Original article

Anal incontinence and severe acquired brain injury: a retrospective study of 347 rehabilitation inpatients

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Abstract: Patients with ABI often present, from the outset, problems with intestinal function such as anal incontinence and difficulty in defecating, in a clinical picture termed Neurogenic Bowel Dysfunction. The aims of the study are to evaluate the incidence of neurogenic anal incontinence at the beginning and end of the intensive rehabilitation period after ABI due to trauma, haemorrhage, anoxia or neoplasm; to evaluate any correlation between its progression and the duration of coma, site of the encephalic lesion, occurrence of paroxysmal sympathetic hyperactivity, presence of diffuse axonal injury (DAI), duration of tube feeding, duration of hospitalisation and discharge setting, incidence since the acute event of healthcare-related infections, in particular Clostridium Difficile infections, and concomitant urinary incontinence when the patient is discharged. At discharge from Neurorehabilitation Unit, only the presence of frontal lesions seems to correlate with persistent faecal incontinence.

Keywords: Acquired Brain Injury; Anal Incontinence; Frontal lobe; Neurogenic Bowel Disease.

INTRODUCTION

Acquired Brain Injury (ABI) may be secondary to a vascular, anoxic or neoplastic injury and is characterised by the onset of coma of variable duration (Glasgow Coma Scale ≤ 8) and the resulting motor, sensory and cognitive impairment[1].

The progression of ABI can be characterised as the passage from coma to Unresponsive Wakefulness Syndrome (UWS), which has replaced the former term 'vegetative state', with its potentially negative connotations.

The patient with UWS presents with eyes open and autonomous functions (cardiovascular control and thermoregulation) intact, even without environmental contact. Emergence from UWS characterises Minimally Conscious State (MCS), defined in 2002 by the Aspen Work-group as a condition in which minimal, but definite, behavioural evidence of self or environmental awareness is demonstrated[2].

Patients with ABI often present, from the outset, problems with intestinal function such as anal incontinence and difficulty in defecating, in a clinical picture termed Neurogenic Bowel Dysfunction (NBD)[3].

Neurogenic anal incontinence is defined as the involuntary loss of faeces (solid or liquid) and gas from the rectum, secondary to neurological pathology[4]. The literature contains few studies describing anal incontinence in ABI; varying incidence is described, from 20% to 70% in the acute phase[5-7], persisting in 12% of cases until the patient is discharged from rehabilitation[5,8], and varying between 2% and 20% at six and twelve months from the acute event[5-7].

It is notable that in 20% of cases, in the chronic phase, persistent constipation occurs in these patients. It is also important to emphasise that, in some cases, faecal incontinence is in reality secondary to constipation even in the acute phase, in particular due to the difficulty of managing constipation pharmaceutically and of device-based treatment in patients with impaired consciousness (and the risk of over-treatment), or the occurrence of pseudo-incontinence due to obstruction by a faecaloma.

In these cases, the consistency of stool, as well as the involuntary loss, can help to establish a working differential diagnosis[9].

The aims of the study are to evaluate the incidence of neurogenic anal incontinence at the beginning and end of the intensive rehabilitation period after ABI due to trauma, haemorrhage, anoxia or neoplasm; to evaluate any correlation between its progression and the duration of coma, site of the encephalic lesion, occurrence of paroxysmal sympathetic hyperactivity, presence of diffuse axonal injury (DAI), duration of tube feeding, duration of hospitalisation and discharge setting, incidence since the acute event of healthcare-related infections, in particular Clostridium Difficile infections, and concomitant urinary incontinence when the patient is discharged.

MATERIALS AND METHODS

Retrospective evaluation was made of 497 patients, admitted consecutively to the Neurorehabilitation Unit in Cuneo following ABI, between January 2000 and December 2013, of whom 347 were male and 150 female, with an average age of 46.5±22.3 years. Patients who presented associated spinal cord injury (11 patients) were excluded, as were those with previous faecal incontinence (4 patients). Also excluded were 135 patients having a value ≤ 3 on the Level of Cognitive Functioning (LCF) Scale[10]. The LCF, including the modified LCF-R version, is a "process scale", that is, it evaluates patient behaviour and thus the patient's cognitive level from the moment he or she goes into coma until recovery; it tracks the patient throughout the process, from intensive care to intensive and then extensive rehabilitation, through to social rehabilitation. It can functions as the instrument for patient evaluation across different units, e.g. it can also be used in the intensive care unit.

LCF is a scale of observation in context, and not a neuropsychological test score. It allows the whole rehabilitation team (nurse, logopedist, occupational therapist, physiotherapist, clinician, psychologist, neuropsychologist, etc.) to use the same metric, since it is not specific to any individual professional. It therefore enables a "dialogue" between all members of the team.

