Date Received : 30 October 2022 | Accepted: 15 March 2023
Indian Journal of Developmental & Behavioral Pediatrics
(Official Journal of IAP Chapter of Neurodevelopmental Pediatrics)

Self injurious behaviours

Authors
Dr. Ruchi R. Mittal[1], Dr. N. Udayakumar[2], Dr. Nandini Mundkur[3]

1. Fellow in Developmental and Behavioral Pediatrics, Karthikeyan Child Development Unit, Sri Ramachandra Institute of Higher Education and Research, Chennai. 2. Head of Karthikeyan Child Development Unit, Professor of Pediatrics, Sri Ramachandra Institute of Higher Education and Research, Chennai. 3. Director, Centre for Child Development and Disabilities, Bangalore

Correspondence: Dr. Nandini Mundkur, Email: nandinimundkur@gmail.com

Definition: Self-injurious behaviour (SIB) was defined in the original study (Oliver et al., 1987) as repeated, self-inflicted, non-accidental injury producing bruising, bleeding or other temporary or permanent tissue damage, and repetitive behaviours that had the potential to do so if preventative measures were not taken. The study therefore included people whose self-injury was very clear and did not include those people who merely showed stereotyped behaviour. (1) It is included in "Conditions for further study" in DSM-V and is coded in ICD under Intentional self-harm X71-X83.

Examples of self-injurious behaviours: repeated wetting and rubbing of hands in the mouth which causes maceration of the skin, biting or picking of skin resulting in open lesions which may get infected, violent banging of head or other body parts against hard surfaces which may result in fractures, retinal detachments, intracranial hemorrhages and even leading to death. (1)

Defining self-injurious behaviour by using an observable behaviour as a criterion may lead to under-estimation of SIB in young children, thereby having implications on the early interventions and thus future outcomes. (2)

Also, this definition when used in prevalence studies, does not give importance to the form of self-injury (For example: . lip and finger biting in Lesch–Nyhan syndrome; hand biting in fragile X syndrome; skin picking in Prader–Willi syndrome).

Self injury is a major reason for intensive special education usage and hospitalisation thus increasing costs of an individual's care. (3) In addition to this, the presence of SIB increases the risk of family, educational and residential placement breakdowns, restrictive practices in primary care settings and use of psychotropic medications. (4)

Prevalence: Within the total population of people with intellectual disability estimates of the prevalence of self-injury vary from 4 to 24%. (2) Prevalence of SIB in individuals with autism is reported to be as high as 50% which is much higher than for individuals with intellectual disability. (4) The limited data available suggests that self-injury is very persistent. Taylor, Oliver, and Murphy (2011) report approximately 84% persistence over 18 years, Emerson et al. (2001) 71% over 7 years and Cooper et al. (2009), using a definition of self-injury with a high threshold, 62% over 2 years. Also, majority of studies suggest that the prevalence of self-injury increases with age into adulthood and persists for many years. (2) With such high rates of prevalence and persistence, it is very important to understand various aspects of self-injury to help in the intervention and management.

Etiology of SIBs:

Environmental and biological factors are associated with both repetitive and self-injurious behaviours. (3)

Environmental factors:

Results from earlier studies have showed that:

(a) environmental events can evoke and modify SIBs,

(b) self-injury can be reduced by manipulation of existing contingencies,

(c) self-injury can be reduced by the introduction of adaptive behaviours that displace self-injury and

(d) self-injury can be reduced by increasing the non-contingent availability of specific reinforcement. These demonstrations support the argument that self-injury can be influenced significantly, favourably and unfavourably, by the immediate environment. (2)

For example, in individuals with prolonged institutionalization, especially when living in confinement, increase in repetitive and self-stimulatory behaviours has been observed. In children with visual impairment, self-injurious behaviour in terms of applying pressure on the optic globe has been observed. In such cases, increasing the environmental stimulation can decrease the occurrence of SIBs.

High levels of environmental stimulation can lead to anxiety, frustration and stress leading to SIB. Also, children with intellectual disability and ASD have deficits in the ability to convey their experiences which also contributes to the frustration.

