Review Article

Topiramate-induced acute angle closure: A systematic review of case reports and case series

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Topiramate-induced acute angle closure (TiAAC) is a potentially vision-threatening side effect of topiramate (TPM) use. The purpose of this article is to review demographic characteristics, clinical features, and management options of TiAAC. A systematic literature search of all reported cases and case series of TiAAC was conducted in the following search engines: PubMed, Web of Science, Google Scholar, Elsevier, and EBSCO. Seventy-three publications describing 77 cases were included. 58 (75.3%) patients were female, and the mean age was 34.88 ± 11.21 years (range, 7–57). The most commonly reported indication of TPM use was migraine headache (59.7%), and the mean duration from starting treatment until the onset of angle closure was 14.1 ± 31.5 days. All cases were managed by immediate cessation of TPM and topical therapy. In addition, systemic medications (carbonic anhydrase inhibitors, hyperosmotic agents, and steroids) were used in 51 patients (66.2%). A laser and/or surgical intervention was performed in 10 patients (13%). After commencement of treatment, the mean duration until the resolution of TiAAC was 3.9 ± 3.6 days (range, 1–18). The findings of our study present a summary of the current body of evidence provided by case reports and case series on TiAAC. In conclusion, the onset of angle closure following TPM use peaks at 2 weeks after initiating treatment, and in most cases, successful management can be achieved by discontinuing TPM and initiating appropriate medical therapy.

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Topiramate (TPM) is a sulfamate-substituted monosaccharide and an antiepileptic drug that was approved by the United States Food and Drug Administration for its efficacy in treating epilepsy and preventing migraine headache. The drug exerts its effect through several mechanisms of action, including sodium and L-type calcium channels blockage, enhancement of gamma-aminobutyric acid (GABA) receptors, α-amino-3-hydroxy-5-methylisoxazole- 4-propionic acid (AMPA) and kainite current suppression, and carbonic anhydrase inhibition.[1] Following its widespread use, several systemic adverse effects were documented following treatment with TPM, such as weight loss,[2] cognitive dysfunction,[3] and kidney stones.[4] In addition, TPM can also lead to a wide range of ophthalmologic side effects such as acute angle closure, acute onset myopia, uveitis, scleritis, visual field defects, suprachoroidal effusions, oculogyric crisis, and retinal hemorrhage.[5]

TPM-induced acute angle closure (TiAAC) is a potentially vision-threatening side effect of TPM use. It occurs secondary to a drug-induced ciliochoroidal effusion that is associated with a forward movement of the lens-iris diaphragm which subsequently leads to acute angle closure.[6] The exact mechanism underlying ciliary body effusion is not clearly understood; however, it is thought to be related to pharmacological stimulation of prostaglandins release leading to vasodilation and increased permeability in the ciliary body.[7] The literature describing TiAAC primarily consists of a large number of case reports, making it difficult to review such reports and draw clinically relevant conclusions. In this article, we conducted a systematic review of these case reports in an attempt to summarize the current body of evidence on clinical features, management options, and prognosis of TiAAC.

Methods

Protocol

This review was written per Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.[8]

Eligibility criteria

Inclusion criteria comprised of published case reports and case series reporting original data of confirmed acute angle closure secondary to the use of TPM in either adult or pediatric patients. Exclusion criteria were as follows: articles written in a language other than English, research papers on non-human subjects, studies other than case reports and case series, and reports of other TPM-related side effects.

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Information sources

A literature search of the following search engines was conducted: PubMed, Web of Science, Google Scholar, Elsevier, and EBSCO was performed on January 2021.

Search strategy

The search was conducted using the term "topiramate" combined with either "glaucoma" OR "acute" OR "angle closure". Search filters were used to narrow the results down to meet the inclusion criteria. The same strategy was used to search all the aforementioned online databases.

Study selection

Two investigators independently screened the search results for papers that meet the required inclusion criteria in two steps: First, the screening was focused on the titles and abstracts of the seemingly relevant articles; then, the full-text articles of the initially selected papers were retrieved, independently reviewed, and ultimately decided for compatibility according to the inclusion criteria. Disagreements were discussed and resolved between the two investigators.

Data collection process

The extracted data included patient demographics (age and gender), the indication of TPM use, dosage, duration of usage until the onset of clinical manifestations, presenting symptoms, ophthalmic examination findings, treatment modalities, and the period until the intraocular pressure (IOP) was controlled. An electronic form was used to facilitate data collection from the selected case reports and series.

Summary measures

The extracted data was revised, coded, and entered into IBM SPSS, Version 22 (SPSS, Inc. Chicago, IL) for analysis. Descriptive analysis was performed for all variables in which categorical data were presented as frequencies and percentages and continuous variables were presented as mean ± SD.

