Review

Life-threatening outcomes associated with autonomic dysreflexia: A clinical review

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Context: Autonomic dysreflexia (AD) is a life-threatening complication of chronic traumatic spinal cord injury (SCI).

Objective: To document and provide insight into the life-threatening sequelae associated with AD.

Methods: A review was conducted to identify literature which documented cases of AD associated with life-threatening outcomes (and death). The search strategy comprised of a keyword search on the PubMed database as well as manual searches of retrieved articles. Outcomes were categorized into three main classes: central nervous system (CNS), cardiovascular (CV), and pulmonary.

Results: Thirty-two cases of death or life-threatening complications of AD were found. Twenty-three (72%) cases were CNS-related, seven (22%) cases were CV-related, and two (6%) cases were pulmonary-related. In total, seven (22%) deaths were noted as a direct result of complications following an AD attack.

Conclusion: AD is a well-known consequence of SCI among individuals with high thoracic and cervical injuries. Many of these individuals experience this condition on a daily basis. Medical personnel, care givers, and individuals with SCI should be aware of the importance of timely diagnosis and management of this life-threatening condition, which can result in a variety of significant complications including stroke, seizures, myocardial ischemia, and death.

Keywords: Autonomic dysreflexia, Hemorrhage, Ischemia, Seizures, Spinal cord injuries

Introduction

Paralysis is an obvious and devastating consequence of spinal cord injury (SCI). However, secondary complications resulting from injury to the autonomic nervous circuits results in various conditions (unstable blood pressure (BP) control, bladder and bowel dysfunctions, and others) that are frequently invisible to the human eye. The changes in sympathetic nervous system activity that occur as a result of loss of supraspinal control of the spinal autonomic circuits are a major cause of these secondary conditions.[1] Injury to the spinal cord results in unbalanced autonomic control that typically presents as diminished sympathetic activity.[1,2] However, following SCI some conditions can precipitate overactive sympathetic episodes that may cause life-threatening events among these individuals.[1] Of the various potential secondary complications, one of the most life-threatening conditions facing individuals with SCI is autonomic dysreflexia (AD).[3,4]

The following is a typical scenario of a clinical presentation of an individual with AD. A 48-year-old man with C3 AIS C (American Spinal Injury Association Impairment Scale) tetraplegia was seen in a sexual health clinic for a sperm-retrieval procedure with vibrostimulation. His resting supine cardiovascular (CV) parameters were arterial BP of 120/60 mmHg with a regular heart rate of 62 beats per minute. During the vibrostimulation procedure, a BP of 200/100 mmHg was recorded briefly, accompanied by 15–20 minutes of intermittent episodes of premature ventricular contractions (Fig. 1). He also developed a significant headache during the procedure, and appropriate measurements were taken to manage the episode of AD. These aberrant CV responses are well known among individuals with high thoracic and cervical SCI and frequently a cause of significant discomfort and emotional distress. Is this individual at risk for life-threatening complications during an episode of

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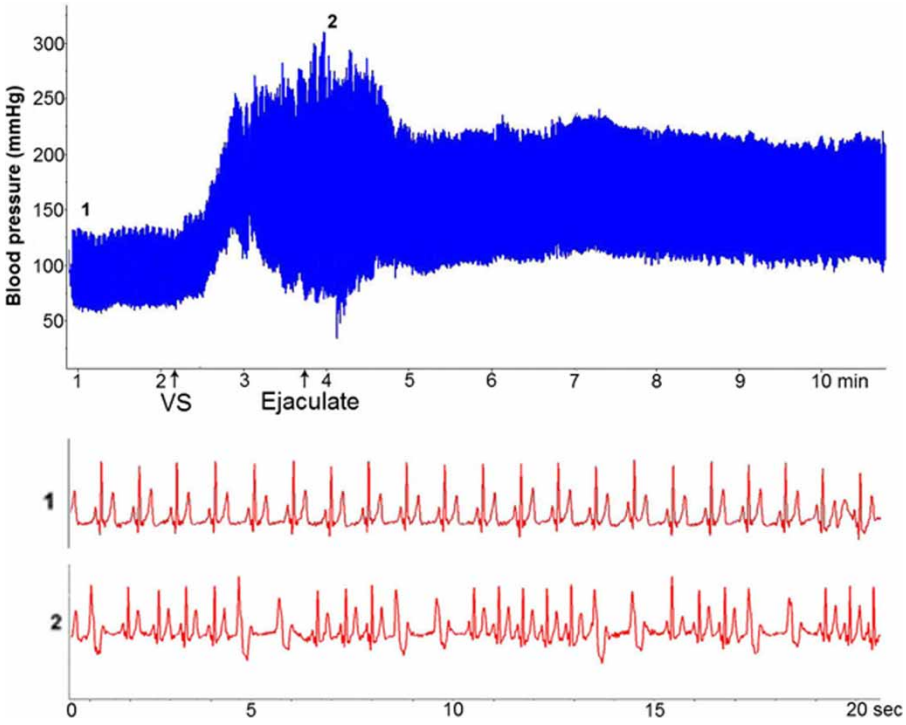