Operant learning influences self-injurious behaviours. (2) As with other behaviours, predictable reactions of caretakers to SIB, can reinforce the behaviours. Caretakers may reinforce the behaviour by allowing access to materials or activities that are otherwise restricted or by removing the task demands. In these situations, SIB may be viewed as a learned behaviour that serves as a form of non-verbal communication. (3)

There is also a role for behaviour dysregulation as indicated in literature. Overactivity, impulsivity and repetitive behaviours are associated with higher levels of SIBs. These are behavioural markers for impairments in behavioural inhibition. Behavioural inhibition comprises both the capacity to inhibit prepotent responses to evoking stimuli and the capacity to inhibit a response once emitted. (4)

Biological factors:

Biological factors are also important in predicting the occurrence of self-injurious behaviours. Table 1 lists the biological risk factors for SIBs.

Table 1:

RISK FACTORS	
Intellectual Disability	More severe deficit in adaptive behaviour Level of ID
Autism	Meeting criteria for autism on a standardised measure
Genetic Syndromes	Cri du Chat syndrome Cornelia de Lange syndrome Fragile X syndrome Prader–Willi syndrome Lowe syndrome Down syndrome
Physical health	One or more health problems Visual impairment
Sensory sensitivity	Tactile hypersensitivity

Syndromes in which the prevalence of self-injury is higher than expected given relevant group characteristics include: Lesch–Nyhan, Cornelia de Lange, Cri du Chat, fragile X, Prader–Willi and Smith–Magenis, etc. The current data demonstrate that the presence of specific syndromes is associated with a two to 35-fold increase in the odds of self-injury. (2)

Alterations in neurotransmitter levels in early development are thought to contribute to SIBs. For example, Dopamine deficiency in Lesch-Nyhan syndrome.

In another hypothesis, it has been suggested that SIBs release endogenous opiates that maintain the behaviour, therefore there have been trials of opiate antagonists (naltrexone) for reducing SIBs. This is thought to occur due to the dysregulation of hypothalamo-pituitary-adrenal axis, the pro-opiomelanocortin (POMC) molecule specifically. The POMC molecule, which is produced mainly in the anterior pituitary, undergoes enzyme cleavage producing a number of biologically active products including the opioid peptide β-endorphin and the peptide hormone adrenocorticotrophin (ACTH).These products of POMC are normally released together by the pituitary in response to stress, and in adults plasma levels of the two

products are normally highly correlated. Studies have suggested however that this normal "coupling" of β-endorphin and ACTH is reduced following episodes of SIB in adults with developmental disabilities, with levels of β-endorphin elevated with respect to levels of ACTH. Subsequent

studies of response to naltrexone revealed a complex pattern of results in which higher basal levels of β-endorphin relative to ACTH and elevation of β-endorphin levels following episodes of SIB were associated with different patterns of response to treatment with naltrexone. (5)

Increased rates of SIBs have been noticed in association with menstruation, physical conditions such as otitis media, fatigue, allergies, etc. These conditions may not directly have an effect on SIBs but may reduce threshold of individual's tolerance towards task demands leading to frustration and thus SIB. (3)

A number of studies in the past have indicated that pain may be directly related to self-injury. For example, children with chronic pain tend to self injure near the site of pain. Some studies showed that gastro-oesophageal reflux was related to self-injury in Cornelia de Lange syndrome, presumably as a result of pain and discomfort. It has been observed that self-injury occurs in response to pain before being subjected to social reinforcement. Self-injury along with reinforcements can then moderate pain perception and thereby increase the pain threshold leading to an increase in SIB. (2)

Self-restraint behaviours:

Self-restraint behaviours are those which restrict the movement of an individual's body parts using clothing, objects or a person's own body. There are many different topographies in self restraint behaviours which can be grouped into three major categories. First would be restricting the movement of body parts by wrapping or entangling them in inanimate objects, like in a cloth. The second category comprises behaviors in which one part of the body is used to restrain another, for example, by clasping hands together, sitting on hands. The third category consists of self-applying, or requesting application of protective equipment or restraints, or holding particular objects. The Self-Restraint Questionnaire (SRQ) which is a 23-item instrument, can be used to assess self-restraint behaviours. (5)