Results

Study selection

Our search through online databases yielded 537 citations. Furthermore, two additional citations were added through manual search. After removing duplicate records, 472 citations remained; 349 of these were excluded after the initial screening of titles and abstracts. After that, the full text of 123 citations was screened for inclusion criteria. Of these, 50 studies were excluded due to lack of full text ($n = 2$), duplicate population ($n = 31$), and failure to meet inclusion criteria (n = 17). Finally, a total of 73 articles (77 patients) were included in the review [Fig. 1].[9-81]

Study characteristics

The years of publication ranged from 2001 to 2020. Tables 1 and 2 present a summary of the baseline and clinical characteristics of the 77 patients included in this review.

Demographics

Out of the 77 patients, 58 (75.3%) were female. The mean age of patients was 34.88 ± 11.21 years (range, 7–57).

TPM usage

The most common indication for TPM usage was migraine headache (59.7%) followed by epilepsy (10.4%) and weight loss (9.1%). Other less common indications were alcohol use disorder, bipolar affective disorder, cluster headache, anxiety, and pain control. In the cases that reported TPM dosage, the daily dose ranged from 12.5 to 200 mg/day. The mean time that elapsed from the initiation of TPM therapy until the onset of TiAAC was 14.1 ± 31.5 days (range, 0–262).

Clinical presentation

The visual acuity at presentation widely ranged from light perception to 20/20. The mean IOP at presentation was 41.04 ± 15.76 mmHg (range, 8–88) in the right eye and 41 ± 15.3 mmHg (range, 6–82) in the left eye. The amount of refractive error at presentation was reported in 42 cases (54.5%), and the mean spherical equivalent in these patients was -5.37 ± 2.2 diopters (range, (-13)–(-0.75)) in the right eye and -5.31 ± 2.18 diopters (range, (-13)–(-0.75)) in the left eye. Only a few cases had further biometric measurements (anterior chamber depth (ACD) reported in 15 cases (19.5%), and axial length (AL) reported in 7 cases (9.1%)). The mean ACD was 1.65 ± 0.63 mm (range, 0.8–2.64) in the right eye and 1.64 ± 0.6 mm (range, 0.8–2.55) in the left eye, whereas the mean AL was 22.32 ± 1.15 mm (range, 20.2–23.8) in the right eye and 22.31 ± 0.98 mm (range, 20.7–23.78) in the left eye. In addition to acute angle closure, other ophthalmologic features reported include ciliochoroidal effusion in 43 cases (55.8%) [Figs. 2 and 3], uveitis in 8 cases (10.4%), exudative retinal detachment in 2 cases (2.6%), and papilledema in 1 case (1.3%).

Medical management

All cases were managed initially by immediately discontinuing TPM. In addition, topical IOP-lowering medications, cycloplegics, and steroids were used. Some patients were also given systemic medications such as oral carbonic anhydrase inhibitors (CAIs) in 40 cases (51.9%), hyperosmotic agents in 28 cases (36.4%), and steroids in 12 cases (15.6%).

Interventions

The majority of cases were managed medically without the need for laser and/or surgical intervention. Of the few cases that underwent an intervention, laser peripheral iridotomy (LPI) was performed in 8 cases (10.4%), anterior chamber paracentesis in 2 cases (2.6%), laser iridoplasty in 1 case (1.3%), choroidal drainage in 1 case (1.3%), and trabeculectomy in 1 case (1.3%).

Resolution of TiAAC

Following initiation of treatment, control of IOP associated with the resolution of TiAAC occurred in half of the cases (50.6%) during the first three days. The mean duration from presentation until the resolution of TiAAC was 3.9 ± 3.6 days (range, 1–18). Our analysis showed that the use of an intravenous hyperosmotic agent was significantly associated with a shorter period of recovery (4.4 ± 3.9 days vs 2.7 ± 2.1 days, $P = 0.0261$).

Discussion

Summary of evidence

The current review describes the demographic and clinical findings of the 77 TiAAC case reports described in scientific literature during the searched period. For the vast majority of cases, the condition was successfully managed by prompt identification of the underlying cause, discontinuing TPM, and initiating appropriate medical therapy. Further surgical

