15). Spinal cord injury at other levels does not appear to have such a strong relationship with PGDU as does the damage to the cervical segment.

In our sample the majority of victims with PGDU (91%) had an ISS ≥ 16 , with a mean value of 26.18 (SD = 11.02), which underlines the role of severity of trauma in the development of posttraumatic ulcers. Simons et al. identified ISS ≥ 16 as an independent risk factor for the occurrence of PGDU (13). We postulated that, in the remaining 9% of cases with ISS < 16, other factors, such as complications of trauma and exacerbation of pre-existing chronic illnesses, also played a part in the development of ulcers.

TABLE 3—Incidence of PGDU in relation to type of injury.

Type of Injury	All Cases	Cases with PGDU	%
Cervical spinal cord injury	8	4	50.0
Burns & scalds	33	9	27.3
Isolated craniocerebral injury	202	45	22.3
Mechanical polytrauma	633	95	15.0

Additional Risk Factors for PGDU

Apart from initial trauma there are a number of other factors which can play a part in the pathogenesis of PGDU, by increasing the risk for their development. Most important among these are pre-existing chronic diseases, extensive surgery and various complications which may develop in severely injured patients.

Pre-existing disease, affecting normal bodily responses to injury and increasing the likelihood for the development of complications including PGDU, can cause significant difficulties in treatment and recovery from trauma. Based on the available hospital notes and information obtained in interviews with next of kin, we found that a considerable number of patients (32 or 20.4%) had some form of cardiovascular disease (arterial hypertension, angina, or cardiomyopathy). In 10 decedents, there was evidence of diabetes mellitus and one suffered with hemophilia. Any form of pre-existing coagulopathy is likely to promote hemorrhage from a stress ulcer (23); prophylactic administration of antacids following trauma is strongly recommended in all such patients. Also to be noted is the fact that 26 (16.6%) patients in our sample had a history of alcohol abuse. Chronic alcohol abuse has an adverse effect on the protective mechanisms in gastric mucosa, promoting the development of gastritis and ulcers. In such cases, subsequent trauma can lead to the development of acute or to exacerbation of chronic ulcers. Alcohol-induced damage to the nervous system and the development of alcoholic neuropathy are a further contributory factor in the pathogenesis of posttraumatic ulcers (24).

Major surgery is another risk factor for the development of stress ulcers. Seventy-six patients (48.4%) in group A underwent one and 11 (7%) two surgical interventions following trauma. Most common were neurosurgical operations (38.6%) followed by abdominal (30.7%) and thoracic surgery. As a rule, all such interventions are complex and involve significant blood loss. Our results are in keeping with the views held by other authors, who highlight the relevance of neurosurgical interventions, as well as those associated with significant intra-operative blood loss, in the development of PGDU (25).

According to previous research, there is a strong association between the development of stress ulcerations and other serious posttraumatic complications, including pneumonia, sepsis, and organ failure such as adult respiratory distress syndrome, renal and hepatic failure (13). These complications usually develop after a prolonged period of survival. In 50% of our cases with PGDU, there were concurrent respiratory complications, most commonly infections. The association between chest infections and PGDU is well recognized and has been highlighted by other authors (9,15,26). Impaired gas exchange and drop in blood oxygen saturations contribute to the development of posttraumatic ulcers and subsequent hemorrhage. Sijacki et al. point to respiratory failure and sepsis as significant risk factors for bleeding from PGDU (27).

In 20% of cases in group A there was evidence of urinary tract infection including, most commonly, cystitis and pyelonephritis. Renal insufficiency in patients on a respirator increases significantly the risk of major hemorrhage from stress ulcers (28). According to our earlier study, uraemia was a frequent finding in injured patients who developed PGDU (9). Other concomitant complications were encountered less frequently (cardiac complications in 6%, thromboembolism in 8%, peritonitis in 4%, CNS complications in 3%, and sepsis in 2% of cases).