Stages of SIB:

According to the Guess and Carr (1991) model of the development of self-injury, in the first stage there is emergence of rhythmic repetitive behaviours. In Stage 2, these repetitive behaviours function to optimise arousal. In Stage 3, these behaviours become sensitive to environmental (social) reinforcement and are shaped into increasingly severe behaviour. While Guess and Carr's model explains the development of self-injury, it does not account for the elevated prevalence of self-injury in ASD or genetic disorders, and the associations between self-injury and painful health problems, repetitive behaviours and impaired behavioural control.

Oliver et al (2015), modified this model by

adding a fourth stage where the SIB becomes less sensitive to the environmental and social triggers and the behaviour is not under the individual's control anymore. Self-restraint behaviours become increasingly evident in this stage in an attempt to control the SIB. Stage 2 was also modified as to include behaviours that are sensitive to internal states and this accounted for the associations between pain and SIB (2)

Evaluation:

The first step in assessment of an SIB, is to describe the behaviour of concern. For example, hitting head with hand. The clinician should take a complete clinical history by reference to previous clinical notes and discussions with the caregivers. The behavior concerned will often have emerged at a relatively young age and may not have been regarded as particularly problematic until it started to result in tissue damage. The clinician should enquire regarding possibly relevant life events, including those with traumatic potential such as abuse and bereavement.

A wide variety of physical health problems may contribute to SIB and initial assessment should include enquiries about pain in any part of the body, headache, migraine, and menstrual pain and about other sources of discomfort such as fever, coughs and colds, and constipation.

Psychological examination will obviously include assessment of ID and ASD with severity levels using appropriate tools and consideration of the possibility of genetic syndromes associated with SIB including Cornelia de Lange syndrome, fragile X syndrome, Lesch-Nyhan syndrome, Rett syndrome, Smith-Magenis syndrome. Assessment should also consider the possible presence of other potentially treatable psychiatric disorders which may contribute to causation and/or persistence of SIB, including anxiety disorders, mood disorders, post-traumatic stress disorder, and psychotic disorders.

Assessment of co-morbid conditions like pain, sleep disturbances, overactivity and impulsivity should be done.

The clinician should gather descriptions of the form, interrelationships, context, consequences, and history relating to a challenging behavior, together with information on the client's communicative abilities and possible socially appropriate behaviors which might be reinforced as functional alternatives to challenging behavior. The Functional Assessment Interview (FAI) includes questions on the topography, frequency, duration, and intensity of the behaviors to be assessed, their co-occurrence, motivating operations, antecedents, and possible maintaining consequences. This helps us identify a communicative function for the SIB. (5)

Interventions:

The intervention needs to be personalised to every child based on the level of intellectual functioning, ASD, genetic factors, internal states and environmental factors. From the stages of SIB described earlier, during the first stage, i.e., when the child has rhythmic repetitive behaviours broad communication training can be done to improve functional communication. Documentation of the child's typical behaviour can also be done when the child is healthy. This will help identify "pain" in the future when there is a change in the typical behaviour.

During the second stage, i.e., when the self-injury is sensitive to internal states, appropriate pain relief measures should be taken. Appropriate medical interventions to relieve pain and painful health conditions should be ensured. Also, regular assessments of physical health and behavioural changes should be carried out.

In stage 3, i.e., when the behaviour is sensitive to environmental factors and social reinforcement, specifically designed communication strategies based on functional analysis report should be used. This is known as functional communication training. This can also be initiated in stage 2 preemptively. Environmental factors should be identified. Environmental modifications and change in caretakers response to SIB should also be targeted. Additionally, precursor behaviours need to be identified and physical health should

be monitored including prompt intervention for pain and painful health conditions.

