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Diplopia

A Complication of Dural Puncture

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DIPLOPIA or extraocular muscle paralysis (EOMP) after dural puncture has been reported occasionally, primarily in the neurology and ophthalmology literature. Because there seems to be a window period before diplopia manifests after dural puncture, the patient and physician may not always believe that the symptom is secondary to dural puncture, particularly when it occurs after resolution of a post–dural puncture headache (PDPH). Diplopia can be so disturbing that it may prompt the patient to seek immediate medical attention. Many patients have been referred to neurologists or ophthalmologists for extensive investigation. Therefore, it is not surprising that anesthesiologists may be unaware of this complication.[1]

The first case of diplopia after dural puncture was reported by Quincke more than 100 yr ago.[2] Nonetheless, the latest major anesthesia textbooks describe little, if any, of this complication. It is important that anesthesiologists, emergency physicians, neurologists, and ophthalmologists recognize and communicate findings related to this distressing complication.

Materials and Methods

To compile case reports of EOMP associated with dural puncture, we performed a computerized search of the medical literature in English, Spanish, French, German, and Japanese from 1966 through December 20, 2002 using PubMed§ and the OVID search engine (Ovid Technologies, New York, NY). Key words used included *dural puncture*, *lumbar puncture*, *spinal anesthesia*, *spinal puncture*, *spinal injection*, *epidural anesthesia*, *myelography*, *diplopia*, *ophthalmoplegia*, *abducens nerve*, *oculomotor nerve*, and *trochlear nerve*. Forty-four related articles were identified, and 41 reports were obtained. Four reports were excluded from the review because of either insufficient descriptions of the cases or confounding factors, such as medicine or underlying disease, that could have otherwise contributed to EOMP.[3–6] An additional 12 reports were obtained after hand-searching reference lists of retrieved reports and review articles. A total of 94 reported cases and one of our own were analyzed for this review (table 1).

Results

Incidence

The reported incidence of EOMP after dural puncture varies from 1 in 400 to 1 in 8,000.[7–9] These incidence reports were either from retrospective reviews of spinal anesthesia in 1947[7] and 1961[8] or diagnostic lumbar punctures in which larger spinal needles were often used.[9] Spinal anesthesia was found to be the most frequently reported procedure involved (47%), followed by myelography (18%), diagnostic lumbar puncture (12%), epidural anesthesia/injection (11%), continuous spinal anesthesia (4%), and other dural puncture procedures (9%).

Affected Cranial Nerves

Although other cranial nerve palsies can occur after lumbar puncture, the abducens nerve (cranial nerve VI) is affected in the majority of cases (92–95%).[7,10] Nearly 80% of the cases are unilateral.[7] Abducens palsy can coexist with oculomotor (cranial nerve III) or trochlear (cranial nerve IV) nerve palsies. Multiple coexisting cranial nerve palsies can be masked by a large esotropia, making the exact diagnosis of these cranial nerve palsies difficult.[11]

Age and Sex

Extraocular muscle paralysis has been reported in patients aged 17–69 yr (mean age, 42 yr). Perhaps the largest survey of EOMP associated with spinal anesthesia was by Thorsen[7] in 1947. He reported that 80% of the patients were younger than 50 yr and 30% of the patients were younger than 30 yr, although most of the patients who had spinal anesthesia were older than 30 yr.

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§ PubMed [database online]. Available at: http://www.ncbi.nlm.nih.gov. Accessed October 9, 2003.