This retrospective cohort study was conducted at an academic, urban, tertiary care hospital. Data were collected from electronic medical records and supplemented with chart review.

The Neurorehabilitation Unit in the Department of Rehabilitative Medicine accepts clinically stabilised patients

with a diagnosis of traumatic brain injury or non-traumatic brain injury. The only criterion precluding access to the Unit is mechanical ventilation. Patients with a tracheotomy tube or percutaneous endoscopy gastrostomy (PEG) tube are accepted and there are no time limits with respect to the acute event, although the earliest possible access is guaranteed.

A detailed medical history was collected for all patients. A physical medicine examination and neurological examination were carried out every week and routine blood tests and, where necessary, radiological and neuroradiological investigations were carried out every month.

The rehabilitation programme for these patients includes the provision of optimal nutrition, control of infections, management of bladder, bowel and autonomic disorders, provision of specialist seating and control of posture and tone problems. Patients underwent one hour of physical therapy treatment and one hour of speech therapy every day, to prevent tertiary injury. Rehabilitative treatment involved passive joint mobilisation and helping/placing patients into an upright sitting position on a tilt table.

All the patients were evaluated upon entry as to their bladder and bowel voiding, combining clinical observations with items specific to related activities contained within the Functional Independence Measure (FIM).

Clostridium Difficile and multi-resistant bacteria were routinely tested for using rectal swabs, even in asymptomatic patients; in cases of fever, increased leucocytosis with neutrophilia, and elevated C-reactive Protein (CRP) and procalcitonin, repeated blood cultures were performed.

The occurrence of neurogenic anal incontinence upon discharge from neurorehabilitation was verified, seeking any correlatable factors such as the pathogenesis of the ABI, duration of coma, site of the encephalic lesion, occurrence of paroxysmal sympathetic hyperactivity, presence of DAI, duration of tube feeding, duration of hospitalisation and discharge setting, incidence since the acute event of healthcare-related infections, in particular Clostridium Difficile infections, and concomitant urinary incontinence upon patient discharge.

STATISTICAL ANALYSIS

Kruskall-Wallis analysis of variance with Bonferroni correction for post-hoc comparisons and Mann-Whitney tests were used to compare ordinal and non-normally distributed continuous variables. Categorical data were analysed by χ^2 test and Fisher's exact test, correlations were checked with Spearman's rho. A correlation matrix was used to determine variables affecting neurogenic anal incontinence, and multiple regression analysis was used to determine the effect of neurogenic anal incontinence on discharge rehabilitation outcome. For a better understanding, data of continuous variables were expressed as means ± SD. The level of significance for all tests was set as $p < 0.05$.

RESULTS

Of the 347 patients included in the study (208 male and 139 female), the ABI was post-traumatic in 111 (31.9%), post-haemorrhagic in 180 (51.9%), and post-anoxic in 56 (16.2%). The average length of stay in acute care units was 28.5 days; the average length of coma was 7.4 days for post-traumatic patients, 9.1 for post-haemorrhagic patients and 15.4 for post-anoxic patients. 51% presented signs of DAI, with localised frontal lobe lesions, unilateral or bilateral, in 32% of cases.

Upon their admission to rehabilitation, faecal incontinence was detectable in 244 patients (70%); all patients were being fed by percutaneous gastroenterostomy or nasogastric tube and treatment continued for 55 days on average. All patients were fitted with a permanent urinary catheter upon admission; lesions of the pelvic girdle were present in 29 patients; in 69 cases (19.8%), paroxysmal sympathetic seizures were indicated in the acute phase.

Table 1 summarises the main demographic and clinical characteristics of the sample upon admission to neurorehabilitation.

Upon admission, the only variables seeming to display statistically significant correlation with the presence of faecal incontinence are the number of previous healthcare-related infection episodes and previous Clostridium Difficile infections (Table 1).

During their hospitalisation, 17 patients died and 21 were transferred back to acute care units due to worsening of their clinical condition. Upon discharge, faecal incontinence was present in 56 of the 309 remaining patients, down from 70% to 18.1%.

Those patients still suffering incontinence were either discharged to their own homes (11 patients), transferred to other rehabilitation facilities (19) or transferred to care homes (26) (p = n.s.).

With regard to the variables taken into account, the LCF scale values upon admission do not seem to affect the persistence of faecal incontinence upon discharge; the same is true of the duration of coma, average duration of tube feeding, presence of diffuse axonal injury, lesions of the pelvic girdle, paroxysmal sympathetic hyperactivity and healthcare-related infections.

At discharge from Neurorehabilitation Unit, only the presence of frontal lesions seems to correlate with persistent faecal incontinence (Table 2).

DISCUSSION

NBD is common in patients suffering medullary lesion, myelomeningocele, multiple sclerosis, Parkinson's disease, and stroke. In particular, suprapontine neurological pathologies can cause changes to supraspinal control mechanisms, leading to the onset of constipation and/or faecal incontinence.