Figure 1 A case of AD accompanied by documented arrhythmia in a man with cervical incomplete SCI (C3 AIS C – American Spinal Injury Association Impairment Scale) during a vibrostimulation procedure to facilitate sperm retrieval. Continuous BP and ECG were recorded during the procedure. A representative recording of BP (top image) and a 20-second ECG recording during the episode of AD (bottom image) are presented. Despite the fact that 5 mg of nifedipine was given ~20 minutes before initiation of vibrostimulation, the individual developed a significant elevation in BP (up to 200 mmHg) and a prolonged period of arrhythmia with groups of premature ventricular contractions.

AD? What potential complications can clinicians expect to see, even in the presence of appropriate management?

AD is characterized by paroxysmal episodes of inappropriate sympathetic activity associated with hypertensive crises.[2,5] AD is known to occur in individuals who have experienced SCI at the T6-level or higher.[6] The condition is commonly triggered by both noxious and non-noxious stimuli experienced below the level of SCI, followed by massive sympathetic output to the peripheral targets including blood vessels and the heart.[3,4] Excessive sympathetic discharge in the absence of descending inhibition (due to SCI) leads to vasoconstriction below the level of SCI and critically elevated BP. Compensatory bradycardia is frequently observed during episodes of AD as a result of a preserved baroreceptor-mediated parasympathetic (vagal) response, although tachycardia can also be observed.[7] The descending inhibitory supraspinal signals can affect sympathetic neurons above the injury but, unfortunately, fail to cross the level of SCI.[8] In addition to bradycardia, vasodilatation and sweating are also observed above the level of injury in these individuals (areas of the body with preserved autonomic control).

It is well known that arterial BP following SCI can fluctuate dramatically due to disrupted central autonomic control.[9] On any given day, individuals with SCI can experience extremely low BP due to orthostatic hypotension;[10] numerous episodes of AD can also occur with systolic BP (SBP) reaching as high as 300 mmHg.[11] A major concern with repetitive BP fluctuation and periodic elevation in BP during the episodes of AD – which distinguishes these hypertensive episodes in individuals with SCI from hypertension in able-bodied individuals – is the possibility of shear injury to the blood vessel endothelium.[12,13] This may predispose these individuals to CV complications.

During the last two decades, the association between SCI and CV dysfunction has been documented in numerous studies.[14–16] It has therefore been shown that diseases of the circulatory and CV system are among the most common causes of death in the chronic SCI population.[14,16] Furthermore, the relationship between SCI and increased incidence of cerebrovascular disease such as stroke has recently been documented in a large cohort of individuals living with SCI.[17] The combination of immobility and disrupted BP control, manifested by volatile changes in

arterial BP, puts individuals with SCI at risk for life-threatening complications.[1,4,18]

The purpose of this review is to provide an overview of the most common documented complications associated with episodes of AD. These results will illuminate the underlying gravity of this serious, yet under-recognized condition. Although documented complications of AD may differ in outcome, they share the common etiology of association with AD in individuals with SCI.

Methods

A comprehensive literature search of original articles, case reports, and review articles was conducted to identify available information, published from 1965 to 2012, describing cases in which episodes of AD have led to potentially life-threatening sequelae or death. The keyword search terms "autonomic dysreflexia" and "autonomic hyperreflexia" were paired with the following terms: death, hemorrhage, infarct, life threatening, myocardial infarction, myocardial ischemia, pulmonary edema, seizure, stroke, and SCI. Studies that did not describe potentially life-threatening complications of AD (i.e. flushing, diaphoresis, retinal detachment) were excluded from this review.

