Syncope and trauma. Are syncope-related traumatic injuries the key to find the specific cause of the symptom?

J. Auer*

Europäklinikum Braunau/Simbach, Department of Internal Medicine and Cardiology, General Hospital Braunau/Simbach, Ringstrasse 60, A- 5280 Braunau am Inn, Austria; Plinganser Strasse 10, D-84359 Simbach am Inn, Germany

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This editorial refers to ‘Physical injuries caused by a transient loss of consciousness: main clinical characteristics of patients and diagnostic contribution of carotid sinus massage’ by A. Bartoletti et al.,† on page 618

Syncope is a prevalent disorder, accounting for 3–5% of emergency department (ED) visits and 1–3% of hospital admissions. A cardiac cause of syncope is an independent predictor of sudden death, and mortality rates are higher in patients with cardiac syncope compared with those of non-cardiac or unknown origin. In addition, significant morbidity may result from falls or accidents resulting from syncope. Bartoletti et al. have provided valuable information about the prevalence and the characteristics of secondary trauma among patients referred to the ED for a transient, self-limited loss of consciousness (TLOC). A total of 1114 patients with a true syncope and 139 individuals with a non-syncopal condition (including seizures, cerebrovascular accidents, dizziness, intoxication, hypoglycaemia, and psychogenic disorders) who presented during a period of 24 months were enrolled in this single-centre study. Among the 1253 consecutive patients with TLOC, 365 reported a trauma, which was classified as severe in 59 cases.

The prevalence and the location of trauma did not differ significantly between patients with syncope and individuals with non-syncopal conditions. Among patients with true syncope, traumatic injuries were more frequent when syncope occurred at home, in the orthostatic position, and without prodromal symptoms. As expected, older age, syncope at home, and TLOC in an upright position were associated with severe trauma. Syncope at home as a predictor of trauma seems to reflect patient characteristics that are associated with increased risk of injury, including advanced age, cognitive impairment, and reduced physical activity. Falls account for 90% of hip fractures, and the risk of falling increases with age as a result of impaired regulation of cerebral blood flow and polypharmacy. Compared with a younger woman, a 70-year-old woman is five times more likely to sustain a hip fracture and three times more likely to incur any fracture during the rest of her life.

The prevalence of trauma did not differ significantly among the main causes of syncope (cardiac, orthostatic hypotension, neurally mediated, and other causes including unknown origin). Thus, based on this finding, prevalence and the characteristics of syncope-related traumatic injuries do not add valuable information for elucidation of the specific cause of the symptom in patients referred to the ED for TLOC. Selecting patients referred to the ED threatens the external validity of the study and does not allow the findings to be generalized to the population at large. A study that only includes patients with syncope initially treated in an ED may overestimate the prevalence of secondary trauma in TLOC patients. Thus, the risk of traumatic injury associated with syncope might be much lower in the general population with TLOC.

It has to be stressed that cardiac syncope can be a harbinger of sudden death. Because patients with this condition have a poor prognosis, with a 6-month mortality rate of >10%, timely and thorough evaluation is warranted. Among patients with severe syncope-related trauma, guidelines-based initial evaluation and further diagnostic work-up as appropriate resulted in a very high rate (>75%) of ‘definite’ diagnosis. Although assessment of this study subgroup (trauma patients) differed from that of previous trials that studied patients referred to the ED for syncope, this finding is surprisingly consistent with those of several prior studies. History and physical examination are the most specific and sensitive ways to evaluate syncope. The diagnosis is achieved with a thorough history and physical examination in up to 50% of patients. Additionally, electrocardiography should be performed in all patients since the presence or absence of heart disease offers

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* Corresponding author. Tel: +43 7722 804 5000, Email: johann.auer@khbr.at/johann.auer@khsim.de
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important information about the need for further evaluation.\textsuperscript{10} In general, young patients without structural heart disease who have a history consistent with the presence of vasovagal, orthostatic, or medication-induced syncope have a good prognosis and can be followed-up expectantly. However, as suggested by current guidelines, additional assessment and treatment may be necessary in high risk or high frequency settings such as recurrent TLOC that alters quality of life or exposes patients at high risk of trauma.\textsuperscript{11} Additional assessment to improve diagnostic accuracy may include the use of an implantable loop recorder that should be limited to patients with rare episodes of syncope, long intervals between episodes, and a high probability of arrhythmic syncope in whom the severity or hazardous nature of the episodes warrants specific treatment. These devices record rhythm abnormalities automatically or when activated by the patient, and have demonstrated incremental diagnostic yield in patients with unexplained syncope. A diagnosis can be made in up to 90% of patients who have these devices implanted.\textsuperscript{12} Although the documentation of bradyarrhythmia concurrent with a syncopal episode is considered diagnostic in the case of a cardioinhibitory neurally mediated event, an additional vasodepressor component cannot be ruled out without haemodynamic monitoring and may lead to recurrent syncopal episodes despite appropriate therapy of the cardioinhibitory component.\textsuperscript{13}

