

Correspondence

A fatal case of Behçet's disease with rare complications

To the Editor

It looks particularly inviting to read a report on a disease that came from the same country where it was first reported. I read the report by Sahin and colleagues¹ on "a fatal case of Behçet's disease with rare complications"; and it was interesting however a few queries have to be raised. To state "right jugular vein" literally reflects a denial of the anatomical fact that there are 3 different right sided jugular veins. More importantly, they projected thrombosis of the right (internal) jugular vein as the single venous pathology that existed on first presentation of their patient. They did not comment on the status of the superior vena cava which, on other published paper,² is more than 15 times (1.4/0.08) as likely to become thrombosed in Behçet's disease on comparison with the internal jugular vein. Moreover, they mentioned edema of the face and neck without specifying affection of the right side alone, if this at all does actually happen in lone right (internal) jugular vein thrombosis, as anatomy and common sense dictate. Furthermore, the appearance of multiple venous collateral on the neck and chest cannot serve, or be an outcome of, lone internal jugular vein thrombosis. They are the classic collateral of obstruction of the superior vena cava to serve diversion of venous flow towards tributaries of the inferior vena cava.³ Thus, the authors' statement that "the right (internal) jugular vein was thrombosed at the proximal part near the superior vena cava" is far more likely to have been thrombosis of the superior vena cava with extension to the adjoining (proximal) part of the right internal jugular vein. This viewpoint fits the presented clinical description, which lacked any comment on the status of the upper limbs and other veins of the neck as evidence, for or against thrombosis of the superior vena cava.³ Incidentally, the bilateral transudative pleural effusions are best explained as due to blockage of the venous drainage of the inner side of the chest wall by the superior vena cava thrombosis extending beyond the drainage site of the azygos vein;

the azygos vein drains both sides of the inner chest wall. In fact, pleural as well as pericardial effusions have been reported in superior vena cava obstruction.³

Sahin et al¹ rightly investigated their patient for thrombogenic conditions such as protein C, protein S, and the others, presumably there exists a thrombogenic factor other than Behçet's disease that deserves intervention. It looks likely that the subsequent thrombotic complications that the patient developed, despite the ongoing therapeutic regime for Behçet's disease, could have been precipitated by another separate thrombogenic factor. Thus, it is strange that their finding of the potentially thrombogenic high plasma homocysteine level of more than 5 times the upper limit of normal range, was not at all considered on treating the patient. The question arises "why had the patient been deprived from folic acid with or without vitamin B₁₂ and pyridoxine supplementation",⁴ that is widely recommended now for reducing blood homocysteine level? These vitamins are harmless, even if the claimed benefit of such therapy is still uncertain.

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Reply from the Author

No reply was received from the Author.

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