The combined key word search strategy identified 156 publications relating AD to potentially life-threatening outcomes or death; 130 of these were excluded on the basis of failure to meet the criteria of articles describing potentially fatal outcomes or death associated with AD in humans. This left 26 unique articles from which information was extracted.

Each article was examined for specific documented cases when episodes of AD propagated life-threatening complications or resulted in death of the individual. These cases were then classified by the organ system that was primarily affected by the major complication – central nervous system (CNS), CV, or pulmonary. All available articles and case descriptions could be distinctly classified into one of the aforementioned groups. Within the category of CNS complications, individual cases could then be sub-classified into the following groups: ischemic complications, hemorrhagic complications, or seizures. Occasionally, a case qualified for more than one sub-classification (e.g. seizure and hemorrhage leading to death).

If BP or heart rate were mentioned within the case report, these data were extracted for statistical analysis. In cases that reported BP at more than one point during treatment of the individual, the highest value directly relating to the specific occurrence of AD leading to a life-threatening complication was recorded. Cases that lacked reported values for BP or heart rate were excluded from this analysis. A two-sample *t*-test was applied to relate reported SBP to the documented outcome of death due to a complication of AD. A simple box plot was then generated with the same data to present the findings.

Results

We were able to identify a total of 26 manuscripts describing 32 cases of life-threatening complications or death associated with episodes of AD. The identified cases were classified by the primary organ system that was affected as a result of an AD episode (Table 1). Of these 32 cases, we noted a total of 7 (22%) cases where episodes of AD resulted in death. Six of the aforementioned deaths were due to CNS-related complications, whereas the remaining death was due to pulmonary edema. None of the CV complications following AD resulted in death.

There were 23 unique cases of CNS-related complications following episodes of AD. The most common type of CNS complication was hemorrhage, which occurred in 11 (48%) out of 23 cases. Cerebral ischemia or infarction was documented in four (17%) cases; whereas seizures or convulsions were described in nine (39%) cases (one individual described by Kursh *et al.*[19] suffered from both seizure and subsequent hemorrhage).

We were able to identify seven cases of CV complications secondary to AD attacks including one (14%) case resulting in cardiac arrest, five (71%) cases involving arrhythmia, and one (14%) case resulting in silent myocardial ischemia. None of these cases resulted in fatality (Table 2).

Finally, two cases of pulmonary complications resulting from AD were identified. Both cases were described as pulmonary edema following episodes of AD. One case documented by Calder *et al.*[43] resulted in death (Table 3).

Statistical analysis using the data reported from case descriptions showed a mean SBP of 181 mmHg in the cohort of individuals who survived their episode of life-threatening AD, compared with a mean SBP of 214 mmHg in the group of cases that resulted in fatality (Table 4, Fig. 2). However, a two-sample *t*-test was not able to demonstrate statistical significance ($P = 0.0584$). It should also be noted that analyzing SBP in isolation may not be as meaningful as measuring a rise from baseline BP. Unfortunately, these data are unavailable in the majority of case reports.

Discussion

The purpose of this review was to evaluate the available literature for documented cases of AD that have

