

moral judgements based only on ‘medical risks.’ Instead, capacity evaluators play the vital role of helping treaters recognise the true source of their moral distress. In those cases, referral to broader decision-making bodies such as ethics committees or the courts is appropriate.

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A contextual approach to routinely elicit a trauma-oriented history

Thanks to Dr Ingrassia for her recent editorial on the Independent Inquiry into Child Sexual Abuse in the UK with an emphasis on the need for the sensitive and well-informed clinician to proactively and routinely ask about sexual abuse.¹

In our study of child sexual abuse (CSA) history among psychiatric consultations in a general hospital emergency room, we found that 38% of individuals (adults and minors) referred for psychiatric consultation over a 2-year period described having experienced sexual abuse during their childhood.²

We used a semi-structured questionnaire with language that was appropriate to age and cultural background in order to routinely enquire whether the patient had experienced physical, emotional or sexual abuse during their childhood in accordance with a widely accepted definition of sexual abuse.³ This approach is consistent with the research that multiple forms of adverse childhood experiences may coexist.⁴ We believe that a contextual approach like this is more likely to promote a discussion of the person’s trauma narrative. Using this paradigm, with appropriate training, it is hoped that medical and paramedical clinicians will be able to sensitively and routinely take a comprehensive trauma-oriented history in every patient. In this way, the patient’s presenting problem may be understood and treated with an understanding of ‘what has happened to this person’.

It is worth mentioning that adverse childhood experiences including sexual abuse is not only associated with an increase in lifetime prevalence of mental illness but also of physical illness. There is evidence linking early-life stress to reduced telomere length in a study of physically and psychiatrically healthy adults with or without a reported history of childhood trauma. These early experiences may affect adult health in two ways: either by cumulative damage over time or by the biological embedding of adversities during sensitive developmental periods.⁴ Mediating factors between CSA and physical illness include neuroendocrine dysfunction, metabolic syndrome and chronic inflammation.⁵

To the best of our knowledge our study is the first to investigate CSA history during hospital emergency room psychiatric consultations. It is hoped that there will be an increased awareness of CSA during psychiatric consultations in a general hospital setting.

Interventions for past CSA should include the nature of early-life trauma and its effects on psychobehavioural factors. When healthcare providers counsel victims of childhood abuse, they should consider the long-term psychological and physical well-being necessary to counter adverse responses to abuse such as disordered eating, lack of exercise, sleeping problems and depressive symptoms. They should also promote healthier ways to cope with trauma. Such psychological interventions would have the potential to prevent or reduce physical health problems in later life.⁴

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Conclusions in Gryglewski et al may not be warranted

A number of issues not addressed in Gryglewski et al require comment and clarification.¹ First, the authors show that a significant increase in volumes in amygdala nuclei, hippocampus, putamen and cortical thickness occurred following a course of electroconvulsive therapy (ECT) in 12 patients. However, it is not stated whether these patients’ brain structures average size at baseline is significantly different to what we would expect to find in a healthy cohort, or what percentage of the sample fall below the norm. If this is not clarified, we need to understand why brain structure sizes that may fall within a normal distribution would require enlarging.

Second, patients had two scans before ECT and the authors present the average of the two scans as baseline measures. The authors omit to say how different the measurements were between the two pre-ECT scans, which would inform the reader as to the accuracy of each magnetic resonance imaging reading. This is important since the same procedure was not employed at termination of treatment.

Third, the authors attribute the increase in volume to a process of neurogenesis, which they consider a positive outcome. However, they do not seem to take into account the possibility that the neurogenesis may not be benign but be the result of the electrical insult inflicted on the brain, and that the proliferation and morphology of the newly created neurons may not be normal. Neurogenesis has also been observed to occur in similar areas of the brain following intake of lithium and other mood stabilisers, but it was found that the number and morphology of the cells were abnormal, with ‘increasing growth of cone formation, leading to the spreading of the neuron and a shorter neuronal axon.’² If such cellular proliferation in the areas connected with memory is a positive outcome, rather than a pathological reaction to a brain insult, then widespread memory and cognitive impairment found in a large percentage of patients who have had ECT³ needs explaining.

Fourth, and related to the last point, there is no data presented on the incidence of adverse effects following ECT (disorientation, confusion, memory loss, concentration, impairment in abstract reasoning, overall level of cognitive functioning, docility, lethargy and apathy), which may impact on the ability to perform a post-treatment test.

Finally, the authors bemoan the difficulty with recruiting ‘suitable patients’ and ended up with a very small sample. In an era of antidepressant-induced treatment-resistant depression,^{4,5} I suspect