Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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doi:10.1016/j.mehy.2005.08.017

No anti-ACTH autoantibody in serum of SARS patients

Dear Editor,

Dr. R. Wheatland was used putting forward a hypothesis: ''*Molecular Mimicry of ACTH in SARS — Implications for Corticosteroid Treatment and Prophylaxis*'' that published in the journal ''Medical hypothesis'' [1]. Dr. R. Wheatland found that expression of amino acid sequences of SARS virus is molecular mimics of the host's adrenocorticotropin hormone (ACTH). When the host produces antibodies against these viral antigens, these antibodies may also bind to the host's own ACTH, which limits the host's stress response by interfering with ACTH's ability to stimulate the secretion of corticosteroids, leading to the dysfunction of adrenal cortex. Therefore, the treatment with corticosteroid may correct the defect in SARS patients. Moreover, this molecular mimicry of ACTH and anti-ACTH autoantibodies were also found in patients with influenza infection [2].

This is a very interesting hypothesis to explain the reason why corticosteroid treatment is effective for SARS patients. However, this hypothesis has three obvious defects: First, anti-coronavirus antibody became detectable first at 5–10 days after the onset of symptoms, and their levels peaked at 20–30 days and then were sustained over 150 days according to the published data [3], therefore, the dysfunction of adrenal cortex in SARS patients should not exist at early stage of SARS and should be severe in 3 weeks after the infection. However, the hypothesis is not consistent with clinical situation. Second, if anti-coronavirus antibody is the reason leading to dysfunction of adrenal cortex, due to molecular mimicry between the coronavirus and ACTH, the vaccine of SARS should have the same complication. However, this situation was also not found by the clinical test of SARS vaccine in China. Third, we detected whether there was anti-ACTH antibody in serum of SARS patients. These sera were collected, respectively, from healthy donors, patients with SARS, patients with severe SARS and patient with SARS in convalescence [4]. Using an ELISA, we found that the serum levels of anti-ACTH antibody in the three SARS groups was comparable with that in the control group and has no statistic difference between these groups. Using an competition ELISA, we also found that incubation with serum of the three SARS groups with anti-ACTH antibody has no significant effect on the detection of anti-ACTH antibody, indicating no increasing anti-ACTH antibody in serum of SARS patients.

Taken together, these results did not support the hypothesis that anti-ACTH autoantibody in SARS patients may lead to the dysfunction of adrenal cortex.

Acknowledgement

This work was supported by a grant from the National Nature Science foundation of China (No 30260015 to Lou J).

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doi:10.1016/j.mehy.2005.08.031

Elevated blood pressure may cause volume reductions of temporal areas and induce psychotic episodes

Sir,

Sumich et al. [1] report a volume reduction of temporal areas in psychotic patients. It has also been reported, that cerebral blood flow (highly associated with blood pressure) of some, interestingly of temporal areas is elevated in psychotic patients, compared to non-psychotic controls [2]. Elevated CBF may lead to volume reduction of damaged cerebral areas after head injury [3]. Moderately elevated blood pressure elevates serum cortisol level and decreases CBF (autoregulation), acutely elevated blood pressure may diminish serum the cortisol level and the absence of this protective factor leads to en elevated CBF and to vasodilatation (breakthrough), which may cause cerebral ischemia, and, if occurring repeatedly, it may lead to a volume reduction of some cerebral areas and it is reported to induce psychotic symptoms [4].

Furthermore, recent literature reports a significant association of hypotension as an adverse side effect of antipsychotics. Some antidepressants (SSRI) [5] as well as psychostimulants [6] sometimes elevate blood pressure and may also induce psychotic symptoms [7]. For that reason, it was our intention to investigate the importance of elevated blood pressure in patients who present short psychotic episodes.

Intending to prove our hypothesis, we investigated three medication free patients, aged 18—24 years, diagnosed as first-episode psychotic were included in our observation. Cardiac diseases were defined as exclusion criteria. Patients showed stable blood pressure during the non-psychotic episodes (125 ± 15; 75 ± 10 mm Hg). We measured systolic and diastolic blood pressure when patients presented optical or acoustical halluzinations and also in intervals, respectively. Psychotic symptoms were associated with statistically significant increases in systolic and diastolic blood pressure (9.5 and 2.5 mm Hg, $p < 0.001$).

This observation leads to the hypothesis, that acutely elevated blood pressure may induce psychotic symptoms. Furthermore, it may also (as a long-term effect) cause a volume reduction of (temporal) areas within the brain and suggests, that before initiating antipsychotic therapy, the volume reduction of temporal areas should be measured, especially if the patient's blood pressure is elevated. Anyway, the patient's blood pressure should be monitored exactly before initiating antipsychotic therapy.

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