Table 1. Case Reports of Extraocular Muscle Paralysis after Dural Puncture

Reference	Year	Age, yr	Sex (M/F)	Mode	Needle Type	Needle Size	CN Palsy	Side	Window Period, days	Duration of EOMP or F/U*	Complete Recovery	Notes
Levine[53]	1930	24	F	SA	NR	NR	6th	R	4	4.5 mo	Yes	
Biggam[54]	1932	22	M	SA	NR	NR	6th	L	12	10 d	Yes	
		21	M	SA	NR	NR	6th	L	6	19 d	Yes	
		30	M	SA	NR	NR	6th	L	3	10 wk	Yes	
		22	M	SA	NR	NR	6th	R	2	4 wk	Yes	
		28	M	SA	NR	NR	6th	L	8	10 wk	Yes	
Hayman and Wood[10]	1942	48	M	SA	NR	NR	6th	R	5	3 wk	Yes	
		17	M	SA	NR	NR	6th	R	6	NR	Yes	
Robinson[55]	1945	26	M	LP	NR	NR	6th	R	5	3 mo	Yes	
Fairclough[37]	1945	36	F	SA	NR	NR	6th	R	5	2 mo	Yes	
		22	F	SA	NR	NR	6th	R	7	16 mo*	No	Asymptomatic
		40	M	SA	NR	NR	6th	R	7	3 mo	Yes	
		65	M	SA	NR	NR	6th	L	8	6 wk	Yes	
		38	F	SA	NR	NR	6th	R	5	3 wk	Yes	
		35	F	SA	NR	NR	6th	R	6	4 wk	Yes	
		47	M	SA	NR	NR	6th	R	11	2 mo	Yes	
		57	M	SA	NR	NR	6th	R	7	6 wk	Yes	
		50	F	SA	NR	NR	6th	L	5	6 wk	Yes	
		46	F	SA	NR	NR	6th	R	5	2 wk	Yes	
Steinberg and Bishop[56]	1946	49	M	SA	NR	NR	6th	R	4	2 wk	Yes	
Rose and Pritzker[57]	1947	37	F	SA	NR	NR	6th	R	6	3 wk	Yes	
Thorsen[7]	1947	42	M	SA	NR	NR	6th	L	A few days	6 yr*	No	
		39	M	SA	NR	NR	6th	Bilateral	A few days	NR	NR	
		47	M	SA	NR	NR	6th	Bilateral	7	5 mo	Yes	
		68	M	SA	NR	NR	6th	Bilateral	10	6 mo	Yes	
		40	F	SA	NR	NR	6th	Bilateral	8	NR	NR	
		49	F	SA	NR	NR	6th	R	NR	6 yr*	No	
		40	M	SA	NR	NR	6th	L	9	NR	NR	
		61	M	SA	NR	NR	6th	L	5	2.5 yr*	No	
Parke[58]	1948	66	M	SA	NR	22	6th	R	10	1 mo	Yes	
Tseng and Ku[38]	1950	32	NR	SA	NR	NR	6th	R	6	4.5 yr	No	Almost asymptomatic
		51	NR	CSA	NR	NR	3rd	R	5	6 mo	Yes	