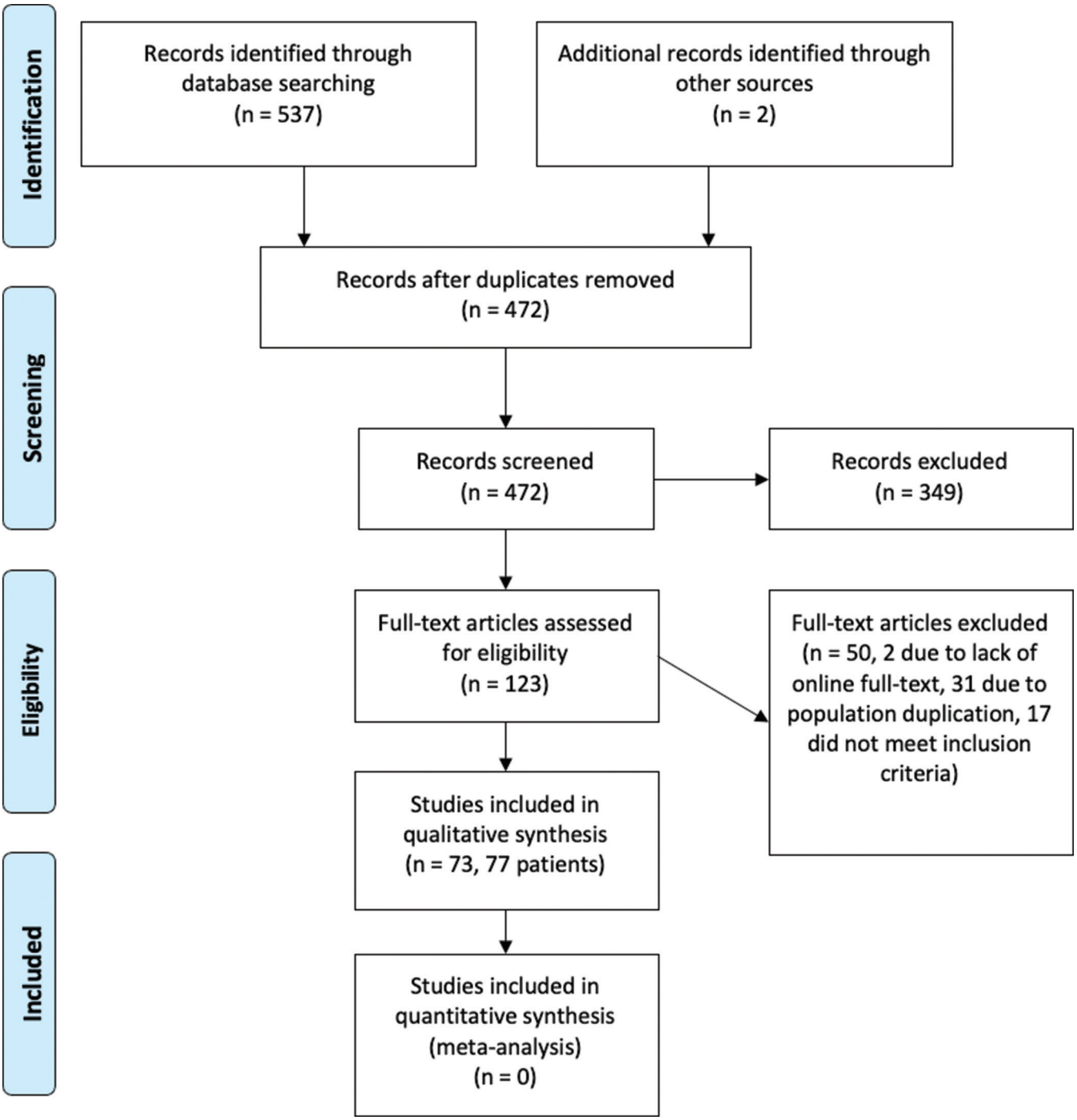

Figure 1: PRISMA Flow Diagram of the systematic review

intervention was only required in two refractory cases that could not be managed medically.[51,56] Below we present a brief discussion of salient findings in our review that are important to physicians involved in the prescription of TPM or the management of TiAAC.

A great proportion of reported patients with TiAAC were females (75.3%); however, this does not necessarily imply that females are at a higher risk of TiAAC. A possible explanation of the higher incidence among females is that migraine headaches, the most common indication of TPM usage in our review, predominantly affects women.[82] The majority of cases (93.5%) were adults above the age of 18 years, and only a small proportion of cases were children, with the youngest patient in our review being a 7-year-old girl who was prescribed TPM for seizures and headache.[57] Although TiAAC usually occurs during the first two weeks after initiating TPM therapy, the onset of angle closure may occur after that time frame. In our review, Czyz CN, *et al*.[23] reported a case of delayed-onset TiAAC 262 days after starting TPM. The authors speculate that the advancement of a subclinical angle closure could be the causal mechanism behind this delayed presentation.

