



Fig. 1: Colour fundus photograph showing scar formation and active chorioretinitis in optic disc superiorly at first visit.

on a regimen of clindamycin, steroids and trimethoprim/sulfamethoxazole for six weeks. Following the treatment, the VA of the patient returned to 10/10 in both eyes with scarring superiorly to the optic disk (Fig. 2).

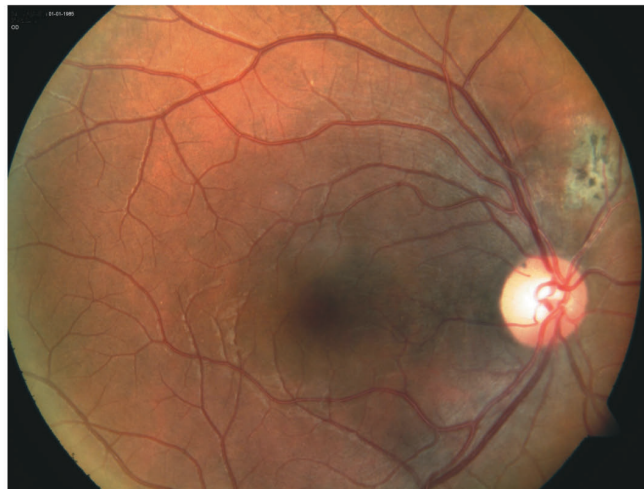


Fig. 2: Colour fundus photograph showing scar formation in right eye after treatment at last visit.

Ankylosing spondylitis is a chronic, progressive and inflammatory rheumatic disease characterized by axial and peripheral joint involvement, which may cause severe disability (1). Currently, if the disease activity continues in spite of the conventional treatments, anti-tumour necrosis factor- α (TNF- α) agents may be used. However, patients should be kept under close monitoring for the side effects of the treatment with biological agents (2). Biological agents may be associated with a higher incidence of granulomatous infections including tuberculosis (3). Still, there is only a limited number of reports regarding patients observed to develop toxoplasma infections during treatment with anti-TNF- α agents and these include rheumatoid arthritis patients with chorioretinitis treated with either etanercept or infliximab (4).

In conclusion, we reported a case of toxoplasmic chorioretinitis in a patient treated with anti-TNF- α agents. The possibility of severe toxoplasma infection during the anti-TNF- α therapy should be kept under consideration due to its serious ocular consequences, which may lead to major sequelae. We are of the opinion that patients should be advised to avoid exposure to infectious agents including toxoplasma before and during treatment with anti-TNF- α .

Keywords: Ankylosing spondylitis, anti-TNF- α , toxoplasma chorioretinitis

I Batmaz¹, F Turkçu²

From: ¹Department of Physical Medicine and Rehabilitation and ²Department of Ophthalmology, Dicle University Medical School, Diyarbakir, Turkey.

Correspondence: Dr I Batmaz, Dicle University Medical School, Department of Physical Medicine and Rehabilitation, Diyarbakir, Turkey. Fax: 90 412 248 8523; e-mail: ibrahimbarmaz82@hotmail.com

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Intracranial Subdural Haematoma after Thoracic Epidural without Signs of Dural Puncture

The Editor,

Sir,

We report the development of an intracranial subdural haematoma (ISH) in a 33-year old male patient who underwent an epidural procedure without evidence of dural puncture, after obtaining his written consent.

The patient presented for surgical excision of a gastrointestinal stromal tumour. He had no history of trauma, headaches or neurological disorders. His coagulation profile was normal. Preoperatively, a thoracic epidural catheter was placed uneventfully and the patient received general anaesthesia under intermittent positive pressure mechanical ventilation (IPPV). Postoperatively, the epidural catheter was used for 72-

hour analgesia. Only once did the patient report a mild, diffuse headache which was relieved with paracetamol. After being discharged, he developed an intense, postural headache, and bilateral sixth cranial nerve palsy (esophoria). A magnetic resonance imaging (MRI) revealed diffuse meningeal thickening with gadolinium enhancement, consistent with intracranial hypotension. The patient preferred conservative treatment over blood patch and his symptoms resolved completely. Twenty days later, he returned to hospital with a severe, persistent headache, not clearly postural, accompanied by nausea/vomiting. He gradually became drowsy and confused, and an urgent MRI revealed a large left ISH (Figure).