Morphology and Distribution of PGDU

Acute PGDU may appear in the form of erosions and/or acute ulcers. Erosions are oval or circular, shallow mucosal defects not

deeper than lamina muscularis measuring, most commonly, between 0.1 and 0.3 cm, although, in some cases, they may be up to 3 cm in diameter. They are usually multiple and involve the stomach mucosa. Acute ulcers are defects which penetrate more deeply than lamina muscularis; they are usually larger than erosions and, in the majority of cases, measure <1 cm. As opposed to peptic ulcers, stress ulcers can arise anywhere in the stomach; they are, however, most frequently encountered in areas where erosions are denser and mucosal congestion more prominent, such as along the greater curve. Stress ulcers are usually multiple but may be solitary. The presence of numerous small ulcers and erosions gives the picture of acute erosive gastritis (Fig. 1). The critical difference in histological appearances between an acute and a chronic peptic ulcer lies in the absence of mature scar tissue at the bottom and of thickened vessel walls in the vicinity of the ulcer. Gastroduodenal erosions could heal in the course of 48 h, while in cases of acute ulcerations this may take a few days or even weeks.

Acute ulcers may also appear in the duodenum, where they are usually solitary. As opposed to other types of posttraumatic ulcerations, Cushing's ulcers are more commonly found in the duodenum; they tend to be deeper, solitary lesions which are more likely to perforate. In adults, Curling's ulcers are evenly distributed in the stomach and duodenum, while in children they occur more frequently in the duodenum (21,29). Severe trauma can also lead to exacerbation of pre-existing chronic peptic ulcers, which represent a point of lesser resistance (9).

Our results are in keeping with earlier research on the subject of PGDU. Acute PGDU were a dominant feature in 138 cases (87.9%), chronic ulcers were found in 15 (9.5%), while in six cases (2.6%) we found a mixed picture of acute and chronic lesions. In the majority of cases the lesions were acute and they involved the stomach (Table 4). Duodenal ulcers were much less frequent; acute lesions were only slightly more common than chronic ulcers. In 13 cases (8.2%) there was involvement both of the stomach and duodenum; in nine cases, the lesions were acute in both sites. In three cases, we found acute gastric ulcers in the presence of a chronic duodenal ulcer, and only in one case a chronic gastric ulcer co-existed with acute duodenal lesions. There was one case with acute ulcerations in the region of the cardia and esophagus and one with a chronic esophageal ulcer.

In the majority of cases where ulcers were found in the stomach (75 or 61.98%) the pathologist did not give a more precise location of the lesions. Duodenal ulcers were most commonly found in the first part of the duodenum.



FIG. 1—Acute erosive gastritis.

TABLE 4—Site of PGDU.

Site	Acute Lesions	Chronic Lesions	Acute and Chronic Lesions	Total	
				N	%
Stomach	115	5	1	121	77.1
Duodenum	11	9	1	21	13.4
Stomach and duodenum	9	—	4	13	8.3
Esophagus and cardia	1	—	—	1	0.6
Esophagus	—	1	—	1	0.6
Total	138	15	6	157	100

Out of the 121 cases with gastric involvement in 110 cases the lesions were multiple and acute (90.1% of 121, i.e., 70.1% of a total of 157 cases with PGDU). Multiple ulcers were found only in 3 out of 21 patients with duodenal involvement; in eight cases, the number of lesions was three or less and in the remaining 10 cases the ulcers were solitary. Exacerbation of a chronic duodenal ulcer was found in 10 cases; the majority had solitary lesions.

In conclusion, the stomach was the most frequent site of PGDU exhibiting, as a rule, multiple acute lesions. Posttraumatic ulcers were rarely found in the duodenum, where they were usually solitary and often presented as exacerbated chronic peptic ulcers.

Complications of PGDU, Their Clinical Manifestations and Significance

PGDU may be silent or may become clinically apparent through hemorrhage, perforation, or penetration of adjacent structures. Such complications, and most importantly significant bleeding, can have an adverse effect on patient's recovery and can, in certain cases, lead to a fatal outcome. Cook et al. highlighted the economic impact of PGDU in cases of survival posttrauma: they complicate the clinical course and can prolong the need for hospitalization in ITU for an average of 4–8 days (28).