Table 1: A summary of baseline characteristics

SN	Reference	Age	Sex	Indication	Drug Dose mg/day)	Duration of Use Prior to Onset
1	Agarwal.[10]	25	F	weight loss	23	11 days
2	Aminlari *et al*.[21]	48	F	pain control	-	2 weeks
3	Aminlari *et al*.[21]	53	M	cluster headache	-	6 weeks
4	Arun *et al*.[32]	25	F	migraine headache	50	1 week
5	Baloch *et al*.[43]	20	F	migraine headache	-	-
6	Banta *et al*.[54]	51	M	bipolar affective disorder	150	2 weeks
7	Behl *et al*.[65]	20	F	migraine headache	50	8 days
8	Bhattacharyya *et al*.[76]	40	F	migraine headache	25	4 days
9	BMR DEO *et al*.[16]	29	F	weight loss	100	-
10	Boentert *et al*.[80]	23	F	epilepsy	50	6 days
11	Boonyaleephan.[81]	23	F	migraine headache	25	1 week
12	Braganza *et al*.[9]	19	M	migraine headache	50	2 weeks
13	Caglar *et al*.[11]	36	F	migraine headache	25	1 day
14	Chalam *et al*.[12]	34	F	migraine headache	100	1 week
15	Cole *et al*.[13]	56	F	migraine headache	50	-
16	Craig *et al*.[14]	25	F	epilepsy and depression	100	1 week
17	Craig *et al*.[14]	45	F	epilepsy	50	10 days
18	Czyz *et al*.[15]	40	F	migraine headache	100	262 days
19	Desai *et al*.[17]	36	F	migraine headache	25	10 days
20	Diaz-Cespedes *et al*.[18]	45	M	headache	50	2 weeks
21	Giuliari *et al*.[19]	13	F	migraine headache	-	1 week
22	Grewal *et al*.[20]	39	F	weight loss	23	1 week
23	Guier.[22]	27	F	migraine headache	50	2 weeks
24	Joshi *et al*.[24]	47	M	migraine headache	25	10 days
25	Kamal *et al*.[25]	32	F	migraine headache	50	-
26	Katsimpris *et al*.[26]	36	F	migraine headache	100	2 weeks
27	Kulkarni *et al*.[27]	25	F	migraine headache	25	3 days
28	Kumar *et al*.[28]	25	F	headache and insomnia	100	2 weeks
29	Kumar *et al*.[28]	18	F	headache	100	-
30	Lan *et al*.[29]	43	F	weight loss	50	1 month
31	Lan *et al*.[29]	34	F	weight loss	25	3 weeks
32	Latini *et al*.[30]	13	F	headache	25	1 week
33	Levy *et al*.[31]	35	F	migraine headache	100	1 week
34	Lin *et al*.[33]	41	F	migraine headache	50	1 week
35	Mahendradas *et al*.[34]	36	F	migraine headache	100	5 days
36	Mansoor *et al*.[35]	51	F	migraine headache	25	1 week
37	Mazumdar *et al*.[36]	38	M	migraine headache	25	1 week
38	Medeiros *et al*.[37]	44	M	bipolar affective disorders	-	3 days
39	Mitra *et al*.[38]	31	F	migraine headache	25	1 week
40	Morales-Leon *et al*.[58]	25	F	lack of satiety (side effect of olanzapine)	25	10 days
41	Natesh *et al*.[39]	23	M	migraine headache	25	5 days
42	Nizamani *et al*.[40]	24	F	migraine headache	25	A few hours
43	Osaba *et al*.[41]	45	F	migraine headache	-	1 week
44	Paciuc-Beja *et al*.[42]	39	F	migraine headache	50	1 week
45	Pai *et al*.[44]	40	M	alcohol use disorders	100	1 week
46	Palomares *et al*.[45]	29	F	migraine headache	-	1 week
47	Parikh *et al*.[46]	51	M	epilepsy	50	15 days
48	Pikkel.[47]	45	M	weight loss	50	1 week
49	Prakash *et al*.[48]	34	M	alcohol use disorders	50	1 week
50	Quagliato *et al*.[49]	55	F	migraine headache	25	1 week

Contd...

Table 1: Contd...

SN	Reference	Age	Sex	Indication	Drug Dose mg/day)	Duration of Use Prior to Onset
51	Raj *et al*.[50]	37	F	weight loss	25	2 weeks
52	Rajjoub *et al*.[51]	36	F	migraine headache	-	-
53	Rapoport *et al*.[52]	7	F	epilepsy and headache	25	2 weeks
54	Reis *et al*.[53]	40	F	alcohol use disorders	50	10 days
55	Rewri *et al*.[55]	43	F	migraine headache	50	9 days
56	Rhee *et al*.[57]	35	F	migraine headache	50	2 months
57	Rhee *et al*.[56]	43	F	migraine headache	-	1 day
58	Rosenberg *et al*.[59]	29	F	depression, anxiety, obesity, and migraine headache	-	1 week
59	Sachi *et al*.[60]	33	F	migraine headache	25	3 weeks
60	Saffra *et al*.[61]	36	F	migraine headache	25	1 week
61	Salim[62]	14	M	migraine headache	12.5	1 week
62	Santos-Nevarez *et al*.[63]	34	M	substance abuse-associated anxiety	100	10 days
63	Sbeity *et al*.[64]	59	F	migraine headache	100	11 days
64	Senthil *et al*.[66]	28	F	migraine headache	50	4 days
65	Senthilkumar *et al*.[67]	42	F	vascular headache	50	1 week
66	Sierra-Rodríguez *et al*.[68]	29	F	epilepsy	50	9 days
67	Singh *et al*.[69]	33	F	migraine headache	50	-
68	Sorkhabi *et al*.[70]	35	F	epilepsy	200	2 weeks
69	Spaccapelo *et al*.[71]	34	M	migraine headache	100	1 week
70	Stangler *et al*.[72]	40	F	migraine headache	-	10 days
71	Tambe *et al*.[73]	54	F	pain control	-	2 weeks
72	Vahdani *et al*.[74]	26	F	migraine headache	25	2 weeks
73	van Issum *et al*.[23]	34	M	epilepsy	-	2 weeks
74	Verma *et al*.[75]	17	M	migraine headache	25	10 days
75	Viet Tran *et al*.[77]	57	M	bipolar affective disorders	50	1 week
76	Willett *et al*.[78]	39	M	migraine headache	50	1 week
77	Ybarra *et al*.[79]	47	F	migraine headache	100	-