The occurrence of NBD following neurological illness is linked to reduced quality of life[3], reduced social interaction and significant economic impact, both on the person affected and on healthcare services[11-14].

TABLE 1. Demographic and clinic characteristics at rehabilitation admission (n=347)

		Continent	Incontinent	
sex	female (n=139)	42	97	p=n.s.*
	male (n=208)	61	147	
Mean age ± SD	female (n=139)	45.3±15.4	42.1±19.2	p=n.s.*
	male (n=208)	47.2±10.2	44.8±9.8	
Lenght of coma	traumatic (n=111)	7.8±5.4	7.1±6.3	p=n.s.*
(days±SD)	hemorrhagic (n=180)	10.8±3.4	8.3±4.7	
	anoxic (n=56)	17.8±3.9	13.7±4.4	
Acute lenght of stay (days±SD)		30,4±28.6	32,6±20.3	p=n.s.**
Frontal lobe injury	present	35	87	p=n.s.*
	absent	68	157	
Diffuse axonal injury	present	67	135	p=n.s.*
	absent	36	109	
Pelvic Injury	present	12	17	p=n.s.*
	absent	91	227	
Paroxysmal sympathetic	present	29	60	p=n.s.*
hyperactivity	absent	74	184	
Tube feeding (days ± SD, acute phase)		16,4±2.6	14,2±7.3	p=n.s.**
Clostridium Difficile infection (n. of episodes, acute phase)		8	32	p=0.008*
Hospital-acquired infections (≥ 3 episodes, acute phase)		29	88	p=0.029*

* Fisher's exact test - ** Unpaired t-test

TABLE 2. Clinic characteristics at rehabilitation discharge (n=309)

		Continent	Incontinent	
Mean age ± SD	female (n=119)	103	16	p=n.s.*
	male (n=190)	150	40	
Lenght of coma	traumatic (n=99)	7.2±3.9	7.7±5.2	p=n.s.*
(days±SD)	hemorrhagic (n=177)	10.7±3.4	9.6±2.9	
	anoxic (n=33)	14.5±5.6	16.7±2.6	
Inpatient Rehabilitation lenght of stay (days±SD)		95.4±38.6	102.5±23.9	p=n.s.**
Frontal lobe injury	present	110	49	p<0.0001*
	absent	143	7	
Diffuse axonal injury	present	138	24	p=0.139*n.s.
	absent	115	32	
Pelvic Injury	present	118	20	p=0.139*n.s.
	absent	135	36	
Paroxysmal sympathetic hyperactivity	present	129	32	p=0.460*n.s.
	absent	124	24	
Clostridium Difficile infection (n. of episodes, acute + inpatient rehabilitation phase)		57	20	p=0.059*n.s.
Hospital-acquired infections (n. of episodes, acute + inpatient rehabilitation phase)		324	68	p=0.057***n.s.
Tube feeding (days ± SD, acute + inpatient rehabilitation phase)		57.2±19.6	62.3±15.6	p=n.s.**
Discharge disposition	home	87	15	p=0.168*n.s.
	other rehabilitation	92	20	
	institutional care	74	21	

* Fisher's exact test - ** Unpaired t-test - *** Kolmogorov-Smirnov test

However, the incidence of NBD resulting from an ABI is still to be determined and healthcare professionals have paid no particular attention to its occurrence in patients suffering from serious changes in their state of consciousness, with the result that research activity has been limited, in particular with regard to therapeutic strategies.

Our case study seems to highlight a single significant element in the persistence of faecal incontinence upon discharge from rehabilitation, namely the presence of frontal lesions.

Together with the peripheral control of intestinal function, various cerebral areas compromised by traumatic and vascular encephalic pathology can cause the partial or complete loss of sensory and motor function in the anorectal tract and pelvic floor[15].

This leads to changes in colonic motility and affects the absorption of electrolytes and water, clearly contributing to change in the normal mechanisms for propulsion of the faecal bolus and for defecation[16,17].

Completing the picture of NBD resulting from ABI are the associated motor and cognitive disabilities. Although these are not always present, they nonetheless represent a major problem for those patients in which they do occur.

Various parts of the central nervous system contribute to the control of gastrointestinal function, such as the limbic system, hypothalamus, periaqueductal grey matter and amygdala.

Information reaches the encephalic centres through the spinomesencephalic and spinotelencephalic tracts, together with parasympathetic and sympathetic projections through the solitary tract nucleus.

Within the reticular substance of the cephalic trunk there are also close connections between gastrointestinal function and certain haemodynamic reflexes (for example, the decrease in cardiac contractility induced by gastric distension), just as the cerebellum seems able to influence gastric and intestinal motility.