Table 1 CNS complications associated with AD

Author; year	Age	SCI information	CV parameters	Outcome
Kursh *et al.*; 1977[19]	15	C5 quadriplegia	BP: 210/140 mmHg	Complete resolution of seizure and extreme blurring of vision
Kursh *et al.*; 1977[19]	37	C4 fracture	BP: 118/80 mmHg	Gradual resolution of confusion and expressive aphasia possibly secondary to subarachnoid hemorrhage
Kursh *et al.*; 1977[19]	30	T1 paraplegia	BP: 230/170 mmHg HR: 48/minute	**Death** following focal seizure and subsequent confirmed intracerebral hemorrhage
Abouleish; 1980[20]	25	T3 paraplegia	Max BP: 220/120 mmHg	**Death** following bilateral cerebral hemorrhage
Kewalramani *et al.*; 1980[21]	–	–	–	Convulsions
Lindan *et al.*; 1980[6]	–	–	–	**Death** as a result of status epilepticus
McGregor *et al.*; 1985[22]	30	C6–C7 quadriplegia	BP: 150/90–210/100 mmHg	Massive intraventricular hemorrhage leading to residual right hemianopia, extraocular muscle dysfunction, short-term memory defects, and cognitive function impairment
Yarkony *et al.*; 1986[23]	25	C7 complete SCI	BP: 204/110 mmHg (first instance of seizure, 1983); 210/120 mmHg (second instance of seizure, 1985)	1983 – EEG showed evidence of focal damage and seizure disorder 1985 – Waking EEG and subsequent sleep-and-wake studies were normal
Yarkony *et al.*; 1986[23]	28	C6 complete quadriplegia	BP: 142/94 mmHg HR: 82/minute	Grand mal seizure lasting 2 minutes; EEG and CT studies of the brain revealed normal results
Yarkony *et al.*; 1986[23]	22	C6 quadriplegia	BP: 160/110 mmHg	Three occurrences of seizure 4 months after injury; EEG 2 years after injury showed residual slow wave abnormalities
Hanowell *et al.*; 1988[24]	39	C4 incomplete Frankel B	BP: 160/100–170/134 mmHg	Gradual recovery after left parietal intracerebral hemorrhage and edema
Eltorai *et al.*; 1992[25]	36	C6 incomplete quadriplegia	BP: 180/90 mmHg HR: 102/minute	**Death** following right cerebral hemorrhage with rupture into ventricles and subsequent cardiac arrest
Sahota *et al.*; 1994[26]	22	C2 quadriplegia	BP: 120/80–150/86 mmHg HR: 72–84/minute	Resolution of two episodes of seizures accompanied by cortical blindness
Colachis *et al.*; 2002[27]	21	C4 AIS B	BP: 180/110 mmHg HR: 86/minute	Resolution of aphasia possibly secondary to cerebral vascular insufficiency
Pan *et al.*; 2005[28]	Mid-30s	C8 AIS B	BP: 200/100 mmHg	Marked improvement after rehabilitation from right putaminal hemorrhage
Vallès *et al.*; 2005[29]	48	T4 ASI A	BP: sustained hypertension of up to 220 mmHg systolic and 120 mmHg diastolic	Residual cognitive deficits secondary to left occipital hemorrhagic lesion with perilesional edema
Dolinak *et al.*; 2007[30]	62	C6 vertebral fracture	BP: 200–230/100–120 mmHg	**Death** following right caudate nucleus hemorrhage rupturing into ventricles
Chaves *et al.*; 2008[31]	55	C5–C6 vertebral fracture, AIS C	SBP 213 mmHg, then EMT measurement of 160/100 mmHg 40 minutes later	Complete resolution of reversible posterior leukoencephalopathy syndrome
Edvardsson *et al.*; 2010[32]	32	C5–C6 vertebral fracture, AIS B	BP: 160/100 mmHg HR: 46/minute	Residual large cerebral infarct secondary to reversible cerebral vasoconstriction syndrome
Vaidyanathan *et al.*; 2011[33]	58	T6 AIS D, then subsequent T11–T12 fracture 2 years later	Not measured during AD episodes	1.2 cm infarct in right basal ganglia possibly due to recurrent AD
Yokomizo *et al.*; 2010[34]	72	C4 AIS C	Max BP: 210/100 mmHg	Residual disturbances in consciousness secondary to cerebellar hemorrhage
Yoo *et al.*; 2010[35]	45	C5–C6 fracture, sensory loss below T4, complete motor loss below C5–C6	BP: 268/185 mmHg HR: 54–58/min	**Death** following left basal ganglia and thalamic hemorrhage rupturing into ventricles
Vaidyanathan *et al.*; 2012[36]	46	C6 AIS A	Not measured during AD episodes	Convulsions and loss of consciousness but no structural abnormalities found on CT of the brain

SCI, spinal cord injury; CV, cardiovascular; BP, blood pressure; HR, heart rate; AIS, ASIA (American Spinal Injury Association) Impairment Scale; EMT, emergency medical technician; EEG, electroencephalography; CT, computed tomography scan.