SN, serial number; M, male; F, female

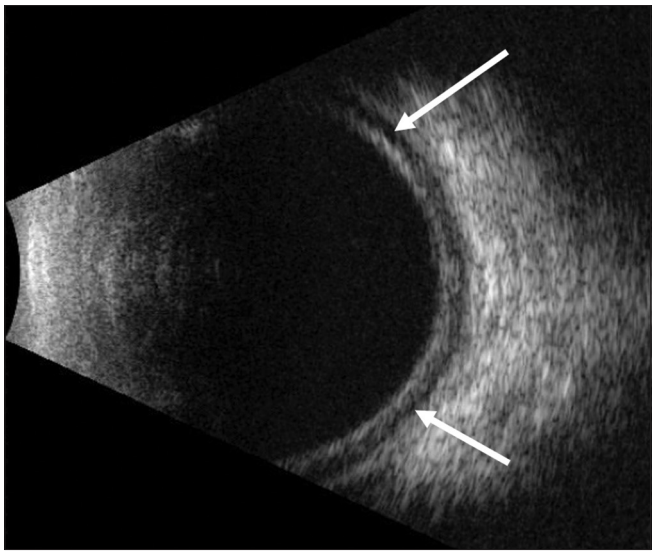

Figure 2: B-scan ultrasonography of a patient with topiramate-induced acute angle closure showing choroidal effusion (arrows)

Visual acuity at presentation was widely variable ranging from cases that had a vision as poor as light perception to patients with 20/20 vision. In patients with poor vision at presentation, it was mainly attributed to severe corneal edema induced by extremely high IOP. Most reported cases had high IOP at presentation as a result of the induced angle closure; however, five cases had normal IOP that was explained by the usage of IOP-lowering medications before presentation,[35,59] concomitant use of furosemide which is known to lower the IOP,[22] and early presentation prior to an impending attack

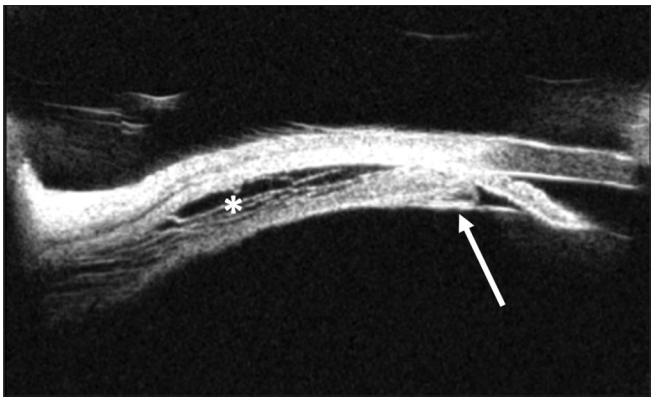

Figure 3: Ultrasound biomicroscopy of a patient with topiramate-induced acute angle closure showing an edematous, anteriorly displaced ciliary process (arrow) and uveal effusion (asterisk)

