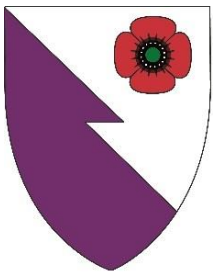
The Royal College of Emergency Medicine

Best Practice Guideline

Guidelines for the Management of Excited Delirium / Acute Behavioural Disturbance (ABD)



Independent Advisory Panel on
DEATHS IN CUSTODY

May 2016

Summary of recommendations

1. RCEM publications must have been subject to the review and approval processes as described within the Terms of Reference of the relevant committee, including peer and lay review.
2. Excited Delirium / Acute Behavioural Disturbance (ABD)[1] is a medical emergency – affected individuals may suffer sudden cardiovascular collapse and/or cardiac arrest with little or no warning.
3. Patient restraint time in ABD should be kept to an absolute minimum - the degree of restraint used must be justifiable, reasonable, for the minimum time necessary and proportional to the situation.
4. Sedation should be with intravenous benzodiazepines, antipsychotics or ketamine. If the intravenous route is not immediately available then intramuscular administration should be used.
5. Early and aggressive management of hyperthermia and acidosis should be instituted and a high index of suspicion for the development of rhabdomyolysis and Disseminated Intravascular Coagulation (DIC) should be maintained.

Scope

To provide a guideline for Emergency Departments to safely and effectively manage adults who attend with Excited Delirium / Acute Behavioural Disturbance (ABD).

Reason for development

The National Institute for Clinical Excellence (NICE) have developed guidelines entitled "Violence and aggression: short term management in mental health, health and community settings"[1]. The NICE guidelines focus on prevention, recognition and reducing the risk of violence, but does not deal in detail with Excited Delirium / ABD which is a very specific presentation of violence and aggression carrying significant clinical risk.

Introduction

Acute Behavioural Disturbance (ABD) is the accepted terminology adopted by the UK Police Forces, the Ambulance Services and the Faculty of Forensic and Legal Medicine[2]. It describes the sudden onset of aggressive and violent behaviour and autonomic dysfunction, typically in the setting of acute on chronic drug abuse or serious mental illness. However, there is not yet a common standardised definition and its incidence has not been clearly quantified.

ABD, or as it is also known 'Excited Delirium,' is the presentation of features of "acute delirium" and hyper-adrenergic autonomic dysfunction and must be considered a medical emergency. Its presentation is associated with sudden death in approximately 10% of cases[3]. High profile deaths of individuals displaying features of ABD have occurred whilst they have been in police custody. This has attracted much media coverage and ABD has become a controversial and emotive illness with significant distress to families involved. The early recognition, intervention and proactive treatment of ABD, with a collaborative response between the Emergency Services (police, paramedics), is likely to result in fewer deaths.

Individuals with ABD most often come into contact with the police initially as they are called to attend due to the individual's sudden onset of bizarre, aggressive and violent behaviour. The presenting behaviour can range

from mildly erratic to a state of extreme agitation and physical exertion. Patients have signs of hyper-adrenergic autonomic dysfunction such as significant tachycardia, marked metabolic acidosis and hyperthermia and these are associated with multi organ failure and death.[3]

The identification of ABD can be challenging clinically as the spectrum of behaviours and signs overlap with many other disease presentations and there is no definitive diagnostic test.

Box 1: Physical symptoms and signs typical of ABD

- Extremely aggressive/violent behaviour
- Excessive strength/continued struggle despite restraint
- Insensitive to pain
- Acute psychosis with fear of impending doom
- Constant physical activity without fatigue
- Hot to touch/profusely sweating/inappropriate state of undress
- Hyperthermia
- Tachypnoea
- Tachycardia

Differential Diagnoses of ABD

- Heat Stroke
- Neuroleptic Malignant Syndrome
- Serotonin Syndrome
- Thyroid Storm
- Sepsis

- Substance intoxication / withdrawal
- Hypoxia
- Hypoglycaemia
- Head Injury / Seizures
- Akathisia

ABD appears to be more common when the weather is warm and humid and deaths more commonly occur in the summer months[4]. Fatalities in ABD typically occur in men in their mid-thirties who have a history of stimulant drug abuse, usually cocaine[6]. Currently the pathophysiology of ABD is not well understood but it is likely to be multi-factorial; furthermore, an individual's pre-disposition to a fatal outcome is also poorly understood.

Many factors have been proposed as contributory to causes of sudden death in ABD such as positional asphyxia secondary to restraint technique, drug toxicity and underlying cardiac disease associated with cardiac arrhythmias. The severe metabolic acidosis associated with ABD is a likely contributing factor in cardiovascular collapse and death[7]. ED physicians must be able to recognise that the presenting signs and symptoms in ABD represent a medical emergency.

Management

The initial aim of management of an individual with ABD should be the rapid tranquilisation and minimisation of their hyper-exertional state. Sedation and de-escalation is of paramount importance in ABD – this reduces the risk of harm to the individual and all involved with their care and enables a full medical assessment to begin with the prompt institution of supportive management.