Symptoms of PGDU include abdominal pain (more frequent in ulcers rather than erosions), nausea, and vomiting. However, the most frequent clinical manifestation of PGDU is gastrointestinal hemorrhage, of varying intensity, usually not associated with other symptoms. Therefore, PGDU should be suspected in patients with mild anemia, presence of occult blood in the stool or, in more serious cases, hematemesis or melaena. A more significant blood loss (of 15 to 20% of circulating volume) may lead to shock and compound the effects of bleeding caused by initial trauma.

Endoscopic studies have shown that petechial hemorrhages and mucosal erosions occur most frequently within the first 24 h postinjury, when they may cause slight bleeding, lasting up to 48 h. Such hemorrhage is usually self-limiting, due to the capacity of gastric mucosa for rapid healing. In milder cases, histological and endoscopic findings may return to normal within 48 h. At autopsy, the presence of blood may sometimes be detected only in isolated segments of the gastrointestinal tract, usually the large intestine, with no demonstrable source of hemorrhage. This would indicate an earlier episode of bleeding from stress-related erosions, which had healed before the blood had been evacuated from the large bowel.

Major hemorrhage from PGDU develops in 5–20% of cases and can occur as early as 2–3 days postinjury. Advancements in treatment have reduced the risk from serious bleeding in severely ill patients, with the exception of those suffering with coagulopathy and respiratory insufficiency (30,31). However, adequate prophylaxis cannot prevent bleeding in all patients. Larger studies carried out in trauma intensive care units have shown clinical evidence of bleeding

in 1–9% of cases despite preventive measures. It must be pointed out that major hemorrhage, requiring blood transfusions or surgery, is relatively rare (2,5,7,17,32). In a small proportion of cases, despite all diagnostic and therapeutic procedures, bleeding from PGDU cannot be controlled and can lead to a fatal outcome. In a prospective 5-year study, Sijacki et al. registered 84 cases with clinically significant bleeding; among those, 13 patients required surgery and 29 (34% of 84) died due to exsanguination, including 7 of the 13 who were treated surgically (27). According to Lu et al. 17% of PGDU progress to clinically significant gastrointestinal bleeding, which has been associated with mortality rates of up to 50% (15).

Complications of PGDU were demonstrated, at autopsy, in 64 out of 157 analyzed cases (40.8%). These included, most commonly, bleeding, found in 62 patients (39.5% of 157). Hemorrhage was usually isolated, while in four cases, it was associated with perforation, and in a further four cases with penetration of adjacent structures (Table 5). Complications of PGDU were regarded as contributory to the cause of death in 32 out of 64 cases of complicated ulcers (20.4% of all 157 cases in group A). Hemorrhage from PGDU was cited as part of the cause of death in 11 cases, and in one case it was the sole cause of death.

It is interesting that in half of the cases with complicated PGDU (mostly with hemorrhage), these complications were not referred to in the final conclusions regarding the cause of death. The extent of bleeding, and its contribution to the fatal outcome, may be difficult to assess based on postmortem findings alone, and may be arbitrary, depending on the pathologist involved. In view of the above, the need for detailed analysis of the patient’s medical records cannot be overemphasized.

Assessment of Clinical Diagnosis

Review of the available hospital notes showed that PGDU, detected at autopsy, were relatively infrequently diagnosed by treating medical practitioners; clinical diagnosis of a bleeding ulcer was only made in 12 patients (19.4% of 62 cases with postmortem evidence of gastrointestinal bleeding); six of those required surgical intervention to control the hemorrhage. In the majority of clinically recognized cases of major gastrointestinal bleeding, death occurred within 24 h of diagnosis; this confirms the severe nature of this type of complication, which can occur, and cause death, despite timely preventive and therapeutic measures. Particularly worrying is the fact that in five cases of fatal gastrointestinal bleeding, diagnosed at autopsy, there was no clinical recognition of major hemorrhage.

Conclusion

In conclusion, according to our research, a patient with the highest risk for the development of PGDU is male, aged over 50,

injured in a traffic accident as a pedestrian, sustaining multiple injuries, including damage to the cervical spinal cord and/or craniocerebral trauma, with ISS≥16, and a survival period of up to 12 days, associated with the development of posttraumatic complications, particularly pneumonia.