Table 2: A summary of clinical characteristics

SN	BCVA		IOP (mmHg)		SE (D)		ACD (mm)		AL (mm)		Associated Findings	Systemic Medications	Interventions	Time Until IOP Control
	OD	OS	OD	OS	OD	OS	OD	OS	OD	OS				
1	20/125	20/200	42	44	-	-	-	-	-	-	ciliochoroidal effusion	-	-	2 weeks
2	20/50	20/50	67	78	-	-	-	-	-	-	-	Hyperosmotic agent	-	1 week
3	20/400	20/400	72	68	-	-	-	-	-	-	ciliochoroidal effusion	Hyperosmotic agent, CAI	LPI OU	-
4	20/800	20/400	56	38	-	-	-	-	-	-	-	Hyperosmotic agent, CAI	-	3 days
5	20/30	20/30	14	14	-6.5	-6.5	-	-	-	-	-	-	-	4 days
6	20/200	20/200	32	38	-	-	0.9	0.9	-	-	ciliochoroidal effusion	Hyperosmotic agent, CAI	LPI OD	-
7	LP	LP	64	48	-	-	-	-	-	-	ciliochoroidal effusion	Hyperosmotic agent, steroids	-	1 day
8	-	-	22	20	-6	-5.5	-	-	-	-	ciliochoroidal effusion	Hyperosmotic agent	-	5 days
9	20/50	20/50	32	32	-7	-7	2.64	2.55	-	-	-	-	-	2 days
10	20/50	20/25	50	50	-6	-4.5	-	-	-	-	-	CAI	-	18 days
11	CF	CF	33	32	-7.5	-7.5	-	-	-	-	-	Hyperosmotic agent, CAI	-	5 days
12	20/400	20/400	40	42	-	-	-	-	-	-	ciliochoroidal effusion	CAI, steroids	-	3 days
13	20/400	20/400	68	70	-6	-6	1.75	1.72	22.21	22.31	ciliochoroidal effusion	Hyperosmotic agent, CAI	-	3 days
14	CF	CF	49	51	-	-	-	-	-	-	ciliochoroidal effusion	-	LPI OU	5 days
15	20/50	20/70	70	70	-	-	-	-	-	-	-	Hyperosmotic agent, CAI, steroids	-	-
16	20/40	20/40	40	39	-5.75	-5.25	2	1.8	-	-	ciliochoroidal effusion	-	-	1 week
17	20/20	20/20	14	14	-2.75	-2	2.1	2	-	-	ciliochoroidal effusion	-	-	-
18	CF	CF	38	37	-6.5	-7.5	-	-	-	-	-	-	-	-
19	20/20	20/20	19	20	-4.5	-5	-	-	-	-	ciliochoroidal effusion	-	-	-
20	HM	20/160	36	32	-	-	-	-	-	-	ciliochoroidal effusion	Hyperosmotic agent	-	8 hours
21	20/20	20/20	45	45	-	-	-	-	-	-	papilledema	CAI	-	1 day
22	20/25	20/25	50	52	-3.5	-3.5	-	-	-	-	ciliochoroidal effusion	Hyperosmotic agent, steroids	-	1 day
23	20/30	20/25	33	26	-5.5	-4.5	-	-	-	-	ciliochoroidal effusion	-	-	1 day
24	20/100	20/100	50	50	-5	-5	-	-	-	-	ciliochoroidal effusion	Hyperosmotic agent, steroids	-	3 days
25	20/400	20/800	37	43	-	-	1.22	1.16	-	-	ciliochoroidal effusion, uveitis	Hyperosmotic agent, CAI	-	1 day
26	20/400	20/400	60	60	-	-	-	-	-	-	ciliochoroidal effusion	-	-	5 days
27	20/400	20/400	34	32	-	-	-	-	-	-	-	CAI	-	1 day
28	20/20	20/20	10	6	-5	-5	-	-	-	-	ciliochoroidal effusion	CAI	-	-
29	20/20	20/20	25	25	-4.5	-4.5	-	-	-	-	ciliochoroidal effusion	-	-	-

Contd...

Table 2: Contd...

SN	BCVA		IOP (mmHg)		SE (D)		ACD (mm)		AL (mm)		Associated Findings	Systemic Medications	Interventions	Time Until IOP Control
	OD	OS	OD	OS	OD	OS	OD	OS	OD	OS				
30	CF	CF	56	60	-	-	2.02	1.94	23.13	23.12	ciliochoroidal effusion	Hyperosmotic agent, CAI	-	<1 day
31	20/20	20/20	26	23	-	-	2.33	2.3	22.86	22.76	-	CAI	-	<1 day
32	20/20	20/20	24	26	-8.5	-7.5	-	-	-	-	-	-	-	2 days
33	20/200	20/200	57	56	-	-	2.2	2.2	23.8	23.78	ciliochoroidal effusion	Hyperosmotic agent, CAI	-	5 days
34	20/20	20/20	44	49	-5.25	-4.75	0.8	0.8	-	-	ciliochoroidal effusion	Hyperosmotic agent	-	2 days
35	20/30	20/30	28	30	-5	-4.75	-	-	-	-	uveitis	steroids	-	1 week
36	CF	CF	38	44	-	-	-	-	-	-	uveitis	CAI	-	1 day
37	20/20	20/20	38	40	-5	-5	-	-	-	-	ciliochoroidal effusion, exudative RD	-	LPI OU	4 days
38	20/100	20/80	60	60	-	-	-	-	-	-	ciliochoroidal effusion	CAI	-	4 days
39	20/20	20/20	28	32	-3.75	-3.25	-	-	-	-	-	CAI	-	1 day
40	20/400	20/400	32	33	-9.15	-7.75	-	-	-	-	-	-	-	30 min
41	20/20	20/20	24	24	-6	-6	-	-	-	-	ciliochoroidal effusion	-	-	1 day
42	CF	CF	32	32	-	-	-	-	-	-	-	Hyperosmotic agent, CAI	LPI OU	4 days
43	20/50	20/50	40	45	-	-	-	-	-	-	uveitis	CAI	-	-
44	20/70	20/70	36	40	-1.87	-2	0.9	1.1	21.8	21.8	ciliochoroidal effusion	CAI	-	1 day
45	20/80	20/60	48	46	-	-	-	-	-	-	-	CAI	-	1 day
46	20/200	20/200	32	30	-7	-7	-	-	-	-	-	CAI	-	1 day
47	HM	HM	57	57	-	-	-	-	-	-	ciliochoroidal effusion	Hyperosmotic agent, CAI, steroids	AC paracentesis OD, choroidal drainage OD	2 weeks
48	20/100	20/100	70	64	-	-	-	-	-	-	ciliochoroidal effusion, uveitis	Hyperosmotic agent, CAI, steroids	-	1 week
49	20/400	20/400	47	43	-8	-9	-	-	-	-	-	CAI, steroids	-	3 days
50	20/25	20/40	48	48	-2	-2	-	-	-	-	-	Hyperosmotic agent, CAI	LPI OU	2 hours
51	20/50	20/50	50	56	-4.37	-4.5	1.9	1.9	-	-	-	Hyperosmotic agent	-	1 week
52	20/30	CF	31	53	-	-	-	-	-	-	uveitis	-	LPI OU, AC paracentesis OS, trabeculectomy OS	-
53	20/20	20/20	40	41	-	-	-	-	-	-	-	CAI	-	8 days
54	CF	20/400	30	30	-	-	-	-	-	-	ciliochoroidal effusion	CAI	-	2 days
55	CF	CF	28	18	-	-	0.8	0.9	20.2	20.7	ciliochoroidal effusion	-	-	-
56	20/100	20/100	88	82	-3.5	-3.5	-	-	-	-	ciliochoroidal effusion	Hyperosmotic agent, CAI, steroids	-	1 day
57	20/20	20/20	29	30	-5	-5	-	-	-	-	-	-	-	5 days
58	HM	HM	40	40	-	-	-	-	-	-	exudative RD	Hyperosmotic agent, CAI	LPI OU	1 day

