TEXT 1

Autoimmune encephalitis causes subacute deficits of memory and cognition, often followed by a suppressed level of consciousness or coma. A careful history and examination may show early clues to particular autoimmune causes, such as neuromyotonia, hyperekplexia, psychosis, dystonia, or the presence of particular tumours. Ancillary testing with MRI and EEG may be helpful for excluding other causes, managing seizures, and, rarely, for identifying characteristic findings. Appropriate autoantibody testing can confirm specific diagnoses, although this is often done in parallel with the exclusion of infectious and other causes. Autoimmune encephalitis may be divided into several groups of diseases: those with pathogenic antibodies to cell surface proteins, those with antibodies to intracellular synaptic proteins, T-cell diseases associated with antibodies to intracellular antigens, and those associated with other autoimmune disorders. Many forms of autoimmune encephalitis are paraneoplastic, and each of these conveys a distinct risk profile for various tumours. Tumour screening and, if necessary, treatment is essential to proper management. Most forms of autoimmune encephalitis respond to immune therapies, although powerful immune suppression for weeks or months may be needed in difficult cases. Autoimmune encephalitis may relapse, so follow-up care is important.

TEXT 2

Autoimmune encephalitis involves several types of diseases with different pathophysiology. Understanding the pathophysiology of these diseases is helpful in using diagnostic testing and choosing appropriate therapies. The first group includes the classic paraneoplastic disorders associated with antibodies to intracellular antigens, such as anti-Hu. These disorders are strongly

cancer-associated and involve T-cell responses targeting neurons. The prognosis tends to be poor due to irreversible neuronal killing by these mechanisms, the severity of associated cancers, and the difficulty in controlling these sorts of immune responses. The antibodies in these disorders are useful tumor markers, and in the appropriate context and titer also useful markers of the paraneoplastic neurological disorders. The antibodies themselves are not directly pathogenic. The second group involves autoantibodies to extracellular epitopes of ion channels, receptors and other associated proteins, such as the NMDA receptor. The cancer associations are variable, and the prognosis tends to be much better. The antibodies in these disorders are thought to be directly pathogenic, causing reversible effects on synaptic function in neurons with relatively little neuronal death. There are also important tumor associations in this group of diseases. For instance, patients with anti-NMDAR encephalitis commonly can recover from a totally unresponsive state to eventually resume a good quality of life. Occupying an intermediate position are diseases with autoantibodies to intracellular synaptic proteins such as GAD65. It is unclear whether this group involves T-cell responses and/or functional effects of antibodies. A final group includes other forms of autoimmune encephalitis in which precise antigens are less clearly established, such as lupus cerebritis or ADEM. Some diseases in
this group have systemic manifestations outside the nervous system.

TEXT 3

Risk factors for autoimmune and infectious encephalitis

Risk factor	Implications
Travel	Consider infectious causes of encephalitis in visited region
HIV	Opportunistic infections, risk depending on CD4 count
Transplantation	Opportunistic infections (CMV, VZV, HSV1, 6, 7); if recently transplanted, consider infection from donor
Systemic autoimmunity	Consider lupus cerebritis, vasculitis
Cancer	Consider specific paraneoplastic syndromes based on tumor, but also lymphomatous/carcinomatous tumor involvement
Prior encephalitis	Consider relapse of initial encephalitis, secondary autoimmune causes, and (if immunosuppressed) opportunistic infections

TEXT 4

Psychiatric manifestations are common early in the course of autoimmune encephalitis. These may include psychosis, aggression, inappropriate sexual behaviours, panic attacks, compulsive behaviours, euphoria or fear. Symptoms may fluctuate rapidly. Although this presentation is well known for anti-NMDAR encephalitis,anti-AMPAR and anti-GABA-B-R both may have prominent early psychiatric manifestations(Overall, anti-NMDAR encephalitis is more common and should be suspected first, especially in young adults and children, but they could each cause this presentation across a wide range of ages).

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Find out from which text this information comes from.

1. Considering a blood donor.
   TEXT 3. Read 3rd line
2. Different kinds of people who may have a chance to get encephalitis.
   TEXT 4
3. The problems linked with the diagnosis of encephalitis.
   No clear explanations but as a whole, it is inTEXT 2
4. About when we take into account a patients immunity
   Text 3 Read the last line
5. Supportive examinations to find out characteristic features
   TEXT 1
6. Different types of autoimmune encephalitis
   TEXT 2
7. Common initial features of encephalitis.
   TEXT 4

NB: this material is made from medical journals available on the internet.