Table 2 CV complications associated with AD

Author; year	Age	SCI information	CV parameters	Outcome
Guttmann *et al.*; 1965[37]	23	T3–T4 fracture-dislocation, transverse spinal cord syndrome complete below T5	BP: 130/80–210/105 mmHg HR: 56–60/minute	Normalization of cardiac arrhythmias after delivery
Forrest; 1991[38]	32	C5 complete quadriplegia	BP: 100/70 mmHg HR: 120/minute	Cardioversion to normal sinus rhythm (treatment for atrial fibrillation)
Forrest; 1991[38]	45	T2 complete paraplegia	BP: 210/110 mmHg HR: 180/minute (when admitted for supraventricular tachycardia)	Atrial fibrillation and recurrent supraventricular arrhythmia with transient ischemic attacks leading to atrioventricular nodal ablation treatment with placement of ventricular pacemaker
Forrest; 1991[38]	60	C5 fracture with C5-level quadriplegia	HR: 120/minute	Atrial fibrillation converted to sinus rhythm within 3 hours of digoxin treatment
Pine *et al.*; 1991[39]	60	C5 incomplete quadriplegia, Frankel C	BP: 160/90 mmHg (first episode) HR: 58/minute (first episode) BP: 110/70 mmHg (second episode) HR: 96/minute (second episode)	Resolution of atrial fibrillation with digoxin treatment
Colachis and Clinchot; 1997[40]	28	C6 AIS A	BP: 108/66–170/76 mmHg	Two episodes of ventricular fibrillation leading to cardiac arrest but no further episodes since defibrillator was implanted
Ho and Krassioukov; 2010[41]	45	C5 AIS A	BP: 220/105 mmHg HR: 50/minute	Resolution of myocardial ischemia with no subsequent evidence of coronary artery occlusion

SCI, spinal cord injury; CV, cardiovascular; BP, blood pressure; HR, heart rate; AIS, ASIA (American Spinal Injury Association) Impairment Scale; mo., months.

Table 3 Pulmonary events associated with AD

Author; year	Age	SCI information	CV parameters	Outcome
Kiker *et al.*; 1982[42]	32	C6–C7 SCI	BP: 162/110–170/118 mmHg HR: 88–100/minute	Resolution of AD-induced pulmonary edema
Calder *et al.;* 2009[43]	50	C5 AIS B	BP: 154/111 mmHg HR: 160/minute	Death following pulmonary edema and asystole

SCI, spinal cord injury; CV, cardiovascular; BP, blood pressure; HR, heart rate; AIS, ASIA (American Spinal Injury Association) Impairment Scale.

Table 4 Analysis of BP and heart rate as reported in the literature

Death	*N*	Variable	Median	Mean	Minimum	Maximum	Standard deviation
0	25	SystBP	180	180.62	100	220	34.76
		DiastBP	100	103.24	70	140	17.63
		HR	82	88.36	46	180	39.62
1	7	SystBP	225	213.67	154	268	40.54
		DiastBP	120	132.67	90	185	36.72
		HR	102	106	56	160	52.12

subsequently led to further life-threatening complications or death. These episodes were primarily documented and described as case reports in the literature. Two cases were mentioned within larger manuscripts, without specific descriptions of case details.[6,21] Findings indicate that out of 32 available cases describing AD associated with a potentially life-threatening outcome or death, 23 (72%) were CNS-related, 7 (22%) were CV-related and 2 (6%) were pulmonary-related. CNS events – particularly hemorrhage and ischemic attacks – were most extensively described in the literature. The most serious outcome, death, occurred in seven (22%) documented cases of AD. These deaths were most commonly due to intracranial

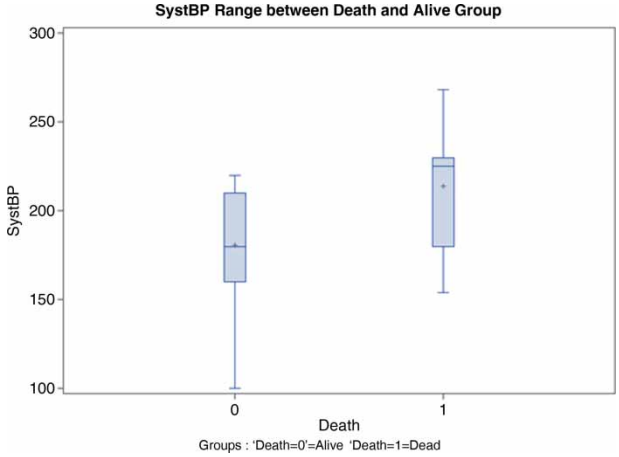

Figure 2 Box plot showing higher mean SBP in the group of patients who resulted in death as the final outcome. However, subsequent analysis did not show statistical significance ($P = 0.0584$).

