

Lumbar Subarachnoid Hemorrhage In A Parturient With HELLP Syndrome Revealed By But Unrelated To Spinal Anesthesia; A Review Of Differential Diagnosis Of Spinal Subarachnoid Hemorrhage In Pregnancy.

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Citation

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Abstract

A 32 year old female with pre-eclampsia presented for cesarean delivery and was found to have leg weakness reported after spinal attempts. MRI revealed lumbosacral subdural and subarachnoid blood felt to be unrelated to the neuraxial anesthesia attempts. The main cause of spinal subarachnoid/subdural hemorrhage in pregnancy is generally a result of neuraxial anesthesia. However, in this case, the lower extremity symptoms preceded the spinal anesthesia attempts. The subarachnoid blood was felt to have an alternative etiology, prompting a thorough review of the differential diagnosis of subarachnoid hemorrhage in the parturient.

INTRODUCTION

Spinal subarachnoid hemorrhages in pregnancy are rarely occurring events most of which are reported following neuraxial anesthesia (NA) or in conjunction with bleeding disorders, subarachnoid cysts or A-V malformations [1]. Reports are limited to intracranial subarachnoid and subdural hemorrhages as opposed to the lumbosacral hemorrhage encountered in this case [2-6]. In our case, a parturient presented with leg weakness and paresthesia and was found to have HELLP syndrome: Hemolysis (H), elevated liver enzymes (EL) and low platelets (LP). Neuraxial anesthesia was attempted and due to continued patient complaints an MRI was done revealing the subarachnoid hemorrhage and subdural hematoma.

CASE PRESENTATION

A 32 year old parturient, at 30 weeks gestation presented to a local hospital with complaints of leg weakness and numbness in addition to a severe headache the preceding week and had abated. With severe edema in all extremities, headache, blood pressure of 202/143 and platelet count of 92k/mm³/L she was transferred to our institution for evaluation. Her BMI was 46.9, she complained of back pain and a left lower extremity weakness was documented. A Mallampati II was noted, as was a thick neck circumference.

Her blood pressure was 198/133. She received 40 mg of intravenous labetalol and was started on an infusion of magnesium. Laboratory examination: WBC 18.1k/mm³, Hct 44.6%, RDW 15.9, AST 118 u/L, LDH 536 u/L, ALT 74 u/L, INR 0.9, PT 9.9, APTT 27.8, Fibrinogen 301, D-Dimer 3.28 mg/L. A semi urgent c-section was called for worsening pre-eclampsia. After three unsuccessful attempts at spinal anesthesia, general anesthesia was performed. Postoperatively, the patient complained of leg weakness and had a sensory deficit. Neurology and neurosurgical consultations were obtained revealing mild weakness and sensory deficit in the lower left lateral extremity. MRI with and without gadolinium demonstrated T2 signal hypointensity within the ventral and left lateral aspect of the thecal sac at the L5-S1 lumbosacral region suggestive of a subarachnoid hemorrhage with a subdural component (Fig1A,2A). The patient was monitored closely and reported waning symptoms in the days following delivery. The headaches and edema began to subside and the platelet count at discharge had improved to 153k/mm³/L. She was re-evaluated by neurosurgery after six weeks with symptoms consisting of mild paresthesias. Six week repeat MRI showed resolution of the subarachnoid hemorrhage and delineated conus medullaris clumping and arachnoiditis (Fig 1B,2B). After 12 weeks, she returned for follow up with

only mild weakness in left hip flexion and knee extension as compared to the right. 12 weeks post-symptoms, MRI with and without gadolinium revealed leptomeningeal enhancement of the left L4-L5 nerve roots consistent with arachnoiditis in the setting of a resolved subarachnoid hemorrhage (Fig1C,2C). MRI with gadolinium enhancement at 5 months post spinal anesthesia shows continued enhancement of the left L4-L5 roots suggestive of arachnoiditis. Neurosurgical evaluation with repeat MRI at 5 months was consistent with arachnoiditis and her occasional mild pain was controlled by oral gabapentin.

Figure 1

Figure 1: A- Axial T2 weighted MRI revealing signal hypo-intensity at the L5-S1 thecal sac (white arrow) performed at initial presentation. B - Axial T2 weighted MRI with signal hyper-intensity at the L5-S1 region (white arrow) felt to represent collected but resolving heme (Image at 6 week follow-up). C- Axial T2 weighted MRI with mild linear enhancement at the left L4-L5 region suggestive of arachnoiditis in the setting of a resolved subarachnoid hemorrhage (Image from 12 week follow-up).