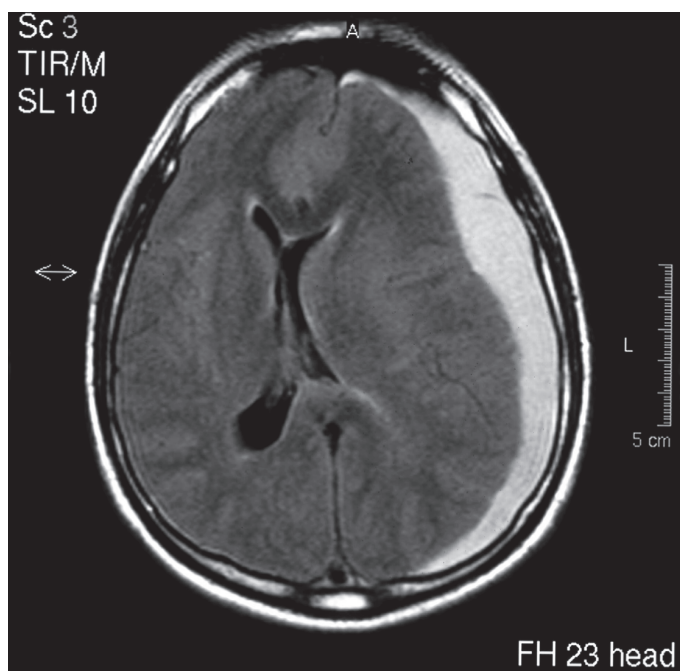


Figure: Cranial magnetic resonance imaging scan on the 45th postoperative day showing a 2.0 cm thick left-sided subacute subdural haematoma with 1.7 cm right midline shift.

The haematoma was surgically evacuated *via* parietal burr holes under local anaesthesia. The patient had a good outcome without neurological deficits.

Intracranial subdural haematoma is associated with cerebrospinal fluid (CSF) leakage and consequent intracranial hypotension; the stretching of bridging veins, along with a compensatory intracranial hyperaemia may lead to rupture of these fragile vessels during their intrasubdural course (1). Development of ISHs after neuraxial techniques is extremely rare, mainly reported in parturients (1). During labour, prolonged Valsalva manoeuvre may increase the CSF pressure and leakage through a dural hole (2). Similarly, IPPV may raise the intrathecal pressure *via* transmission of intrathoracic pressures through the intervertebral foramina.

In the present case, a pre-existing low CSF pressure (primary intracranial hypotension) could possibly explain the ab-

sence of obvious CSF outflow (3). Intracranial subdural haematoma development due to rupture of aneurysm/arteriovenous malformation seems unlikely; no vascular abnormalities were detected on the MRI, while such ruptures usually result in subarachnoid and intraparenchymal haemorrhage (4).

Although it seems reasonable that blood patching would prevent postdural puncture ISHs, they may still develop (5). Moreover, blood patch may produce intracranial hypertension in patients with undiagnosed ISH.

Warning signs of ISH are changes of postdural puncture headache characteristics; it becomes non-postural, more intense and resistant to analgesics (1). Further symptomatology includes nausea/vomiting, vision abnormalities, motor/sensory dysfunction, confusion/disorientation/drowsiness, seizures and coma (1). Diagnosis is based on computed tomography scan with contrast media or cerebral MRI (4). Regarding management, ISHs under 5 mm may resolve spontaneously (1), while large haematomas producing serious neurological symptoms require prompt evacuation.

In conclusion, after neuraxial techniques, the development of intense, worsening non-postural headache resistant to analgesics requires further investigation to exclude serious intracranial pathology.

Keywords: Epidural technique, intracranial hypotension, intracranial subdural haematoma

C Staikou¹, E Stamatakis², K Spengos³, A Fassoulaki¹

From: ¹Department of Anaesthesia, Aretaieio Hospital, Medical School, University of Athens, Athens, Greece, ²Department of Anaesthesia, Alexandra Hospital, Athens, Greece and ³Department of Neurology, Eginitio Hospital, Medical School, University of Athens, Athens, Greece.

Correspondence: Dr C Staikou, Department of Anaesthesia, Aretaieio Hospital, Medical School, University of Athens, 76 Vassilissis Sophias Ave, 11528, Athens, Greece. Fax: +30210 7211007; e-mail: c_staikou@yahoo.gr

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