At the cortical level, the perception of rectal distension is predominantly in the right prefrontal cortex[18]; the limbic cortex in turn modulates gastrointestinal function through the pathways descending to the dorsal nuclei of the vagus nerve: its stimulus determines gastric distension and the reduction of gastrointestinal motility.

A number of recent studies have finally shown how cortical influences in emotional terms can affect the behaviour of the gastrointestinal tract in inflammatory disease[19]. However, despite all these observations, we still cannot be certain of the effects on colorectal physiopathology following a stroke.

The association between faecal incontinence and frontal lesions therefore has particular foundation in cases of traumatic ABI, where the frontal cortex is usually involved[20], just as the limbic system is involved in all forms of ABI, whether primarily from the lesion or secondarily in the post-acute phase.

However, our observations do not seem to confirm the data linking persistent anal and urinary incontinence with cognitive functional deficit[21] and modest functional recovery[22]: the LCF value upon discharge does not differ significantly between the two groups (continent and incontinent).

The brain mapping confirms, however, that the prefrontal cortex normally controls continence via connections with other areas, as well as executive functions, damage to which causes changes in cognitive and behavioural functions[23]. The data relating to the discharge setting also seem to confirm the lack of correlation between persistent faecal incontinence and cognitive-behavioural deficit; there is in fact no statistically significant difference between the discharge settings of the two populations (continent and incontinent). This all points toward an interpretation of persistent faecal incontinence as secondary to frontal lobe impairment rather than to the cognitive-behavioural effects caused by such impairment.

Finally, it remains to point out some clinical situations that are possible causes of iatrogenic diarrhoea and therefore correlatable to healthcare activity; it is often impossible to evaluate precisely the connection between diarrhoea and faecal incontinence in patients with impaired consciousness.

Clostridium Difficile is the microorganism most easily identified as a cause of nosocomial diarrhoea[24]. In our case study, the number of Clostridium Difficile infection episodes in the acute phase alone correlates positively with the presence of faecal incontinence upon admission into rehabilitation, while the total number of Clostridium Difficile infection episodes (acute phase and hospital rehabilitation phase) does not correlate with persistent faecal incontinence upon discharge.

Similarly, diarrhoea and the presence of loose faeces can commonly be secondary to tube feeding via nasogastric tube or PEG, or to antibiotic treatment for the frequent nosocomial infections[25]. These elements combined make difficult the diagnostic interpretation of faecal incontinence in the acute phase, but again, our case study shows no apparent correlation with the duration of tube feeding, upon either admission to, or discharge from the rehabilitation unit.

Another element of our study that does not match previously presented data[5] is the lack of any correlation between persistent faecal incontinence upon discharge from inpatient rehabilitation and pelvic girdle fractures.

In conclusion, from our observations, a correlation emerges between unilateral or bilateral prefrontal lobe damage and persistent faecal incontinence upon discharge from rehabilitation, although there was no corresponding correlation with incidences of cognitive-behavioural impairment.

The most obvious limitations of this study arise from the retrospective evaluation of the data, which are often incomplete as to any incidence of faecal incontinence prior to the acute event. Similarly, our observations may be affected by the non-correlation with the feeding arrangements.

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Comment

This study effectively points out that bowel dysfunctions causing fecal constipation and/or incontinence are not only secondary to a spinal cord injury (SCI), but also constitute a major clinical and Quality of Life issue, even after a brain injury in its acute phase. Unlike SCI, where the percentage of patients with no sphincter control remains unvaried, even in later stages, that is during neurorehabilitation and after discharge, in patients with ABI this percentage significantly decreases, as shown in this study, where it drops from 70% at admission to 18% at discharge. Anyway, it is still a considerably high percentage and a more effective therapeutic rehabilitation approach is desirable, if compared, it has to be said, to what is currently proposed for the management of these disorders in our rehabilitation or long-term care units.

The correlation between persistent fecal incontinence and frontal lesion is also very thought-provoking. It is as if patients are losing what they acquired in their first two or three years of life, when they learned to control their pelvic floor and sphincter muscles, so that, with equal efficiency, they can hold the content of bowel and bladder when social conditions requires it, and expel it when they decide to. In fact, it is a complex learning (we learn to walk and talk first!) that is compromised by neurological deficits, ranging from more peripheral lesions (pudendal nerve neuropathies, dyssynergies and pelvic floor myopathies) to frontal lobe impairment, as this study underlines.

The definition of "no sphincter control" according to the FIM Scale, on the basis of which this study was conducted, does not allow a very sophisticated symptom classification: it does not distinguish between defecation which is indeed physiological, but which occurs at an inappropriate time, and fecal incontinence or soiling, and not even between paradoxical diarrhea and colorectal fecal impaction. Therefore, more prospective studies with a significant follow-up are necessary to return data on this issue, which is absolutely critical both from a clinical point of view and in terms of costs for patient care.

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