1. Restraint

Verbal calming and de-escalation techniques maybe be used as the first line intervention in attempting to manage individuals displaying ABD. However, these patients are often highly agitated and aggressive with an altered mental status making their response to de-escalation techniques unpredictable. Physical restraint to facilitate their initial management may be inevitable. This should be kept to a minimum using a level of force that is justifiable, reasonable and proportional to the individual case and rapidly followed by sedation with close monitoring of vital signs[1].

Physical or manual restraint should be viewed as an intervention of almost last resort prior to providing definitive chemical restraint / rapid tranquilisation; prolonged physical restraint must be avoided. Physical restraint has been associated with injuries to patients as well been a contributing factor in patient deaths and particular care must be exercised to ensure that at no time the patient's airway is compromised, this is particularly likely if the patient is kept in a face down position (e.g. due to spitting or biting) with pressure applied on the patient's neck or shoulder

[1] https://www.england.nhs.uk/wp-content/uploads/2015/12/psa-vital-signs-restrictive-interventions-031115.pdf

region. Keeping the patient in a prone position MUST be avoided. It should further be remembered that significant physiological derangements (acidosis, electrolyte abnormalities, cardiac arrhythmias etc.) can occur due to the underlying condition (eg. excited delirium) or as a result of resisting restraint and may be exacerbated by comorbidities (eg. cardiac disease) or medication / illicit substances. If a patient is being restrained in the Emergency Department, even if the police are providing this intervention, ultimate responsibility for the patient's safety and well-being rests with the doctors and nurses of the emergency department.

There is insufficient research on the effects of TASER on ABD however its use as a rapid takedown method to minimise restraint time and activity and allowing expeditious medical intervention may be a necessary alternative once nonphysical methods have failed.

2. Sedation

Sedation (rapid tranquilisation) will be required to facilitate rapid intervention and institution of potentially lifesaving treatments if an individual displaying ABD fails to respond to de-escalation techniques. The rapid control and calming of an individual displaying the extreme physical exertion associated with ABD is essential to prevent further worsening of their metabolic status[3]. Ideally sedation should be administered via the intravenous route however this route is unlikely to be immediately available.

The clinician must therefore make a decision regarding the safety of the patient (minimizing duration of restraint) and team (avoiding both physical and needlestick injury) as to whether it is better to attempt cannulation (accepting the difficulty of the procedure in an uncooperative patient) or whether to administer an intramuscular agent of sufficient strength to allow rapid control of the patient followed by cannulation and monitoring in a high dependency area. It is important to remember that the absorption of IM medication can occur far more rapidly when an individual is agitated or physically overactive. It should also be remembered that individuals displaying ABD may well need much higher doses of sedative agents than are typically required or recommended[12].

There are currently three groups of agent used for sedation in ABD - benzodiazepines, antipsychotics and ketamine (see appendix 2). However, there is a paucity of high quality evidence in the medical literature to determine the most suitable single agent or combination of agents.

a. Benzodiazepines are the most commonly used sedative agents in the ED and are familiar to all ED physicians. They generally have a wide safety profile and have multiple routes of administration. However, there is variability in the dose response relationship between individual patients and this often necessitates active titration – this may be undesirable in ABD where rapid and predictable sedation is essential. There is a relatively slow, and often unpredictable, onset time when benzodiazepines are given IM and this route is more commonly associated with adverse events. However, IM lorazepam is recommended by NICE as the first line agent for use in rapid tranquilisation for the short- term management of violence and aggression[1]. The most common serious adverse effect with benzodiazepine use is respiratory depression which may exacerbate the acidosis. These may be compounded by alcohol or other hypnotics already consumed by the individual – although the acute intoxication of these agents in individuals with ABD is thought to be uncommon. The use of benzodiazepines has the advantage that a reversal agent exists (Flumazenil).

b. Ketamine has many properties that make it a useful sedative agent in the management of ABD. It has a very rapid onset of action when administered IV or IM and has a wide therapeutic window producing consistent effects at predictable doses. Ketamine protects airway reflexes and increasing doses lead to more prolonged duration of sedation whilst rarely affecting respiratory drive[9].

However, ketamine does inhibit the reuptake of catecholamines leading to the potential for sympathomimetic side effects such as an increase in heart rate, blood pressure, cardiac output and myocardial oxygen consumption. There is, therefore, the theoretical risk of worsening any cardiovascular instability present in ABD. Ketamine may be associated with unpleasant emergence phenomenon, although this is readily managed by the administration of benzodiazepines.

c. Antipsychotics are dopamine receptors antagonists and are commonly used for the sedation of agitated psychiatric patients. Anti-psychotics offer the theoretical pharmacological benefit in the management of ABD due to their dopamine receptor antagonism, as one of the postulated causes of ABD is excess dopamine levels[10]. Traditional neuroleptics such as haloperidol have well recognised side effects such as prolongation of the QTc interval and cardiac arrhythmias. NICE does recommend the use of haloperidol with promethazine for rapid tranquilisation in the management of violence and aggression but only if the patient has taken antipsychotic medication previously or they have previously had an ECG[1]. However, this background information for an individual with ABD is unlikely to be available at the time of their initial presentation.