hemorrhage, which caused fatality in five cases, in addition to one case of status epilepticus and one case of pulmonary edema. Although most surviving individuals experienced prompt resolution of AD after removal of the triggering stimulus and appropriate management, in some individuals, AD has been documented to protract for several days.[44]

While recognition and treatment of chronic SCI and its complications are continuously improving, mortality in individuals with SCI is still elevated when compared with their able-bodied counterparts.[14] Until recently, the correlation between chronic SCI and an increased risk of stroke has been hypothesized, but not thoroughly investigated.[17] In a nationwide Taiwanese cohort of individuals with SCI, Wu *et al.*[17] demonstrated an increased risk of stroke, in particular, ischemic stroke. In our review, we noted that CNS-related events were by far the most extensively discussed cases in the literature when associated with AD. These cases consisted of complications including hemorrhage, ischemia, and seizures. Death itself occurred most commonly when associated with CNS-related complications, accounting for six out of seven deaths that were noted in this review. Five such deaths were related to hemorrhage, whereas one was documented during an episode of status epilepticus.

The association between chronic SCI and CV disease has been extensively discussed in the literature.[14–16] Further, it has been shown that CV disease is the most common cause of death in chronic SCI when considering underlying and contributing causes together.[14] Although we did not find any cases describing CV events associated with AD that subsequently led to mortality, it should be noted that AD has been known to present asymptomatically.[45] Asymptomatic AD was also reported in one of our studies that evaluated CV responses in individuals with SCI during a vibrostimulation for sperm retrieval.[7] Electrocardiogram (ECG) abnormalities were present in 11 out of 13 subjects with SCI undergoing a vibrostimulation procedure, whereas 10 out of the 13 developed AD.[7] The majority of these individuals remained unaware of their arrhythmias and ECG abnormalities, nor did they report symptoms characteristic of AD.[7] One case, reported by Trønnes and Berg,[46] described a 10-month-old male with SCI who experienced cardiac arrest. After reviewing the details of this case, we concluded that the unique presentation described in the case was due to a vagally-induced bradycardia that is typically observed in the acute period of SCI. Because the BP documented in this case would not be considered elevated in a normal 10-month-old child, this case was excluded from analysis because we believe that this death was not AD-related.

In addition, clinicians should be aware that AD can be self-triggered intentionally by athletes with SCI in an attempt to level the playing field during competitions such as the Paralympics.[47] The increase in BP that accompanies AD within a laboratory environment has been shown to enhance athletic performance.[48] However, considering the life-threatening nature of this practice, known as "boosting", athletes are placed at a much higher risk for CV disturbances and the International Paralympics Committee has banned this practice during competition.[49] Pulmonary complications are also among the leading causes of death in the chronic SCI population.[14,50] However, these pulmonary conditions are primarily accounted for by pneumonia and influenza.[50] Pulmonary complications secondary to AD are poorly described in the literature, accounting for a total of 2 (6%) out of 32 cases that we found documenting life-threatening sequelae following AD attacks. Both of these cases were described as pulmonary edema, of which one case resulted in death. Both cases of pulmonary edema were neurogenic in etiology, suggesting that an insult to the CNS may be a possible underlying cause.[42,43] Further studies in this area are necessary to elucidate the mechanism and appropriate treatment of this poorly described complication secondary to AD.

We also acknowledge several limitations in our study due to the nature of this type of review. The data presented in the review are based only on the published cases of life-threatening episodes of AD. From personal clinical experience and communication with other colleagues in the SCI community, the authors are aware

that the incidence of the life-threatening episodes of AD and related death is probably much higher than the literature suggests. Not every case of AD with serious life-threatening consequences for individuals with SCI will be reported.

Statistical analysis of the retrieved data (Fig. 2) revealed that the mean SBP in the group of cases that resulted in fatality was higher than in the comparison group where individuals survived from their AD attacks. However, a two-sample *t*-test between the two groups failed to show a significant difference between the reported SBPs. It should be noted that the sample size for this review was relatively small ($N = 32$), and only seven cases resulted in death as an outcome. Furthermore, five case reports neglected to state a measurement of BP at any point during the documented AD episodes. Even within the cases that reported measured values of BP, we cannot be certain that these values were measured at the same time relative to their respective episodes of AD. Further studies with larger sample sizes are necessary to show a distinctive link between AD-related death and BP.

