Why do patients meeting criteria for borderline personality disorder deliberately harm themselves? Some hypothesised neurobiological correlates

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Summary

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The majority of patients meeting criteria for borderline personality disorder deliberately harm themselves at some time in their lives. This deliberate self-harm is nearly always subsequent to interpersonal loss. This paper postulates that the physiological arousal, which precedes deliberate self-harm, has a distinctive pattern specific to borderline personality disorder. The clinical manifestations are: baseline distress, followed by an environmental trigger factor (separation), which sets off an acute response of increasing distress, usually accompanied by ruminations. This develops into an intensely disagreeable subjective state, with simultaneous cutaneous anaesthesia or dissociative symptoms. It is this highly unpleasant state which is rapidly and very effectively relieved by soft tissue injury.

In borderline personality disorder a dysfunctional stress response is hypothesised to underlie the acute subjective distress and the accompanying somatic symptoms that occur prior to deliberate self-harm. Central neuro-humoral mechanisms, via peripheral afferent stimulation as a result of soft tissue injury, are hypothesised to relieve the subjective tension by re-establishing homoeostasis. This paper examines the underlying biological aspects of separation distress and the mechanism by which it is relieved. In this sense separation distress is conceptualised in behavioural terms as the reaction to an environmental event. The psychological mechanisms of attachment and separation are not examined.

Deliberate self-harm in borderline personality disorder may thus be understood as a coping mechanism, a potent means of overcoming unbearable subjective distress. A clearer understanding of the mechanisms of this behaviour may lead to more effective and compassionate treatments.

Keywords: borderline personality disorder; deliberate self-harm

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Introduction

Borderline personality disorder as defined by the 4th edition of the Diagnostic and Statistical Manual (DSM IV) of the American Psychiatric Association [1] requires the presence of at least 5 out of a possible 9 characteristics. In theory, there are 151 possible combinations leading to a diagnosis of borderline personality disorder [2], some of which would only share one criterion. This has led researchers to look at dimensional aspects of borderline personality disorder such as impulsivity which have known and readily measurable biological correlates. Unfortunately this may overlook years of detailed observation that led to the grouping of, apparently disparate, symptoms into one clinical entity.

Deliberate self-harm, as distinct from suicide attempts, is seen in several psychiatric conditions and is also one of the diagnostic criteria of borderline personality disorder [1]. Intense effort to avoid abandonment is also a diagnostic criterion of borderline personality disorder. Thus, disruption in the interpersonal sphere is considered integral to borderline personality disorder. According to the research literature, deliberate self-harm in borderline personality disorder patients usually occurs in the context of disruption of an important relationship [3]. Is there an underlying biological relationship between deliberate self-harm and interpersonal loss in patients meeting criteria for borderline personality disorder? Clinical descriptions abound where fears of abandonment and self-harm can be

considered as paradigmatic of borderline personality disorder. This paper suggests that indeed, the two are related.

Effective treatment of deliberate self-harm poses a considerable problem. There is substantial overlap between suicide and deliberate self-harm [4, 5], 40–50% of people who die as a result of suicide have prior episodes of deliberate self-harm. However, at the time of self-harm, the majority of patients are not trying to kill themselves [4, 6]. The question is then, what prompts patients to act in this way? This paper sets out to explain this behaviour in patients meeting criteria for borderline personality disorder and postulates neuro-humoral mechanisms that may be involved.

Clinical description

The following description is based on our clinical observation of over 200 patients meeting criteria for borderline personality disorder (in our specialised treatment programme) and concurs with descriptions already found in the literature [7, 8].

Prior to self-harm, patients report subjective feelings of general distress, for example a chronically stressful personal situation or a depressive episode. Even a relatively minor event may aggravate the situation, causing increasing suffering. At this stage ruminative ideas and a sensation of solitude (sometimes even in the presence of other people) may co-occur. Without active intervention, the distress will continue to increase in intensity and develop into severe emotional pain. This sensation is not only difficult for patients to describe and communicate, but rapidly becomes intolerable. Partial or complete cutaneous analgesia almost invariably occurs at this point. Many patients also describe dissociative symptoms. The patient, driven to find relief, intentionally self-inflicts soft tissue damage and extremely rapidly the unbearable distress subsides to tolerable levels. Because of the concomitant analgesia, no somatic pain is perceived at the time of injury (it often appears later). The dissociative experiences also disappear instantly and the patients often describe "feeling themselves again". Many patients experience relief following relatively minor tissue damage, such as bleeding scratches, whereas other patients describe longer lasting relief with more severe injury. Deliberate self-harm occurs more commonly in the evenings. Thus, patients meeting criteria for borderline personality disorder have a distinct pattern of physiological arousal, which gives rise to, and is relieved by, deliberate self-harm.