Stage 4, i.e., when there is loss of behaviour control, restraint fading should be done. This is done by brief removal of restraint and replacement with another behaviour that can easily be faded. Compliance training is then implemented to increase the time out of the restraint. Following this, generalisation of the habit is facilitated. (2)

Drugs used in the treatment of SIB:

Drugs can be tried in SIB when a communicative function of SIB is not identified. (3)

Risperidone initially came into the market as a treatment for Schizophrenia. Risperidone acts by modulating the levels of Dopamine and Serotonin in the brain. Schroeder had a hypothesis that SIB may be caused by a depletion of dopamine and an excess of serotonin in the basal ganglia region of the brain. He thus pursued Risperidone as a treatment option for SIB. (4)

Other atypical anti-psychotics like olanzapine and ziprasidone have also been tried. However, caution must be exercised while using these medications as they have serious side-effects like dystonia, weight gain, neuroleptic malignant syndrome, hypotension, etc. (3)

Research on the use of Selective serotonin re-uptake inhibitors (SSRIs) in the past have shown equivocal results with some reductions in the rate and frequency of SIBs. (2)

The most consistent evidence in the use of medications for SIB has been seen with opiate antagonists (naltrexone). The probable mechanism of action of naltrexone in SIB has been described earlier in the article. It is possible that naltrexone and naloxone act by simply increasing the pain experienced from self-injury and hence influencing the response cost of an operant behaviour. (2)

For patients with severe refractory SIBs where a trial of behavioral and pharmacotherapy has been given, neuromodulation can be considered as a last resort. There are multiple areas found along the limbic system which when subjected to deep brain stimulation lead to an improvement in the outcome. It is believed that the neuronal circuits connecting the amygdala, the hippocampus and the periaqueductal gray, control reactive aggressiveness and this is moderated by the ventromedial frontal cortex. (6)

Prognosis of SIB:

Current literature suggests that SIB remains stable over lifetime. It remains persistent in adolescence and adulthood both among patients with and without ASD. Age-related decline in SIB is seen. This is also associated with reduction in symptomatology, stereotyped and repetitive behaviours in patients with ASD. Despite this, current studies have shown persistence of approximately 40% over a 10-year period in children with autism. Persistence of SIB is observed to be much higher in children with ID with one study quoting a persistence of 84% over an 18-year period. Persistence of SIB beyond 20 years of age is considered to be a chronic problem requiring professional intervention. These data support arguments advocating early intervention to prevent SIB from occurring and therefore persisting over time. (4)

References:

1. Taylor, L., Oliver, C. and Murphy, G. The chronicity of self-injurious behaviour: A long-term follow-up of a total population study. Journal of Applied Research in Intellectual Disability. 2011. 25, 107-117. DOI: 10.1111/j.1468-3148.2010.00579.x
2. Oliver C, Richards C. Practitioner Review: Self-injurious behaviour in children with developmental delay. Journal of Child Psychology and Psychiatry. 2015. 56:10, pp 1042–1054. doi:10.1111/jcpp.12425
3. Carey WB, Crocker AC, Coleman WL, Elias ER, Feldman HM. Developmental-Behavioral Pediatrics. 4th ed. Philadelphia: Saunders Elsevier; 2009. Chapter 49, Behavioral challenges and mental disorders in children and adolescents with Intellectual disability; p. 486-487
4. Laverty C, Oliver C, Moss J, Nelson L, Richards C. Persistence and predictors of self-injurious behaviour in autism: a ten-year prospective cohort study. Molecular Autism. 2020. 11:8. https://doi.org/10.1186/s13229-019-0307-z
5. Frederick Furniss, Asit B. Biswas. Self-Injurious Behavior in Individuals with Neurodevelopmental Conditions. Switzerland: Springer Nature Switzerland AG; 2020. Chapters 3,5,6,7
6. Heiden Petra, Weigel Daniel Tim, Loução Ricardo, Hamisch Christina, Gündüz Enes M., Ruge Maximilian I., Kuhn Jens, Visser-Vandewalle Veerle, Andrade Pablo. Connectivity in deep brain stimulation for self-injurious behavior: multiple targets for a common network? Frontiers in Human Neuroscience. 2022. 16. doi:10.3389/fnhum.2022.958247