Contd...

Table 2: Contd...

SN	BCVA		IOP (mmHg)		SE (D)		ACD (mm)		AL (mm)		Associated Findings	Systemic Medications	Interventions	Time Until IOP Control
	OD	OS	OD	OS	OD	OS	OD	OS	OD	OS				
59	20/200	20/200	55	34	-2.75	-3.5	2.14	2.28	22.21	22.31	ciliochoroidal effusion	CAI	-	3 days
60	20/30	20/40	24	25	-6.25	-5.5	-	-	-	-	ciliochoroidal effusion	steroids	-	1 day
61	20/20	20/20	29	29	-9	-9	-	-	-	-	ciliochoroidal effusion	-	-	-
62	20/20	20/20	56	38	-4.5	-4.5	-	-	-	-	-	CAI		1 week
63	20/30	20/100	45	43	-	-	-	-	-	-	ciliochoroidal effusion	Hyperosmotic agent, CAI	iridoplasty	1 week
64	20/30	20/30	34	34	-5	-5	-	-	-	-	-	-	-	3 days
65	20/80	20/100	60	64	-	-	-	-	-	-	ciliochoroidal effusion, uveitis	-	-	2 weeks
66	CF	CF	38	38	-13	-13	-	-	-	-	-	Hyperosmotic agent	-	a few hours
67	LP	LP	48	48	-	-	-	-	-	-	-	Hyperosmotic agent, CAI	-	3 days
68	20/30	20/30	29	31	-0.75	-0.75	1.1	1	-	-	ciliochoroidal effusion	CAI	-	1 week
69	-	-	40	40	-5.5	-5	-	-	-	-	-	CAI	-	2 days
70	CF	20/400	40	38	-4.5	-	-	-	-	-	ciliochoroidal effusion	Hyperosmotic agent, CAI	-	3 days
71	-	-	55	53	-	-	-	-	-	-	-	-	-	1 day
72	CF	CF	30	30	-5	-4	-	-	-	-	ciliochoroidal effusion	CAI	-	5 days
73	20/20	20/20	34	40	-6	-6	-	-	-	-	ciliochoroidal effusion	-	-	4 days
74	-	-	36	28	-3.5	-4	-	-	-	-	ciliochoroidal effusion	-	-	4 days
75	20/140	20/80	28	28	-3.5	-2.75	-	-	-	-	ciliochoroidal effusion, uveitis	-	-	1 week
76	20/100	20/100	70	70	-	-	-	-	-	-	ciliochoroidal effusion	Hyperosmotic agent, CAI, steroids	-	1 week
77	20/200	20/100	36	38	-	-	-	-	-	-	-	-	-	2 hours