From the clinical description above, it can be seen that when patients present to medical services following deliberate self-harm, the acute subjective and physical state has already receded. It is understandable then, that if health workers are ignorant of the preceding clinical picture, they may be subject to feelings of perplexity and often hostility towards these patients. Deliberate self-harm may be better understood as a coping mechanism, a highly effective means of overcoming unbearable psychological suffering. In behavioural terms deliberate self-harm is under the control of consequences (operant behaviour). The immediate, strongly positive consequence, relief of suffering, will increase the likelihood of recurrence of the same behaviour in similar circumstances.

With this clinical picture in mind, it is readily understandable why some patients harm themselves, even repeatedly. It also should alert the clinician to the remarkable intensity of suffering which would drive someone to act in such a way.

Hypothesis

In patients meeting criteria for borderline personality disorder, with baseline distress, an environmental trigger (usually separation) produces a characteristic pattern of intense subjective distress accompanied by cutaneous anaesthesia or dissociative symptoms. This highly unpleasant state is relieved by deliberate self-harm. A dysfunctional stress response is hypothesised to cause the acute state, and central neuro-humoral mechanisms via peripheral afferent stimulation, following soft tissue injury, are hypothesised to restore homoeostasis and relieve the subjective distress.

Neurobiological mechanisms associated with separation distress

The perceived loss of a significant relationship in all individuals produces an acute response. This reaction is common to all mammals and is important for survival and group cohesion. The neurobiological mechanisms that are activated during separation produce a highly unpleasant subjective state. This may be contrasted with those which become activated with the renewal of contact, and which serve to appease the unpleasant subjective state and imbue a sense of well-being. Separation distress is powerfully inhibited by β-endorphin, prolactin and to a lesser extent, oxytocin. These circuits are developmentally related to pain path-

ways [9]. The interplay between these mechanisms governs social behaviour in a tonic fashion. Opiate antagonists increase social behaviour in humans and non-human primates, whereas the administration of opiates reduces contact seeking. Humans develop marked separation distress at around six months of age and this normally lasts for many years. The attenuation of separation distress is mediated in part by maturation of the pituitary-gonadal axis. This effect is more marked in males because of the powerful inhibitory effect of testosterone. When the social isolation of immature animals is prolonged, the animals demonstrate lifelong problems of social attachment [10–12]. Personal histories of disturbances or loss in early relationships are risk factors for developing borderline personality disorder [13]. Interestingly, non-human primates with enforced early separation from the mother also demonstrate self-injurious behaviour [12].

Endogenous opiate system and stress responses

The endogenous opiate system participates in many important physiological functions, including neuroendocrine systems, analgesia, stress, temperature, dependence, reward and learning amongst others. The system is characterised by marked genetic diversity. There are three receptor subtypes, μ, δ, and κ. The ligands at these receptors are the opioid peptides, β-endorphin, enkephalins (met & leu) and dynorphin, derived from pro-opiomelanocortin (POMC), pro-enkephalin and pro-dynorphin respectively. Although the anatomical distribution of the endogenous opioids differ, they are all involved in the modulation and processing of nociception [14].

In humans stress produces activation of the hypothalamo-pituitary-adrenocortical axis (HPA). Corticotropin releasing hormone (CRH) is released from the hypothalamus into the hypothalamo-pituitary portal circulation and is then delivered to the anterior lobe of the pituitary. There it produces cleavage of the POMC precursor molecule to produce adrenocorticotropic hormone (ACTH), β-endorphin and melanocortin. Thus, β-endorphin as well as ACTH participates in the stress response [15, 16]. It should be noted that serotonin (5 HT) may affect POMC mRNA expression and may thus underlie a biologic or dispositional vulnerability to abnormal stress responses [17]. Transient stress-related analgesic states are well known, and the endogenous opiate system has been implicated in the aetiology of these states [14]. The endogenous opiate system has also been implicated in the genesis of dissociative states [18].