Bryce-Smith and Macintoch[59]	1951	42	F	EA	Tuohy	16	6th	L	4	3 mo	Yes	
		49	F	EA	Tuohy	16	6th	R	4	1.5 mo	Yes	
Kennedy and Lockhart[60]	1952	56	F	SA	NR	NR	6th	L	1	4 mo	Yes	
		36	M	SA	NR	NR	6th	L	11	1 mo	Yes	
Naito *et al.*[35]	1970	50	M	SA	NR	20	5th and 6th	L	6	3 mo*	NR	
Liegl[61]	1977	32	M	LP	NR	NR	6th	Bilateral	4	5 mo	Yes	
		29	M	Myelo	NR	NR	3rd and 6th	L	8	7 wk	Yes	
		30	F	Supine surgery	NR	NR	3rd	L	7	6 wk	Yes	
Seyfert and Mager[62]	1978	37	M	Myelo	NR	NR	6th	Bilateral	7	3 mo	Yes	
		50	F	Myelo	NR	NR	6th	R	8	5 mo	Yes	
		36	M	Myelo	NR	NR	6th	L	4	3 mo	Yes	
		35	M	Myelo	NR	NR	6th	Bilateral	9	5 wk	Yes	
		50	F	Myelo	NR	NR	6th	L	16	5 mo	Yes	
Huismans[63]	1979	55	M	SA	NR	NR	6th	L	5	6 wk	Yes	
Gupta *et al.*[64]	1980	29	F	CSA	Tuohy	18	6th	R	5	2 mo	Yes	
Insel *et al.*[65]	1980	60	F	LP	NR	22	6th	R	4	1.5 mo	Yes	
Newmark *et al.*[39]	1981	27	M	LP	NR	18	NR	NR	8	1 mo	Yes	
		20	F	Pneumo	NR	NR	6th?	R	7	10 yr*	No	Asymptomatic
Heyman *et al.*[31]	1982	34	F	EA	Tuohy	17	6th	R	3	6 wk	Yes	
Miller *et al.*[66]	1982	29	M	Myelo	NR	NR	6th	Bilateral	6	5 mo	Yes	
		33	F	Myelo	NR	NR	6th	Bilateral	14	5 mo	Yes	
		56	M	Myelo	NR	NR	6th	Bilateral	21	4 mo	Yes	
Arne *et al.*[67]	1982	24	F	EA	NR	NR	6th	R	7	2.5 mo	Yes	
Moster *et al.*[68]	1984	25	F	EA	NR	NR	6th	NR	5	3 mo	Yes	
		36	M	Myelo	NR	NR	6th	NR	4	1 mo	Yes	
Perlman and Barry[69]	1984	36	M	LP	NR	21	6th	Bilateral	7	2 mo	Yes	
Salazar *et al.*[70]	1985	58	M	SA	NR	22	6th	R	8	1 mo	Yes	
Hotton and Hummel[71]	1986	56	M	SA	NR	18	6th	R	6	8 wk	Yes	
King and Calhoun[11]	1987	33	F	SA	NR	NR	4th and 6th	R	6	13 mo*	No	6th nerve recovered
Montalban *et al.*[19]	1988	25	NR	Myelo	NR	NR	6th	Bilateral	6	6 wk	Yes	