Medicolegal Aspects of PGDU

Medicolegal expertise in cases of fatal trauma, compounded by the development of PGDU and associated complications such as hemorrhage, perforation, or penetration of adjacent structures, requires a precise scientific assessment of causal relationship between initial injury, PGDU, and fatal outcome. The medicolegal expert should explain all the risk factors leading to the development of posttraumatic ulcers and assess the contribution of this type of complication to the fatal outcome. This may present no significant problem in cases of major trauma. However, in a small proportion of cases, particularly in the elderly and in those with pre-existing disease and/or other contributory factors, less serious injuries may lead to the development of acute gastroduodenal ulcers, sometimes with fatal consequences. Such cases are particularly challenging to the forensic pathologist who may be asked to differentiate between the role of trauma and other risk factors in the development of acute gastroduodenal ulcers.

Fatal complications of PGDU can also lead to allegations of medical negligence, particularly in instances where there is a failure to diagnose and treat a consequent catastrophic gastrointestinal hemorrhage. Furthermore, in such cases the defendants and their legal teams may seek to shift the responsibility for the fatal outcome away from the initial injury, hiding behind complications which develop, allegedly, due to inadequate medical treatment. In order to assist the Court, medicolegal experts should give a scientifically based and unbiased opinion as to whether all preventive, diagnostic, and therapeutic measures were promptly and adequately performed.

References

1. Dragovic M, Gerzic Z. The principles of surgery (Osnovi hirurgije). The electronic PDF issue, DAN Design 1998;1294–6.
2. Fennerty MB. Pathophysiology of the upper gastrointestinal tract in the critically ill patient: rationale for the therapeutic benefits of acid suppression. Crit Care Med 2002;30(6 Suppl):351–5.
3. Sesler JM. Stress-related mucosal disease in the intensive care unit: an update on prophylaxis. AACN Adv Crit Care 2007;18:119–26.
4. Grube RR, May DB. Stress ulcer prophylaxis in hospitalized patients not in intensive care units. Am J Health Syst Pharm 2007;64:1396–400.
5. Popovic N. Importance of prevention of acute mucosal lesions in patients in intensive care units. Acta Chir Jugosl 2007;54:47–50.
6. Robertson MS, Wilson SJ, Cade JF. Acute stress ulceration prophylaxis: point prevalence surveys in intensive care units in Victoria, 1997 and 2005. Crit Care Resusc 2008;10:18.
7. Zarzaur BL, Kudsk KA, Carter K, Pritchard FE, Fabian TC, Croce MA, et al. Stress ulceration requiring definitive surgery after severe trauma. Am Surg 2001;67:875–9.
8. Shafer N, Shafer R, Wolf C. Stress and ulceration: medico-legal aspects. Leg Med 1985;93–107.
9. Djurovic V, Savic S, Pandurovic S. Post-traumatic gastroduodenal ulcerations. Srp Arh Celok Lek 1985;113:991–6.
10. Kringsholm B, Thomsen JL, Jensen JL, Hansen JP, Voigt J. Acute esophago-gastro-duodenal ulceration in forensic pathology. Ugeskr Laeger 1979;141:1074–7.
11. Mendez J, Hadengue A, Terris G. Medico-legal problems posed by post-traumatic gastroduodenal ulcer. Med Leg Damage Corpor 1970;3:173–87.
12. Zeltzman D, Rowland M, Shanavas Z, Kerstein MD. Is the incidence of hemorrhagic stress ulceration in surgical critically ill patients affected by modern antacid prophylaxis? Am Surg 1996;62:1010–3.

TABLE 5—PGDU and fatal outcome.

	Complications of PGDU				Total
	H	H & Perf	H & Pen	Pen	
PGDU as part of cause of death	6	3	1	—	10
PGDU as only cause of death	1	—	—	—	1
PGDU and other complications of trauma as cause of death	16	1	3	1	21
PGDU not included in cause of death	31	—	—	1	32
Total	54	4	4	2	64

H, hemorrhage; Perf, perforation; Pen, penetration.

