and other body cavities. They show focal pericytic pattern with spindling of cells, broad zones of hyalinization, and on IHC virtually all cases are CD34-positive.[1,2]

A lipomatous hemangiopericytoma was first described in the German literature by Theunissen et al. in 1990.[2] In 1995, Nielson et al gave a detailed account of this tumor. Subsequently, more cases have been reported and few even at unusual sites such as head and neck.[5] Few authors even favor a unifying concept of lipomatous hemangiopericytoma and SFT.[1]

Our case had many unusual features. The site of occurrence of this tumor in the mediastinum is exceedingly rare.[1–3] To the best of our knowledge, only one more case has been described in the mediastinum.[3] CD34 negativity on IHC is also uncommon.[1–3] The closest differential diagnosis in our case is the SFT. But in our case, the architectural pattern was typical of lipomatous hemangiopericytoma and was present throughout the tumor. Spindling of the cells was not present.

Lipomatous hemangiopericytoma is an extremely unusual tumor. In some cases it may be mistaken for a well-differentiated liposarcoma.[11] Hence it is important to keep this entity in mind especially when only a small biopsy is available. Clinicoradiological features of a well-circumscribed mass and typical histology with lack of lipoblasts favor a diagnosis of lipomatous hemangiopericytoma.[1,4]

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References

Topiramate induced secondary angle closure glaucoma

Sir,
Topiramate[11] (Topomax), primarily an antiepileptic agent is being widely used because of its broad spectrum of therapeutic activity. The adverse effects associated with its usage are nonocular like oligohydrosis and hyperthermia, which are potentially life threatening, and ocular side effects like abnormal vision, acute secondary-angle closure glaucoma, nystagmus and diplopia. Failure to act at the first sign of some of the ocular side effects can lead to permanent visual problems. Here we report a case of Topiramate induced secondary angle closure glaucoma that responded dramatically with conservative management alone after discontinuation of Topiramate.

A 33-year-old woman presented with sudden onset of blurring of vision, redness, photophobia, and discomfort of both the eyes of 24 h duration. Her medical history revealed migraine for the past 3 years under treatment with Topiramate 25 mg as a single daily dose for the past 3 weeks.

Visual acuity was 3/36,N6 in both the eyes with a manifest refraction of –2.00 DS/–1.50DC at 100° in the right eye and –3.50DS/-0.50DC at 120° in the left eye. She was previously emmetropic. Pupils were reacting to light. Slit-lamp examination showed conjunctival congestion, clear corneas, shallow anterior chambers, and clear lens in both the eyes (Figure 1). Intraocular pressures with applanation tonometer were 55 mm Hg in the right eye and 34 mm Hg in the left eye. Gonioscopy revealed closed angles in both the eyes (Figure 2).

A-Scan Biometry revealed an axial length of 22.21 mm in the right eye and 22.31 mm in the left eye, while the respective anterior chamber depths were 2.14 mm and 2.28 mm. Lens thickness was 4.64 and 4.68 mm in the right and left eyes, respectively. Ultrasound Biomicroscopy (UBM) revealed choroidal effusion with anteriorly rotated ciliary processes in both the eyes (Figure 3).

A diagnosis of secondary angle closure glaucoma was made. She was started on oral carbonic anhydrase inhibitors, topical β-blockers, and topical steroids and Topiramate was withdrawn. Examination on the third day of onset of symptoms showed deepened anterior chambers with intraocular pressures of 5mm Hg in both the eyes and resolution of choroidal effusion with normally positioned ciliary processes on UBM. Hence, antiglaucoma treatment was withdrawn and steroids were tapered and stopped. Nine days after the onset of symptoms her unaided visual acuity was 6/6, N6 in both the eyes with intraocular pressures of 12 mm Hg in both the eyes.

Topiramate is a sulfamate-substituted monosaccharide and is prescribed for various disorders such as seizures, infantile spasms, neuroophthalmic migraine, and depression, and also off-label for bipolar disorders and as a weight reducing agent.[11] The precise mechanism of its action is unknown. It has weaker inhibitory action on some of the iso enzymes of carbonic anhydrase.

So far, there are 115 case reports of ocular side effects,[2] 86 cases of secondary angle closure glaucoma,[11] and seven cases of permanent visual loss reported with Topiramate usage.[2] The presentation of secondary angle closure would typically be similar to that of acute pupillary block with intraocular pres-
Male anterior urethral diverticula with Cobb’s collar and a giant stone

Sir,

A 35-year-old man presented with diminution of the urinary stream, frequency, dribbling and a painful perineal protrusion for the past two decades. Examination revealed a hard tender perineal swelling and a small non-discharging blindly ending perineal sinus. Plain X-ray of the pelvis [Figure 1a] showed a giant elliptical urethral calculus (3.5 cm). The retrograde urethrogram revealed a large anterior bulbar urethral diverticulum and a Cobb’s collar, (congenital distal urethral narrowing) [Figure 1b]. The diagnosis of a congenital anterior urethral diverticulum, giant calculus and congenital distal urethral narrowing was confirmed by urethroscopy. A preliminary suprapubic cystostomy, diverticulectomy, stone removal and urethroplasty was carried out [Figure 2]. Histopathology of the excised diverticular wall revealed true urothelial lining. Infrared spectroscopic analysis of the urethral stone revealed 70% magnesium ammonium hexahydrate and 30% calcium phosphate (suggestive of an infection stone). A third month

Our patient was only on Topiramate, the overall score based on the algorithm for the operational assessment of adverse drug reactions[4] is +3, indicating that the cause–effect relationship to be possible as opposed to few case reports[5] where the causal relationship was inconclusive owing to concomitant drug therapy. The pathogenetic mechanism underlying the angle closure in our case was suprachoroidal effusion as evidenced by UBM. Clearly, there is no role of peripheral iridotomy as the mechanism for angle closure is remote from the pupillary block. Our patient was on minimum therapeutic dosage of 25 mg/day, which suggests that ocular side effects are not dose-dependent.

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References