(*Table continues*)

Table 1. *Continued*

Reference	Year	Age, yr	Sex (M/F)	Mode	Needle Type	Needle Size	CN Palsy	Side	Window Period, days	Duration of EOMP or F/U*	Complete Recovery	Notes
Richer and Ritacca[20]	1989	58	M	SA	NR	NR	6th	R	21	4 mo	Yes	
Justo Firvida *et al.*[72]	1989	53	NR	SA	NR	22	6th	R	4	5 mo	Yes	
Whiting *et al.*[73]	1990	63	F	SA	NR	22	3rd and 6th	L	8	2 mo	Yes	
		47	F	CSA	Tuohy	17	6th	Bilateral	6	4 mo	Yes	
		56	M	CSA	Tuohy	17	6th	L	7	3 mo	Yes	
Bell *et al.*[74]	1990	33	F	LP	NR	NR	6th	L	1	2 wk	Yes	
Balseiro Gomez *et al.*[75]	1991	61	M	SA	NR	NR	6th	Bilateral	14	9 mo*	NR	Symptom improvement
Dierking *et al.*[76]	1991	60	M	SA	NR	25	6th	L	5	11 wk	Yes	
Espinosa *et al.*[33]	1993	69	M	VA shunt	NR	NR	6th	R	9	2 mo	Yes	
		69	M	VP shunt	NR	NR	6th	Bilateral	7	11 mo*	No	Surgery performed
		67	M	VP shunt	NR	NR	6th	Bilateral	14	11 wk	Yes	
Bell *et al.*[77]	1994	39	F	Myelo	NR	NR	6th	L	10	6 mo	Yes	
		46	F	Myelo	NR	NR	6th	R	12	4 mo	Yes	
		41	F	Myelo	NR	NR	6th	L	7	3 mo	Yes	
		33	F	Myelo	NR	NR	6th	L	1	2 wk	Yes	
		47	F	Myelo	NR	NR	6th	L	5	3 mo	Yes	
Dunbar and Katz[30]	1994	26	F	EA	Tuohy	17	6th	NR	8	3 mo	Yes	
		31	M	LP	NR	20	6th	Bilateral	10	4 mo	Yes	
De Veuster *et al.*[32]	1994	47	F	EA	NR	NR	6th	L	7	2 mo	Yes	
Dinakaran *et al.*[78]	1995	47	F	Myelo	NR	NR	6th	R	6	4 mo	Yes	
Brocq *et al.*[79]	1997	44	F	E inj	NR	NR	6th	NR	2	6 mo	Yes	
		54	F	IT inj	NR	NR	6th	NR	8	6 mo	Yes	
Johnson *et al.*[34]	1998	32	F	EA	Tuohy	16	6th	L	10	1 yr*	No	Surgery performed
Dumont *et al.*[14]	1998	38	F	IT inj	NR	NR	6th	R	8	4 mo	Yes	
Szokol and Falleroni[18]	1999	32	F	EA	Tuohy	17	6th	L	4	8 wk	Yes	
Thomke *et al.*[9]	2000	61	M	LP	NR	22	6th	R	7	4 mo	Yes	
		33	M	LP	NR	22	6th	Bilateral	9	7 mo	Yes	
Romero Aroca *et al.*[80]	2000	58	M	SA	NR	NR	6th	R	2	3 wk	Yes	
Velarde *et al.*[25]	2000	58	M	IT pump	Tuohy	14	6th	L	6	3 wk	Yes	
Vial *et al.*[48]	2001	26	F	SA	Whitacre	25	6th	L	3	8 mo	Yes	
Follens *et al.*[13]	2001	37	M	LP	NR	NR	4th and 6th	R/L	5	18 mo*	No	Surgery performed
Niedermuller *et al.*[21]	2002	43	M	LP	Yale	20	6th	L	4	4 mo	Yes	
Nishio *et al.*	2002	59	M	SA	Whitacre	25	6th	R	10	3 mo	Yes	
Mean		42	M/F = 50/41				R/L/Bilateral = 38/32/18		6.9	2.8 mo	Recovery rate = 89%	
SD		13							3.6	1.8 mo		

* Follow-up.

CN = cranial nerve; CSA = continuous spinal anesthesia; EA = epidural anesthesia; E inj = epidural injection; EOMP = extraocular muscle paralysis; IT inj = intrathecal injection; IT pump = intrathecal drug delivery pump; LP = diagnostic lumbar puncture; Myelo = myelography; NR = not reported; Pneumo = pneumoencephalography; SA = spinal anesthesia; VA = ventriculoatrial; VP = ventriculoperitoneal; Window period = period between dural puncture and the onset of extraocular muscle paralysis.

The incidence of PDPH in women has been reported to be twice as high as in men.[12] This sex pattern does not hold true for EOMP: Thorsen[7] (1947) reported a predilection of abducens nerve palsy for men, whereas Hayman and Wood[10] (1942) stated that women were more susceptible. In our review, no significant sex predilection of this complication was found (male *vs.* female: 55% *vs.* 45%, respectively).

Diagnosis

The window period for EOMP to manifest is 1 day to 3 weeks after dural puncture, but it most often presents 4–10 days after dural puncture (mean, 7 days; median, 6 days) (table 1 and fig. 1). This finding is consistent with classic reports.[7,10] EOMP associated with dural puncture is almost always preceded by PDPH, but EOMP can occur either before or after the headache subsides. If the cranial nerve (III, IV, or VI) palsy is isolated, is preceded by PDPH, and occurs within 3 weeks after dural puncture with no other neurologic deficits, it is likely that cranial nerve palsy is a postdural puncture complication. Diagnosis of this complication is based purely on clinical presentation, and there is no specific test for its accurate diagnosis.