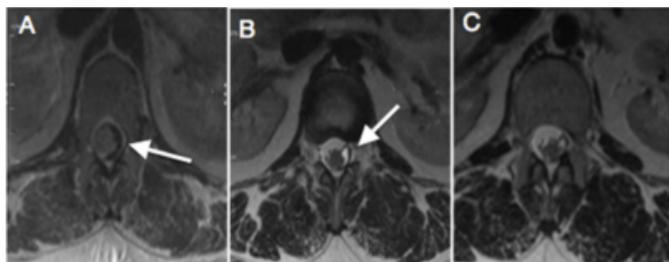


Figure 2

Figure 2: A- Sagittal T2 weighted MRI with hypo-intense areas surrounding the conus medullaris at the lumbosacral region (Image on initial presentation). B- Six week follow-up sagittal T2 weighted MRI and C- twelve week follow-up sagittal T2 weighted MRI with gadolinium contrast both showing improvement of signal hypo-intensity compared with initial presentation.



DISCUSSION

Early identification of spinal subdural and subarachnoid hemorrhage following NA is of utmost importance as failure to diagnose/treat can lead to irreversible complications or death. The estimated prevalence of peripartum SAH is 5.8 cases in 100,000 pregnancies and has steadily increased over the past two decades (7). The majority of atraumatic hemorrhages in pregnancy are intracranial and generally present with a severe headache [1]. Rare causes include DIC, anticoagulation therapy, cyst rupture and platelet disorders. However, clots in the lumbosacral region are extremely rare as CSF drainage in this area tends to dilute and remove blood prior to clot formation [1,8]. Preeclampsia and eclampsia are major causes of spontaneous intra-cerebral hemorrhage during pregnancy. Subarachnoid hemorrhage can be divided into bleeds occurring inside the cranium or those outside or extra axially including subdural and epidural hematomas or subarachnoid hemorrhage. In a large nationwide thirteen-year study of peripartum subarachnoid hemorrhage, Bateman et al. identified risk factors for all cause bleeding in this subset of patients. Forty percent of patients with pregnancy related SAH were found to have hypertensive disorders compared with 7.6% in the control group, providing an odds ratio of 5.97-8.24 [7]. Additionally, patients with pregnancy related SAH had a 40.1% prevalence of hypertensive disease compared with a 29.5% prevalence of hypertensive disorders in non-pregnancy related SAH in the general population [7]. Other significant risk factors for intracranial or extra axial SAH were: age greater than 25 years, African American or Hispanic race, coagulopathy, tobacco or drug use [7]. Severe hypertension is associated with a significant risk of SAH in pregnancy and may result from ruptured pia-arachnoid vessels (9). In the consensus guideline report, 16 cases of post neuraxial bleeding were reported 9 of which occurred in the setting of coagulopathy [10]. Moen et al, reported 2 cases of spinal hematomas both occurring in parturients with HELLP syndrome [11]. In this case, the patient's blood pressure was 202/143 and with the relatively low platelet count may be the inciting factor. A number of review articles suggest an increase in the rate of neurological complications following NA in patients with preexisting spinal pathologies [12,13]. Hebl et al. estimate the increased risk for complications following NA in these patients to be 1.1% [13].

The practice guidelines provided by the American Society of Anesthesiologists task force on obstetric anesthesia for does

not specify a platelet level at which they deem neuraxial anesthesia to be safe, rather the decision is deferred to the clinician [14]. It has been suggested that NA may be considered safe for if the platelet count is 50-100k/mm³/L or greater and without evidence of DIC; our patient had 92K/mm³/L at the time of NA [15,16]. In this case, given the onset of symptoms prior to placement of the neuraxial block, it is likely for the hemorrhage to have occurred in the days prior to delivery in the setting of hypertension and low platelets. The severe headache the week prior to delivery may indicate the timing of the neurologic insult. Given the left sided symptoms which preceded neuraxial anesthesia and left sided arachnoiditis suggested by MRI, it would seem that the spinal attempts revealed the preexisting neurological injury. The heme may have resulted from a hypertensive vascular rupture in the setting of pre-eclampsia. The definitive cause for the onset of the lower extremity symptoms remains clandestine. The most common cause for heme occurring in this area is generally a result of NA however we must be vigilant to identify other factors in the differential diagnosis to assure proper treatment.

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