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Reference:

Verslegers Lieven, Schotsmans Katlijn, Montagna Massimiliano, Feyen Bart, De Jong Lars, Crols Roel, Engelborghs Sebastiaan.- Severe bilateral subdural hematomas as a complication of diagnostic lumbar puncture for possible Alzheimers disease
Clinical neurology and neurosurgery - ISSN 0303-8467 - 152(2017), p. 95-96
Full text (Publisher's DOI): <https://doi.org/doi:10.1016/J.CLINEURO.2016.12.003>
To cite this reference: <http://hdl.handle.net/10067/1406700151162165141>

Severe bilateral subdural hematomas as a complication of diagnostic lumbar puncture for possible Alzheimer's disease.

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Introduction

Post lumbar puncture headache (PLPH) is a benign condition and the most frequent complication of spinal anesthesia or lumbar puncture (LP). Subdural hematoma (SDH) is a rare but potentially life-threatening complication. The true incidence of post-LP SDH is unknown¹. The multicenter study on LP feasibility showed that diagnostic LPs can safely be performed in memory clinics. Severe complications were very rare (0.7% of the study population) and no SDH was reported². Only a few weeks after the multicenter LP study ended and to which our center participated, we diagnosed a severe bilateral SDH in a patient who underwent diagnostic LP for possible Alzheimer's disease (AD).

Case report

A 39-year old woman presented with progressive cognitive decline characterized by progressive loss of short-term memory, disorientation in space and word-finding problems. Her medical history consisted of Ehlers-Danlos syndrome (EDS), classic type, with a history of a treated cerebral aneurysm. The patient is known to have a translocation between chromosomes 9 and X. Early onset familial AD was suspected, also because of the familial history (mother: Alzheimer's disease (AD) at onset age 60; a DNA analysis was not performed). Blood sampling (including platelet count and coagulation status) was normal. The patient did not take anticoagulants or antiplatelet drugs. The pre-LP brain MRI scan did not show a SDH or any other contra-indication for LP. In order to analyze AD cerebrospinal fluid (CSF) biomarkers ($A\beta_{1-42}$, tau, P-tau_{181p}), an LP was performed, with the subject in lying position using a 20 Gauge, 3.5 inch Quincke point spinal needle. The LP succeeded after one attempt and a total CSF volume of 20mL was collected (free flow) as the subject participated in a CSF biomarker research protocol. During the month following LP, the patient developed progressively worsening headache without neurological deficits. The patient was admitted to the hospital at 46 days post-LP because of reduced consciousness (Glasgow Coma Scale 13/15). Brain CT scan revealed bilateral SDH (right >left) with a midline shift to the left (figure 1). After trepanation and evacuation of the SDH that consisted of old bloody fluid with membranes, the patient developed recurrent epi- and

subdural bleedings at the right side urging three consecutive surgical evacuations. Ten days later she was discharged with only a mild residual left hemiparesis (Medical Research Council Scale 4+/5). At long term follow up no neurological deficits were present anymore. Unfortunately, due to low grade wound infection revision surgery was necessary. The patient underwent a complete and detailed coagulation analysis, which was normal.

The final diagnosis was possible dementia due to AD despite the fact that the CSF biomarkers ($A\beta_{1-42}$, tau, P-tau_{181P}) were normal. Arguments in favor for this diagnosis are the progressive nature of the cognitive deterioration (as substantiated by repeated neuropsychological examinations), the brain MRI scan showing cortical, mesio-temporal and hippocampal atrophy, the brain perfusion SPECT scan showing a relative hypoperfusion of the frontal, temporal and parietal brain regions as well as clinical symptoms of REM sleep behavior disorder. The diagnosis of AD is supported by an $\epsilon 4\epsilon 4$ *APOE* genotype. DNA analysis is ongoing but analysis of *PSEN1* and *APP* was normal.

Discussion

SDH is a very rare complication following diagnostic LP: not a single case was reported in the multicenter LP feasibility study (n=3422)². More than 50 cases with post-LP SDH have been reported following spinal anesthesia^{1,3}. The pathophysiological mechanism for post-LP SDH is not fully understood. It has been hypothesized that CSF leakage causes a combination of lower intraspinal and intracranial pressure together with congestion, dilatation, and tearing of the subdural veins. This could explain the occurrence of SDH in patients with ventriculoperitoneal shunts and in those with spontaneous CSF hypovolemia, where the ICP is chronically low. However, in case of spinal anesthesia, the volume of CSF collected is minimal in contrast to diagnostic LPs for CSF analysis, in which the risk of consequent intracranial hypotension is higher. In diagnostic LP's the extent of leakage is directly related to the size of the needle, so the risk of PLPH and post-LP SDH can be decreased by using small needles. The use of larger needles used in case of spinal anesthesia could

possibly explain the discrepancy in prevalence of SDH following LPs for spinal or epidural anesthesia as compared to diagnostic LPs.

EDS is a clinical and genetic heterogeneous disorder, consisting of a variable combination of dermal fragility, internal organ and vessel ruptures and joint hypermobility. Possibly, EDS has contributed to the development of post-LP SDH in our patient. Most previously reported cases of post-LP SDH were unilateral ; however, our case involved massive bilateral subdural hematomas. This finding may be attributable to the volume of CSF collected and possibly to the connective tissue disorder⁴. No similar cases of post-LP SDH in a patient with a history of EDS have been reported. In patients suffering from EDS we presume that the brittle nature of the connective tissue is a supplementary risk for developing post-LP SDH. A causal link is possible as also spontaneous CSF leaks in patients with connective tissue disorders have been reported⁵.

PLPH is the most frequent and benign complication of spinal anesthesia and lumbar puncture. Headache is the most common symptom of both PDPH and post-LP SDH. This common feature usually delays or masks the diagnosis of post-LP SDH, potentially resulting in morbidity or mortality. Patients with PLPH must therefore be examined carefully. In our patient, prolonged and severe headache and reduced consciousness were indications to perform a brain CT scan. Patients developing PLPH unrelieved by conservative measures, as well as the change from postural to non-postural, require careful follow-up for early diagnosis and management of possible subdural hematoma.

The management of subdural hematoma is either conservative or surgical. Hematomas of <5 mm usually resolve spontaneously; therefore, close clinical observation is the best choice. Surgery should be performed if neurological impairment or significant brain compression is present. However, in patients with EDS the higher risk of diffuse bleeding and increased time requested for soft-tissue repair after surgery should be taken into account.

Conclusion

Although SDH has not been reported in a large multicenter study on feasibility of diagnostic LPs in a memory clinic setting, we here report a case with severe bilateral SDH following a diagnostic LP for possible AD. EDS and the relatively large volume of CSF sampled might have contributed to the development of SDH as complication of a diagnostic LP in this case.

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Figure 1 - Brain CT scan showed bilateral subdural hematomas more extensive at the right side (white large arrow) than at the left (white short arrow) with midline shift to the left.