Magnetic resonance imaging of the brain has occasionally shown signs of cerebrospinal fluid (CSF) volume depletion and intracranial hypotension, such as diffuse pachymeningeal enhancement, descent of the brain-

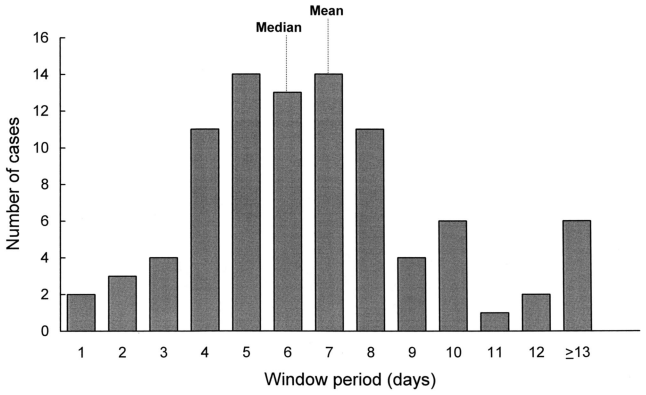

Fig. 1. Window period, onset of extraocular motor paralysis after dural puncture. This most often occurs 4–10 days after dural puncture; however, it can manifest as late as 3 weeks. It took 14 days in three cases,[33,66,75] 16 days in one case,[62] and 21 days in two cases[20,66] for extraocular motor paralysis to manifest.

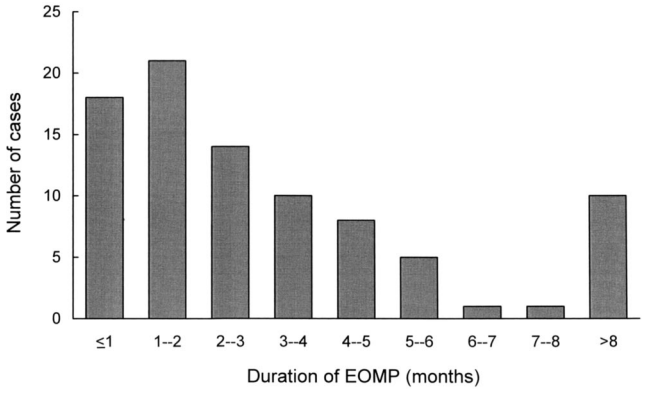

Fig. 2. Duration of extraocular motor paralysis (EOMP). EOMP that lasted more than 8 months was found to be permanent.

stem, and subdural fluid collections.[9,13,14] However, these findings are not specific for EOMP after dural puncture and can be seen in spontaneous intracranial hypotension.[15–17] There may be no abnormality found[18–21] when magnetic resonance imaging is performed after PDPH resolution.

The differential diagnosis of acquired EOMP is varied, such as neoplasm, ischemia, trauma, aneurysm, multiple sclerosis, encephalitis and myasthenia gravis. The greatest proportion of abducens nerve palsies are of unknown origin; however, the overall spontaneous recovery rate is close to 80%.[22,23] Despite the good prognosis of the nerve palsy as well as the low yield and low specificity of diagnostic studies for this complication, magnetic resonance imaging of the brain may still be of value to rule out other serious conditions that require treatment. Indeed, subdural hematoma or hygroma can rarely occur after dural puncture, from tearing of the bridging dural veins associated with acute intracranial hypotension.[24–28] Subdural hematoma should be included in the differential diagnosis if prolonged PDPH loses the postural dependence of symptoms and/or is accompanied by other neurologic signs.

Treatment and Prognosis

An epidural blood patch is highly effective for PDPH, with the reported success rate being as high as 93% for the first attempt,[29] but it has consistently failed to show efficacy in treating EOMP after dural puncture.[13,18,30–32] Abducens palsy is associated with a favorable outcome in general, and its prognosis after dural puncture is even better. In our review, 80 of 90 patients (89%) fully recovered in 2 weeks to 8 months (table 1). The majority of those recovered within 6 months (figs. 2 and 3), consistent with classic reports.[7,10]

Conservative treatment (such as an eye patch or prism glasses) is generally adequate to minimize the patient's discomfort. Isolated abducens palsy in the absence of other neurologic signs or symptoms should be observed for improvement for 8 months. Further investigation is unwarranted if the deficit resolves spontaneously.

