Tourette syndrome

Medicine's recognition of Tourette syndrome (TS) is now one hundred years old. There was little published concerning this condition after the initial description until the 1960s when it was rediscovered. At that time it was considered to be a rare psychiatric disorder. Over the last 10 to 15 years, however, it has become obvious that TS and other related chronic tic disorders are relatively common conditions. Professionals have become aware of the diagnosis and are making it with increasing frequency, and the laity has also learned much about the disorder through the popular press and other media.

With the renewed interest in TS has come a large number of clinical and laboratory studies presenting strong evidence that the disorder has an organic, possibly genetic, origin.[1–3] As a reaction to the previous psychiatric theories, there has been an attempt to "depsychologize" the disorder and to consider all behavioral abnormalities to be reactions to the stress caused by the bizarre nature of the symptoms.

Unfortunately, mind-brain relationships are not that simple. It has been noted that attention deficit disorder (ADD), learning disabilities, and obsessive-compulsive symptoms are greatly increased in frequency in individuals with Tourette syndrome. There have been attempts to define a common genetic etiology for both TS and ADD, with the implication that they may be different manifestations of the same underlying functional or structural abnormality.[4]

The work of Erenberg, Cruse, and Rothner in this issue is significant in that it presents an analysis of a large, well-defined, consecutive case series.[5] This report clearly documents the high incidence of ADD (35%), learning disabilities (22%), and serious psychiatric disorders (9%). Fifty-eight percent of the patients had at least one of these problems. The group with serious psychological problems always showed obsessive and compulsive symptoms that were often more disabling than the tic disorder. One striking and unexplained finding was that there was no correlation between the severity of TS and the severity of ADD.

A possible bias in this study is that selection required referral to this specialty group. Although this might be expected to exclude the milder cases, these researchers found, in fact, a higher percentage of mild cases than in previous series of patients in the literature. The lack of correlation between the severity of TS and that of ADD indicates that no major bias existed.

Good clinical research raises at least two new questions for every one it answers. The critical clinical issue that must now be faced is what is appropriate therapy for the child with both TS and ADD. There is evidence that psychostimulants, the drugs of choice when therapeutic intervention in ADD is required, exacerbate the tic disorder in as many as 20% to 50% of patients.[6] Haloperidol and pimozide, the most predictably useful drugs for treating TS, do not alleviate the symptoms of ADD. In some children ADD is a greater limiting factor in the child's adjustment than TS. Managing such a child presents the practitioner with a clinical dilemma that is currently unresolved. The Cleveland Clinic group is actively attempting to resolve the issues and develop a rational treatment strategy.

The second question raised by the strong association of TS and ADD concerns the underlying neurobiology of both disorders. There is evidence implicating abnormalities of dopaminergic and serotonergic systems in TS and norepinephrinergic or dopaminergic systems in ADD. These central neurotransmitters obviously do not function in isolation. The response of some children with TS to treatment with cloni-

dine, an alpha-adrenergic agonist-antagonist, is an example of these complex and poorly understood interrelationships. As detailed knowledge of the interactions of these neurotransmitters is developed, major strides can be made in understanding not only TS and ADD, but also the broader area of mind-brain relationships.

References

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