All antipsychotics can lower the seizure threshold, have anti cholinergic effects, precipitate acute dystonic reactions and may rarely lead to neuroleptic malignant syndrome. Antipsychotics such as droperidol, or the newer atypical agents such as olanzapine, have been shown to be more effective sedating agents than midazolam with less adverse events[11, 12]

Whichever sedative agent is chosen, it must be one that the treating ED physician is familiar with. Full patient monitoring in line with the RCEM guidance on safe procedural sedation, including $EtCO_2$ monitoring, must be used in all cases in which sedation is administered if possible. Early involvement of other specialties such as anaesthetics should be considered.

3. Supportive Management in hospital

Procedural sedation should be followed by a rapid assessment of the patient. This should consist of a thorough physical examination with documentation of the individual's temperature, standard laboratory investigations (including CK and coagulation profile) should be sent and an ECG performed. Arterial blood gas analysis is essential– this will typically show a severe metabolic acidosis with low $PaCO_2$ as the individual with ABD will be tachypnoeic in an attempt to physiologically correct their lactic acidosis[7].

Other investigations such as further imaging (e.g. CT) will be dependent on each individual case. A collateral history should also be obtained to search for the possible causes of ABD with subsequent tailoring of management.

Hypovolaemia is common in ABD, attributable to the excess physical activity and hyperthermia, so all patients should receive IV crystalloids[9]. These supplementary IV fluids will help to correct metabolic acidosis and prevent end organ damage. The use of sodium bicarbonate to specifically treat metabolic acidosis in the absence of hyperkalaemia in ABD is not recommended - it may exacerbate intracellular acidosis and have a negative inotropic effect on an ischaemic myocardium[13].

Hyperthermia is common in individuals presenting with ABD. This is due to a combination of the individual's state of constant physical exertion, which itself will generate heat, and hyperthermia due to dopamine dysfunction. Hyperthermic patients need to be cooled to standard body temperature (and not below). This should be achieved with the institution of basic cooling methods such as the removal of clothing and placing the patient in a cool environment. Active cooling with cooled intravenous fluid, ice packs in the axilla and groins should be used as required[9].

Patients should be closely monitored for signs of the development of rhabdomyolysis, hyperkalaemia and DIC, all having been reported in cases of ABD[10]. The provision of standard care such as urinary alkalinisation with sodium bicarbonate for rhabdomyolysis is advocated.

Ongoing management such as need for organ support and Intensive Care will be dependent on each individual case and each individual with ABD must have their medical needs met fully before usual police proceedings for any criminal activity begin.

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Review

Usually within three years or sooner if important information becomes available.

Conflicts of Interest

None

Disclaimers

The College recognises that patients, their situations, Emergency Departments and staff all vary. This guideline cannot cover all possible scenarios. The ultimate responsibility for the interpretation and application of this guideline, the use of current information and a patient's overall care and wellbeing resides with the treating clinician.

Research Recommendations

None identified

Audit standards

There should be a documentation and audit system in place within a system of clinical governance.

Key words for search

Acute Behavioural Disturbance (ABD), Excited Delirium, management, guideline

Appendix 1

Methodology

Where possible, appropriate evidence has been sought and appraised using standard appraisal methods. High quality evidence is not always available to inform recommendations. Best Practice Guidelines rely heavily on the consensus of senior emergency physicians and invited experts.

Evidence Levels

1. Evidence from at least one systematic review of multiple well designed randomised control trials
2. Evidence from at least one published properly designed randomised control trials of appropriate size and setting
3. Evidence from well-designed trials without randomisation, single group pre/post, cohort, time series or matched case control studies
4. Evidence from well-designed non experimental studies from more than one centre or research group
5. Opinions, respected authority, clinical evidence, descriptive studies or consensus reports.

Appendix 2

Rapid Tranquilisation in ABD: please consult formulary

Medication	Route	Typical dose (mg)	Onset (min)	Duration (min)
Midazolam	I/N	5	3-5	30-60
	IM	5	10-15	120-360
	IV	2-5	1-5	30-60
Lorazepam	IM	4	15-30	60-120
	IV	2-4	2-5	60-120
Diazepam	IM	10	15-30	15-60
	IV	5-10	2-5	15-60
Haloperidol	IM	10-20	15-30	180-360
	IV	5-10	10	180-360
Droperidol	IM	5	10-30	120-240
	IV	2.5	10	120-240
Olanzapine	IM	10	15-45	
	IV (unlicensed)	5		
Ketamine	IM	2-4mg/kg	3-5	60-90
	IV	1-2mg/kg	1	20-30

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