In our review, EOMP cases that lasted more than 8 months were found to be permanent. No apparent trend was found among the cases of permanent EOMP, probably because of a small number of cases and the lack of detailed description in some of the reports (*e.g.*, size and type of dural puncture needle). Corrective surgery on the extraocular muscles, such as recession of the medial rectus muscle, was performed in some patients, allowing them to resume social activities or previous occupations.[13,33,34] However, some suggest that surgical correction be postponed until at least 18 months have lapsed, considering cases of protracted recovery.[35,36] Indeed, some patients became asymptomatic after several months to years, although residual hyperdeviations persisted.[37–39] The decision should be individualized based on the duration (at least longer than 8 months) and severity of symptomatic EOMP as well as the risks and benefits of the corrective surgery.

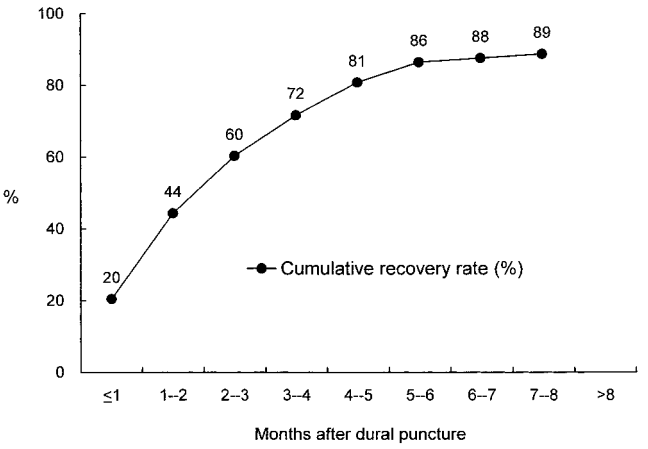

Fig. 3. Cumulative recovery rate of extraocular motor paralysis after dural puncture. The majority of patients who recovered did so within 6 months after dural puncture. No spontaneous recovery was reported after 8 months.

Etiology and Proposed Pathologic Mechanisms

Similar to PDPH, intracranial hypotension due to CSF leakage is the generally accepted cause of cranial nerve palsies after dural puncture because this complication can occur after diagnostic lumbar puncture where no medication is injected into the intrathecal space. Spontaneous intracranial hypotension with orthostatic headache that occasionally presents as diplopia (commonly abducens nerve palsy) supports the hypothesis.[15–17] The brain descends caudad with upright posture, and the CSF "cushion" for the brain is displaced. This downward traction could damage some of the cranial nerves that anchor the brain in the skull.

The time course (of weeks to months) for the cranial nerve palsies with good prognosis suggests neurapraxia (focal segmental demyelination) or axonotmesis (axonal interruption and Wallerian degeneration with preservation of supporting tissue framework) as a potential pathologic mechanism.[40] Even after axonotmesis, electrical activity and conductivity may be present in the axonotmetic distal stump for a day or two, but the axon then quickly becomes unresponsive with degeneration.[41,42] This may explain why an epidural blood patch is not effective in treating the cranial nerve palsies. There are both functional disturbances and structural lesions in the nerve by the time the nerve palsy manifests.

Preferential damage to the abducens nerve can be explained by its anatomic course. As the nerve emerges into the subarachnoid space from the caudal pons, it immediately ascends the clivus, crosses branches of the basilar artery, and pierces the dura mater. Then, the nerve bends at nearly a right angle over the petrous apex of the temporal bone.[43,44] The abducens nerve runs in the direction of the typical caudad displacement of the brain with intracranial hypotension. As a result, traction associated with changes in intracranial pressure is fully transmitted to the nerve. The nerve can be stretched by caudal displacement of the pons, and it also may be compressed at the dura, petrous apex, or basilar artery if (1) the penetration aperture for the nerve in the dura or petrous apex is sharply edged or (2) branches of the basilar artery are well developed.