Interestingly, within the group of neurally mediated syncope, carotid sinus syndrome (CSS) and—to a lesser extent—situational syncope were associated with a higher risk of traumatic injury as compared with vasovagal syncope.\textsuperscript{3} As this subgroup analysis was based on only 37 patients diagnosed with CSS, these results should not be overestimated and confirmation in further larger scale studies is warranted. In a randomized controlled trial, Rose Kenny and colleagues\textsuperscript{14} investigated whether cardiac pacing reduces falls in older adults with cardioinhibitory CSS. A total of 175 patients above 50 years of age attending an ED facility because of a non-accidental fall were randomized to dual-chamber pacemaker implant or standard treatment without pacing. Paced patients were significantly less likely to fall than were controls (with a relative risk reduction of 58%). Syncopal events were also reduced during the follow-up period, but there were far fewer syncopal events than falls. Remarkably, injurious events were reduced by 70%. Falls without TLOC were reduced by two-thirds. In those patients, pacing may have prevented subsequent falls by modifying bradycardia-induced hypotension, insufficient to cause loss of consciousness, but sufficient to result in gait and balance instability. Considering these data that suggest beneficial effects of pacing in older adults with cardioinhibitory CSS complicated by injuries beyond prevention of syncope recurrences, such patients with TLOC complicated by a trauma and with a non-diagnostic guidelines-based initial evaluation, carotid sinus massage (CSM) should be part of the additional diagnostic work-up. The main complications of CSM are cerebrovascular accidents, with an incidence of 0.2–0.5%. Despite the low rate of neurological complications, CSM should not be performed in patients with a recent episode of a stroke or a transient ischaemic attack, or in patients with carotid bruits. Among the entire population investigated by Bartoletti and colleagues,\textsuperscript{3} no cases of death were observed as a direct consequence of trauma. However, as pointed out by the authors, this finding has to be considered with caution, since immediate death as a consequence of trauma and specific referral strategies for trauma patients after TLOC might have introduced bias. Thus, the benign nature of the disorder, as suggested by these findings, may be restricted to the population enrolled in this particular study, and extrapolation of its findings to the population at large might not be appropriate. Unfortunately, follow-up data (including mortality, recurrences of syncope, and TLOC with trauma) of the entire study population with TLOC and trauma and information about therapeutic interventions that have been applied following establishment of a final diagnosis of TLOC are lacking. Such data would provide valuable information about the natural history of these disorders and the appropriateness of diagnostic work-up and therapeutic strategies.

In conclusion, the prevalence and the characteristics of syncope-related traumatic injuries do not add valuable information for elucidation of the specific cause of the symptom in patients referred to the ED for TLOC. Considering the selected population included in the study of Bartoletti et al.,\textsuperscript{3} the finding that traumatic injuries are common complications of TLOC can hardly be generalized to the population at large.

Appropriate diagnostic work-up can establish a very high rate of final and definite diagnosis of TLOC even in patients with severe trauma. Considering the impaired prognosis of patients with cardiac syncope, timely and thorough evaluation is of paramount importance.

Finally, CSM should be part of the additional diagnostic work-up in patients with TLOC complicated by severe trauma, since such patients with cardioinhibitory CSS may benefit from a permanent cardiac pacemaker.

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References
Hamman’s syndrome: an atypical cause of chest pain

Eduardo Barge-Caballero*, Alberto Bouzas-Mosquera, Daniel Gulias-Soidan, and Alfonso Castro-Beiras

A 17-year-old man presented to the Chest Pain Unit of our hospital 1 h after sudden onset of acute, sharp, neck-irradiated chest pain while sleeping. He had no relevant medical history except for current cigarette smoking. On examination, he was in good general condition, with temperature of 36.7°C, blood pressure 122/77 mmHg, heart rate 83 bpm, and normal cardio-pulmonary auscultation. Asthenic constitution and pectus excavatus were also noticed. Twelve-lead electrocardiogram showed no rhythm or repolarization abnormalities. As pointed out in the figure, moderate pneumomediastinum and mild pneumopericardium were first observed in a posteroanterior chest X-ray film and subsequently confirmed by CT scan. Specifically asked about, the patient did not report catarrhal symptoms, intense physical activity or chest trauma during the previous days. In this setting, clinical and radiological findings strongly suggested the diagnosis of spontaneous pneumomediastinum—also known as Hamman’s syndrome—, an infrequent entity that should be considered for differential diagnosis of acute chest pain specially among young people without risk factors for ischaemic heart disease. Patients with Hamman’s syndrome usually have a good outcome with conservative management and serious complications are exceptional. In our case, the patient was admitted for clinical observation and he did well with medical therapy alone, remaining completely asymptomatic during hospital stay. Four days later, complete radiological resolution was confirmed in a new chest X-ray film, so he was discharged.