SN, serial number; BCVA, best corrected visual acuity; IOP, intraocular pressure; SE, spherical equivalent; ACD, anterior chamber depth; AL, axial length; OD, right eye; OS, left eye; OU, both eyes; CF, counting fingers; HM, hand movement; LP, light perception; RD, retinal detachment; CAI, carbonic anhydrase inhibitor; LPI, laser peripheral iridotomy; AC, anterior chamber

of angle closure.[13,25] New-onset myopia was another clinical feature reported in association with TiAAC. It is thought to be a result of the forward movement of the lens-iris diaphragm. Another postulated mechanism is a disturbance in the osmotic state of the lens leading to swelling and subsequently a change in the refractive lens power.[83] Future studies including specific measurements of the lens thickness during and after the attack would be useful to support this mechanism. Uveitis, exudative retinal detachment, and papilledema were three other clinical signs reported in a few patients. The mechanism underlying uveitis is presumed to be an idiosyncratic reaction to TPM in which drug metabolites bind with intraocular proteins leading to a complex that is recognized as a foreign body stimulating an immune reaction,[69] whereas the occurrence of retinal detachment is thought to be related to a TPM-induced effusion of fluids into the subretinal space.[63] Finally, in the case that had TiAAC associated with papilledema, the patient was later diagnosed with pseudotumor cerebri to which the papilledema was attributed.[27]

Management of TiAAC is primarily medical consisting of IOP-lowering medications, cycloplegia, and steroids. The use of CAIs was controversial between studies. Around half of cases (51.9%) were treated with a systemic CAI to provide a further reduction in IOP, whereas CAIs were avoided in the remaining cases to prevent the occurrence of an idiosyncratic reaction that may lead to progression of the ciliochoroidal effusion and subsequently a paradoxically increased IOP.[84] Although systemic hyperosmotic therapy was only used in slightly above one-third of cases (36.4%), our analysis showed a faster resolution of TiAAC with the use of hyperosmotic treatment ($P = 0.0261$). Therefore, we recommend the use of a systemic hyperosmotic agent in TiAAC especially in refractory cases with high IOP and impending flat anterior chamber.

Given that the mechanism of TiAAC does not involve pupillary block, there is no theoretical justification for performing a LPI. Despite that, eight cases (10.4%) in our review underwent LPI during the course of treatment for various reasons. Chalam K. V., *et al*.[20] Aminlari A., *et al*.[11] and Banta J. T., *et al*.[14] performed LPI because they presumed that a relative pupillary block mechanism was present. Secondly, Rosenberg K., *et al*.[63] proceeded with LPI in their patient since visual acuity did not improve despite IOP control; however, as it eventually turned out, the patient had concomitant macular neurosensory retinal detachment that explained the visual decline. Thirdly, a plateau iris configuration was noted in the case reported by Rajjoub L. Z., *et al*.[56] for which LPI was performed. Fourthly, the choice of LPI in the cases presented by Nizamani N. B., *et al*.[46] and Quagliato L. B., *et al*.[54] was justified by attempting to provide further IOP lowering and prevent synechiae formation, consecutively. Finally, the case reported by Mazumdar S., *et al*.[42] underwent LPI before establishing a diagnosis of TiAAC as the patient was initially thought to have acute angle closure. In our opinion, LPI does not provide any benefit in cases with an established diagnosis of TiAAC and treatment should be targeted toward managing the underlying ciliary effusion. In the presence of an untreated effusion, an LPI would neither be expected to provide further IOP lowering nor will it prevent synechiae formation.

Two patients (2.6%) in our review required a surgical intervention to control their condition. The first patient[56] required trabeculectomy with mitomycin in the left eye to achieve adequate IOP control. Surgical intervention was chosen because the patient had uncontrolled IOP despite maximum medical therapy associated with clinically evident glaucomatous disc damage. Furthermore, the patient had reported a history of intermittent headache prior to starting TPM and upon examination, a plateau iris configuration was noted. These findings suggest that this patient might have had pre-existing angle closure before developing TiAAC. In the second patient, choroidal drainage was performed in the right eye because the patient progressed to a flat anterior chamber and corneal edema that necessitated urgent surgical intervention to drain the effusion and reform the anterior chamber.[51]

Limitations

Our review has some limitations. First, given that case reports only capture short periods of follow-up, our review lacks long-term clinical findings that may occur after an attack of TiAAC, such as persistent corneal edema and cataract. Second, the quality of reporting certain biometric measurements in the cited articles was low. Only a few papers presented data on ACD and AL measurements. Furthermore, the refractive error at presentation was only reported in 54.5% of cases. The presence of such information would have provided more insight into the mechanisms underlying TiAAC. Finally, given the anecdotal nature of case reports, our systematic review could not provide data on the frequency of angle closure among patients treated with TPM.

Conclusion

In conclusion, our systematic review provides an updated summary on the reported cases of TiAAC. Practitioners involved in prescribing TPM (e.g., neurologists, psychiatrists) should be alert to this possible adverse effect, especially during the first two weeks of therapy. From an ophthalmologic perspective, TiAAC should be ruled out in any patient presenting with acute angle closure by inquiring about their drug history. If diagnosed, TiAAC can be successfully managed by stopping TPM and initiating appropriate medical treatment. Surgical intervention is rarely needed as the majority of cases can be managed medically and resolved within the first few days.

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Conflicts of interest

There are no conflicts of interest.

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