was often associated with higher levels of acyclovir than expected [3], other reports did not find a correlation between serum or CSF levels and symptoms [4]. The neurotoxic effect of acyclovir is usually reversible, and recovery occurs earlier when the patient is on hemodialysis [4].

The distinction between acyclovir neurotoxicity and spread of the viral infection to the central nervous system can sometimes be difficult. In our patient the presence of fever, headache, and temporal lobe EEG changes suggested a diagnosis of encephalitis on the one hand; the normal findings in the CSF on the other hand raised doubts but did not dismiss it entirely. The periodic localized episodic activity, as seen in our patient before she lapsed into coma, was previously thought to be almost pathognomonic of herpes simplex encephalitis but this has recently been questioned [5]. When faced with this dilemma, it is important to recall that herpes encephalitis is a potentially lethal disease if untreated, and even with therapy neurologic sequelae are common. Given the adverse outcome of failure to treat herpes virus encephalitis compared to the complete reversibility of the neurotoxicity, we chose initially to err on the side of treatment. This was also the approach of Rashiq et al. [3].

Acyclovir is a valuable drug for the treatment of herpes encephalitis, both simplex and zoster. Its potential for neurotoxicity should be recognized by all physicians using this drug. While it has not always been shown to be dose-dependent, the prescribing physician should be aware of the dose reduction necessary in the presence of renal dysfunction. The manufacturer recommends increasing the dose interval to 12 hours if creatinine clearance is between 25 and 50 ml/minute to 24 hours with a creatinine clearance of between 10 and 25 ml/min, and reducing the dose to 50% of usual dosing and increasing the dose interval to 24 hours with creatinine clearance of 0–10 ml/min. For the patient on hemodialysis, 60–100% of the loading dose should follow each dialysis session [1].

References

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**Trichotillomania: A Possible Therapeutic Strategy for the Family Doctor**

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**Key words:** Trichotillomania, family practice, patient history

For some years now, trichotillomania has been recognized as a psychopathologic condition [1]. It does not respond well to any specific treatment modality, whether pharmacologic or different psychotherapy techniques [1]. Severe cases are usually treated by psychiatrists over a long period [2]. We report a case of trichotillomania that was treated within a family practice setting using family intervention techniques. Ten years follow-up revealed no recurrence.

**Patient Description**

An 11 year old boy presented to his family physician due to three episodes of loss of consciousness during the previous 10 days. A complete physical examination and comprehensive laboratory and imaging investigations did not reveal any pathology. Distinct areas of hair loss were observed on the scalp.

The patient’s history revealed that he was an only child. His parents divorced when he was 3 years old and he moved with his mother to live with her parents. His grandfather took over the father’s role. Initially he saw his real father twice a year, for a day each time, but then all contact was discontinued. When the patient was 10 years old his grandfather died suddenly, and 3 months later the patient immigrated to Israel with his mother and grandmother. During the subsequent year, the boy learned the language (Hebrew) and was successful academically, he was tough but socially very isolated. He never invited
friends home and was not allowed to play outside. He spent most of his time watching television. Occasionally, on Sunday afternoons, he would accompany his mother outside to sit and gossip with some of his mothers friends. He would insist that his mother be with him when he was at home. When his mother once invited a male work colleague, the boy had such a temper tantrum that she never dared repeat it.

After a family assessment was completed, it was decided that the family doctor would have a 1 hour session with the boy at home, doing ‘male’ activities. The last half-hour included the boys mother, and the discussion was focused on the father, the grandfather, and the meaning of loss. After six sessions, it was agreed that the boy could play football in the neighborhood, and his mother could go out alone once a week. The boy was also introduced to the trainer at the local sports center, and was taken on as an assistant. At the same time it was observed that his hair-pulling had virtually ceased. A month later the mother met a widower, and this time the boy welcomed him into the family circle. Today, 10 years later, the mother is happily married, the boy has a sister 11 years younger than him, and trichotillomania has never recurred.

**Comment**

Trichotillomania is defined by the DSM IV as hair-pulling resulting in noticeable hair loss. It occurs in up to 4% of the general population and is more common in females under the age of 17. The etiology is unknown, but it is more common in people who have suffered significant loss including separation during childhood [1]. The condition warrants treatment for various reasons, the esthetic one notwithstanding. The severe form of the condition is quite uncommon and is very difficult to treat [2–4].

In our patient the following observations were made after the initial assessment:

- The boy had trichotillomania, diagnosed by exclusion of other possible causes of alopecia. Hair-pulling was observed.
- The issue of loss had not been resolved in the family.
- The boy had no father figure in the critical prepuberty stage of development.
- The boy and his mother were too enmeshed for any sort of independent development.

Considering these initial observations collected during the first few meetings, the following treatment strategies were adopted:

- Creating a differentiation between the various family members.
- Understanding the hidden conflicts.
- Defining the roles and boundaries.
- Developing trust.
- Developing more appropriate feedback patterns between the family members.

In conclusion, various treatment modalities for the alleviation of trichotillomania have been described. Medications such as antidepressants and lithium afford a benefit of short duration only, if at all [3]. Other treatment modalities include psychoanalysis, behavioral hypnosis, and family therapy [2,4,5]. We discuss a technique of family therapy used successfully to treat a severe case of trichotillomania by the family physician. We suggest that even in severe cases of trichotillomania, and certainly in the less severe types, consultation with the family doctor is appropriate and perhaps even beneficial. His prior knowledge and understanding of the family dynamics and relationships and the trust that he has with the family may give him a therapeutic advantage.

**References**


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**Capsule**

**A model of overweight**

Individuals with visceral (intraabdominal) obesity are particularly prone to develop a cluster of metabolic disturbances, termed “metabolic syndrome,” that include glucose intolerance, insulin resistance, plasma lipid disorders, and hypertension. Because visceral obesity has been associated with high levels of glucocorticoids, Masuzaki et al. studied the role of 11β hydroxysteroid dehydrogenase type 1 (11β HSD-1), an enzyme that can amplify glucocorticoid action and is overexpressed in the adipose tissue of obese humans. Transgenic mice that modestly overexpressed 11β HSD-1 in adipose tissue developed visceral obesity and, remarkably, displayed many of the defining features of the metabolic syndrome.

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