Peripheral and central neuronal mechanisms activated by deliberate self-harm

Mechanical injury to the skin tissues involves local inflammatory and neuronal mechanisms [19]. Central sensory and affective pain processes share common sensory mechanisms in the periphery [20, 21]. Highly specialised nerve fibres in the skin, known as nociceptors, transmit, via the dorsal horn and spinal cord, sensory afferents to the brain. Differentiation of sensory and affective processing begins at the dorsal horn following spinothalamic and spinoreticular pathways respectively. The central processing of nociceptive signals to produce affect takes place in extrathalamic pathways that project to the neo-cortex. Of particular note are nociceptive afferent systems transmitting through limbic brain mechanisms notably the dorsal noradrenergic bundle, originating in the locus coeruleus, the ventral noradrenergic bundle and the HPA. Of these the dorsal noradrenergic bundle is most central to negative affect. The dorsal noradrenergic bundle projects from the locus coeruleus through the entire limbic system to all of the neo-cortex. The locus coeruleus reacts invariably to nociceptive stimuli. These structures also react to non-nociceptive threatening events including aversive emotional states [22, 23]. Thus, central nociceptive, threatening non-nociceptive and affective systems are interconnected.

As mentioned above the hypothalamus is also important in the physiological response to tissue injury. The hypothalamic paraventricular nucleus receives afferents from the ventral noradrenergic bundle, which in response to noxious stimuli will in turn stimulate corticotrophin releasing hormone (CRH). CRH neurons then release CRH into the portal circulation. The hormonal cascade following CRH release is detailed above. The paraventricular nucleus also receives afferents from reticular areas, limbic areas and the spinal cord.

Opiate antagonists in the treatment of deliberate self-harm

Opiate antagonists have received considerable attention in the pharmacological treatment of deliberate self-harm, which would imply abnormal endogenous opiate secretion in patients with the condition [24–26]. One study found increased

met-enkephalin levels in borderline personality disorder patients with deliberate self-harm [7]. Another showed uncoupling of POMC fragments immediately after deliberate self-harm, as compared with perfectly correlated POMC products measured in the same subject in the morning [27]. In that study elevated β-endorphin levels were associated with a positive response to opiate antagonists as evidenced by reduced deliberate self-harm. Opiate agonists and antagonists have also been associated with acute dysphoric reactions in human subjects including controls [28, 29]. The latter were attributed to pre-existing elevated stress levels.

Discussion

These findings taken together would suggest that there is a carefully modulated homoeostatic mechanism, which regulates the physiological arousal to separation and its relief in normal individuals. This mechanism is developmentally related to pain systems. In individuals with normal development it becomes less reactive, particularly after sexual maturation and this is more marked in males.

For reasons remaining to be determined, in patients meeting criteria for borderline personality disorder this mechanism is either dysfunctional or more readily knocked out of balance. The problem could be related to the enormous genetic diversity in the endogenous opiate system, and thus an inherited defect. It could also be that developmental disturbances prevent the integration of normative functioning, or a combination of the two. In any case for patients suffering from this type of acute subjective distress, by activating nociceptive and non-nociceptive neuro-humoral systems, deliberate self-harm restores homoeostasis of the separation distress system at the cost of tissue damage.

Painful experience, whether psychological or somatic, is difficult to study. Nearly all research focuses upon the elements of painful experience associated with physical illness. Little attention has been paid to differences in the degree or the intensity of psychological distress arising from psychiatric conditions. Furthermore, the appreciation of painful states by health care workers is often simply erroneous with women and unattractive subjects faring worse [30]. In studies of somatic pain it has been shown that pain sensation and pain unpleasantness are two dimensions [31]. In other words the subjective quality of the experience is only partly related to the intensity of nociceptive stimulation. It is likely then that in some psychiatric conditions the subjective distress experienced by the sufferer in no way correlates with the perception of the entourage, the severity of the triggering event or the overall functional disability.

Until now, peripheral mechanisms, primarily visceral, have been regarded as manifestations of, or secondary to, centrally arising (emotional) stress, as exemplified in the physical manifestations of panic attacks. This paper suggests that peripheral mechanisms, in this case tissue damage, can actually modulate the central appreciation or experience of negative affect.

Conclusion

There is evidence suggesting a contributory role of a dysfunctional stress response to interpersonal loss in the genesis of transient dysphoric states in patients meeting criteria for borderline personality disorder. This may be the result of a relative imbalance of the CRH, ACTH and the endogenous opiate systems. Deliberate self-harm may be understood, in this context, as a compensatory mechanism, restoring homoeostasis. Better understanding of these mechanisms may open the door to more effective treatments and perhaps more compassion towards the suffering of these patients. This hypothesis paper is part of the development of a specific treatment intervention for deliberate self-harm and preparation for a prospective study.

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