Ikeda *et al.*[45,46] have shown that a combination of nerve stretch and compression even to a mild degree can be more detrimental to a nerve than either alone, causing severe axonotmesis, with not only Wallerian degeneration but also retrograde degeneration from the injured site ("dying-back degeneration"). The wide variation in duration of the nerve palsies may be associated with varying degrees of nerve injuries from mild neurapraxia with conduction block to severe axonotmesis with extensive degeneration.

Prevention

After EOMP occurs, little can be done to change its course. Therefore, prevention is of great importance. Vandam and Dripps[47] reported in their survey of 10,098 patients undergoing spinal anesthesia that diplopia (secondary to probable abducens nerve palsy) occurred only in patients who underwent continuous spinal anesthesia with a 16-gauge needle. The incidence of EOMP was high (1:140). Because intracranial hypotension associated with CSF leakage seems to play a major role in the pathogenesis, minimizing CSF leakage with smaller, pencil-point needles should reduce the risk of EOMP. However, EOMP can occur after otherwise uncomplicated spinal anesthesia using a 25-gauge Whitacre needle.[48]

Bed rest has been advocated in cases of dural puncture by some clinicians. However, a recent meta-analysis failed to show that bed rest after dural puncture was better than immediate mobilization in reducing the incidence of PDPH.[49] Bed rest can be associated with a higher incidence of PDPH in particular patient groups.[50,51] In theory, however, upright posture may exacerbate compression–stretch injury of the abducens nerve by promoting further caudad displacement of the brain. Because of the low incidence of EOMP, it seems unlikely that the value of routine bed rest in an effort to prevent EOMP will ever be determined.

Does early application of an epidural blood patch after dural puncture prevent cranial nerve palsy from occurring by restoring intracranial pressure? There are no studies to support this idea, nor is such a study feasible because of the low incidence of this complication. EOMP could occur shortly after an epidural blood patch procedure for PDPH because of the slow manifestation of EOMP. In this circumstance, the blood patch procedure may be blamed for abducens nerve palsy if diplopia was not recognized as a complication of dural puncture. On the other hand, a blood patch can actually cause EOMP and/or aggravate neurologic symptoms in a patient with PDPH or spontaneous intracranial hypotension when the mass effect of the coexisting subdural hematoma can no longer be compensated by the disappearance of CSF leak by the patch.[28,52] A thorough history and physical examination assessing signs or symptoms suggestive of subdural hematoma is mandatory before an epidural blood patch procedure, and close observation after the procedure is strongly recommended. Should mental status changes or any neurologic signs/symptoms manifest, the patient will need immediate medical attention and imaging studies.

Conclusion

Although the current incidence of EOMP after dural puncture is unknown, it can occur with smaller pencil-point spinal needles. Abducens nerve involvement is most often unilateral. EOMP seems to be very rare in elderly patients, and male and female patients seem to be equally vulnerable. EOMP usually occurs 4–10 days after

dural puncture but can manifest as late as 3 weeks. Full recovery can generally be expected in 2 weeks to 8 months, although permanent cases have rarely been reported. Anesthesiologists, emergency physicians, neurologists, and ophthalmologists should be aware of this complication and communicate the information so that early diagnosis can alleviate patient anxiety. The exact pathophysiology is unclear, but a nerve lesion such as neurapraxia or axonotmesis caused by stretch and/or compression secondary to intracranial hypotension due to CSF leakage is the generally accepted mechanism. Treatment is supportive except for persistent or permanent cases, for which corrective surgery may be necessary. An epidural blood patch does not seem to be an effective treatment, whereas the benefit of a prophylactic blood patch is unknown. Avoiding, if possible, or minimizing CSF leakage associated with dural puncture may be the only measure for now to potentially minimize the risk of this rare but distressing complication.

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