13. Simons RK, Hoyt Db, Winchell RJ, Holbrook T, Eastman AB. A risk analysis of stress ulceration after trauma. *J Trauma* 1995;39:289–93.
14. Mutlu GM, Mutlu EA, Factor P. Prevention and treatment of gastrointestinal complications in patients on mechanical ventilation. *Am J Respir Med* 2003;2:395–411.
15. Lu WY, Rhoney DH, Boling WB, Johnson JD, Smith TC. A review of stress ulcer prophylaxis in the neurosurgical intensive care unit. *Neurosurgery* 1997;41:416–25.
16. Alain BB, Wang YJ. Cushing's ulcer in traumatic brain injury. *Chin J Traumatol* 2008;11:114–9.
17. Hatton J, Lu WY, Rhoney DH, Tibbs PA, Dempsey RJ, Young B. A step-wise protocol for stress ulcer prophylaxis in the neurosurgical intensive care unit. *Surg Neurol* 1996;46:493–9.
18. Chernov VN, Miziev IA. Acute gastric and duodenal ulcers in burns. *Klin Med* 1998;76:42–4.
19. Birchmeyer MS, Mitchell EK. Wischnewski revisited. The diagnostic value of gastric mucosal ulcers in hypothermic deaths. *Am J Forensic Med Pathol* 1989;10:28–30.
20. Tsokos M, Rothschild MA, Madea B, Rie M, Sperhake JP. Histological and immunohistochemical study of Wischnewsky spots in fatal hypothermia. *Am J Forensic Med Pathol* 2006;27:70–4.
21. Sevtitt S. Duodenal and gastric ulceration after burning. *Br J Surg* 1967;54:32–41.
22. Michek J, Svoboda P, Zelnicek P, Kantorova I, Ochmann J. Stress ulcers in patients with polytrauma. *Rozhl Chir* 1996;75:194–6.
23. Hiramoto JS, Terdiman JP, Norton JA. Evidence-based analysis: postoperative gastric bleeding: etiology and prevention. *Surg Oncol* 2003;12:9–19.
24. Dixon MF, Genta RM, Yardley JH, Correa P. Classification and grading of gastritis. The updated Sydney system. International Workshop on the Histopathology of Gastritis, Houston 1994. *Am J Surg Pathol* 1996;20:1161–81.
25. Raicevic R, Jovicic A, Ostojic M, Markovic Lj, Zoric L, Marenovic T, et al. Stress ulcer in patient with a subacute subdural hematoma. *Vojnosanit Pregl* 1999;56:435–7.
26. Fabian TC, Boucher BA, Croce MA, Kuhl DA, Janning SW, Coffey BC, et al. Pneumonia and stress ulceration in severely injured patients. A prospective evaluation of the effect of stress ulcer prophylaxis. *Arch Surg* 1993;128:185–91.
27. Sijacki AD, Popović N, Karamarković A, Stefanović BD, Djukić V, Lausević Z, et al. Risk factors for bleeding from stress ulcer in severely injured and critically ill persons. *Acta Chir Jugosl* 2007;54:77–81.
28. Cook DJ, Griffith LE, Walter SD, Guyatt GH, Meade MO, Heyland DK, et al. The attributable mortality and length of intensive care unit stay of clinically important gastrointestinal bleeding in critically ill patients. *Crit Care* 2001;5:368–75.
29. Pruitt BA, Goodwin CW. Stress ulcer in the burned patient. *World J Surg* 1981;5:209–20.
30. Shuman RB, Schuster DP, Zuckerman GR. Prophylactic therapy for stress ulcer bleeding? A reappraisal. *Ann Intern Med* 1987;106:562.
31. Brett S. Science review: the use of proton pump inhibitors for gastric acid suppression in critical illness. *Crit Care* 2005;9:45–50.
32. Dubracic D, Pulanic R, Katicic M, Car D, Georgijevic A, Cvitanovic B. Hemostasis and treatment of stress hemorrhage in the upper gastrointestinal tract of trauma patients. *Acta Med Austriaca* 1993;20:127–30.

Additional information and reprint requests:
 Slobodan Savic, M.D., Ph.D.
 Institute of Forensic Medicine
 School of Medicine, University of Belgrade
 Deligradska 31a
 11000 Belgrade
 Serbia
 E-mail: brana.s@eunet.rs