Although the complications and consequences of chronic SCI have been well described in the literature,[7,14,17,50] there is little information regarding complications associated with AD specifically. AD is a life-threatening complication of chronic SCI, but death as a final outcome is often due to subsequent conditions such as hemorrhage,[19,20,25,30,35] seizure,[6] or pulmonary edema.[43] The CNS and CV-related sequelae of AD have been described by several articles, but the same cannot be said of complications regarding the pulmonary system. To the best of our knowledge, only two documented cases of neurogenic pulmonary edema secondary to AD have been described and the mechanism of this complication is still poorly understood. Further investigations should aim to gain a better understanding of neurogenic pulmonary edema associated with AD, as well as enhancement of management strategies to reduce mortality secondary to AD-associated CNS complications.

Finally, we also have to keep in mind the fact that there is evidence from the literature that suggests that individuals with SCI consider regaining autonomic functions and elimination of AD among their highest priorities for recovery following injury.[51]

Conclusion

AD is a well-known consequence of SCI among individuals with high thoracic and cervical injuries. Many of these individuals experience this condition on a daily basis. Medical personnel, caregivers, and individuals with SCI should be aware about the timely diagnosis and management of this life-threatening condition that can result in a variety of significant complications including stroke, seizures, myocardial ischemia, and death. Despite the paucity of published cases with life-threatening consequences and death following AD in the chronic SCI population, it is well known that the leading causes of morbidity and mortality in this population are due to CV-related diseases.[14,16] Despite the presence of guidelines on the management of AD,[52] the authors feel that there is a need for a national survey which could help to determine the current practices regarding management of episodes of AD by medical personnel in various settings. This could also allow us to develop some minimum requirements for the standard reporting of AD and eventually develop

Table 5

Take home messages

What is AD?

AD is episodic hypertension that is characterized by collection of signs and symptoms that commonly occurs in individuals with SCI above the T6 spinal segment. BP rises by more than 20 mmHg above baseline, but may or may not produce symptoms. AD can happen at any time after SCI.[54]

Who suffers from AD?

AD occurs in up to 90% of individuals with SCI above the T6 neurological level.[55]

Signs and symptoms

Signs – diaphoresis, cardiac arrhythmias, bradycardia (however, tachycardia may occur), piloerection, BP elevated ≥20 mmHg above baseline.[56]

Symptoms – severe headache, anxiety, flushing above the level of injury, blurred vision, dry and pale skin below the level of injury, nasal congestion.[56]

What are the common triggers?

AD can be triggered by both noxious and non-noxious stimuli. The most common trigger is bladder distension. However, renal stones, urinary catheter blocks, urinary tract infections, pressure sores, and sexual intercourse are also known triggers. Additionally, seemingly benign triggers include sunburns and tight shoelaces.[55]

Management of AD

1. Sit the patient upright
2. Loosen or remove any form of clothing that may be restrictive
3. Monitor BP and heart rate every 5 minutes
4. Consider possible triggers of AD
5. If SBP remains above 150 mmHg, administer a short-acting antihypertensive such as nifedipine or captopril.[52]
6. Consider other possible triggers of AD (such as bowel).

Prevention

Bowel and bladder routines should be appropriately managed to reduce the likelihood of triggering AD. The initiation of any medical procedure should be prefaced by a consultation with a SCI specialist.[55]

Education

AD is a life-threatening condition that requires urgent and appropriate medical attention!

Individuals with SCI, predisposed to development of AD (SCI at T6 and above) and their caregivers should be educated about AD and carry a pocket PVA information card.[57,58]

Knowledge on management of episodes of AD has to be transferred outside of the rehabilitation hospitals to emergency rooms, family doctors' offices, and paramedic personnel.

an AD dataset similar to existing SCI datasets for other conditions.[53] Key information for clinicians caring for individuals with SCI is summarized in Table 5.[55–58]

Further research in this area is necessary to better understand the pathophysiology of these complications, as well as what steps are necessary in order to reduce fatality among individuals with SCI.

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