A 19-year-old at 37 weeks gestation with an acute acetylsalicylic acid overdose

The Harvard community has made this article openly available. Please share how this access benefits you. Your story matters

<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Published Version</td>
<td>doi:10.1093/ndtplus/sfr104</td>
</tr>
<tr>
<td>Citable link</td>
<td><a href="http://nrs.harvard.edu/urn-3:HUL.InstRepos:16120889">http://nrs.harvard.edu/urn-3:HUL.InstRepos:16120889</a></td>
</tr>
<tr>
<td>Terms of Use</td>
<td>This article was downloaded from Harvard University’s DASH repository, and is made available under the terms and conditions applicable to Other Posted Material, as set forth at <a href="http://nrs.harvard.edu/urn-3:HUL.InstRepos:dash.current.terms-of-use#LAA">http://nrs.harvard.edu/urn-3:HUL.InstRepos:dash.current.terms-of-use#LAA</a></td>
</tr>
</tbody>
</table>


Case Report

A 19-year-old at 37 weeks gestation with an acute acetylsalicylic acid overdose

Huma Farid1, Monica H. Wojcik1 and Kenneth B. Christopher1,2

1Harvard Medical School, Boston, MA, USA and 2Renal Division, Brigham and Women’s Hospital, Boston, MA, USA

Correspondence and offprint requests to: Huma Farid; E-mail: hfarid@post.harvard.edu

Abstract

Acute salicylate overdose in pregnancy is potentially fatal for both the mother and fetus and presents a unique challenge in intensive care management. While suggested thresholds exist for hemodialysis in adults with toxic salicylate ingestion, it is unclear if these thresholds remain appropriate for the gravid patient, particularly given that medications such as acetylsalicylic acid may cross the placental barrier and accumulate in the fetal bloodstream. We describe a case of a gravid patient at ~37 weeks gestational age with a self-reported acetylsalicylic acid ingestion of 32.5 g and review prior cases of both acute and chronic salicylate ingestion in pregnancy in order to determine the clinical precedent for hemodialysis in this situation.

Keywords: acetylsalicylic acid; hemodialysis; overdose; pregnancy; salicylate toxicity

Case report

A 19-year-old, gravida 1, para 0 woman at 39 weeks gestation with a past medical history notable for mild mental retardation, bipolar disorder and hypothyroidism presented to an outside hospital with bloody emesis and tinnitus after a self-reported ingestion of 100 tablets (32.5 g) of aspirin several hours prior. Upon initial presentation, she was agitated and tachypneic, with a respiratory rate of 40 breaths/minute and tachycardic, with a heart rate of 120–130 beats/minute, but otherwise hemodynamically stable. Her laboratory data were notable for a salicylate level of 57.5 mg/dL, bicarbonate of 16 mmol/L and an anion gap of 4. An ultrasound showed a fetal heart rate of 160–170 beats/minute. The patient was given bicarbonate and glucose and transferred to the medical intensive care unit, where she was continued on a bicarbonate drip for urine alkalinization. A repeat salicylate level was 41.69 mg/dL with an arterial pH of 7.60. She remained persistently alkalotic due to the bicarbonate drip. Coagulation studies, including prothrombin time (PT), activated partial thromboplastin time (PTT) and international normalized ratio (INR), were all within normal limits. Her hematocrit was 37% and platelets were 487 000/mm3. The patient remained confused and agitated and continued to complain of tinnitus. A series of her serum salicylate levels is shown in Figure 1.

As the threshold for hemodialysis for aspirin overdose at our institution is a serum salicylate level of 100 mg/dL, the patient was determined not to be a candidate for hemodialysis. Her serum salicylate level continued to decrease with intravenous fluids and bicarbonate therapy. A fetal ultrasound indicated a gestational age of 37–38 weeks (by fetal biparietal diameter and femur length). The fetal heart tracing showed a baseline fetal heart rate of 160–170 beats/minute with minimal variability and multiple spontaneous decelerations. The patient’s medical and mental status continued to improve. She also began to contract regularly, with cervical change to 3 cm dilation, consistent with early labor. The fetal heart tracing continued to be non-reassuring. Given the non-reassuring fetal heart tracing and the signs that the patient was entering early labor, she was extensively counseled as to the risks and benefits of a Cesarean delivery for both herself and the fetus and she consented to the procedure. To minimize risk of maternal hemorrhage, she was given a platelet transfusion prior to transfer to the operating room. A second unit of platelets was also started as the patient was taken to the operating room for an emergent Cesarean section under general anesthesia. A vertical skin incision, rather than a Pfannenstiel incision, was used to decrease the bleeding risk. Thick meconium was noted at the time of delivery, but the procedure was otherwise uncomplicated. Blood loss was estimated at 700 cc.

The female neonate weighed 3100 g at delivery and was initially limp and apneic with thick meconium-stained fluid. Apgars were 1 and 7 at 0 and 5 min, respectively. The initial heart rate was <60 beats/minute and chest compressions were not performed; the neonate was subsequently intubated. Arterial and venous cord gases drawn immediately after delivery were pH 7.32, CO2 43.6 mmHg, base excess −4 and pH 7.36, CO2 36.6 mmHg, and base excess −4.3, respectively. A salicylate level also drawn at this time was 33.5 mg/dL, while a level drawn simultaneously from the mother was 27.06 mg/dL. Initial complete blood count and coagulation studies at 1 h of life revealed a white blood cell count of 23 900 cells/mm3, hemoglobin 13.6 mmol/L, hematocrit 40.3%, platelets 285 000/mm3, PT 26.5, PTT
Acute acetylsalicylic acid overdose at 37 gestational weeks

There have been several prior reports of in utero salicylate toxicity at or near term [4], but fetal outcomes were not followed. In our case, a gravid patient presented with initial salicylate levels below those in prior case reports where significant morbidity or mortality was the outcome. Although the fetus showed signs of distress upon presentation, the decision was made to delay delivery until maternal status had been optimized. It was unclear whether the benefits of maternal hemodialysis would be conferred upon the fetus, as hemodialysis would have little effect on the salicylate that had already entered fetal circulation. In addition, fetal distress in this situation could be attributed to derangements in fetal acid–base status, as mentioned in previous case reports [4], with fetal acidosis persisting despite our patient’s alkalosis due to intravenous bicarbonate therapy. Indeed, it has been hypothesized that maternal hemodialysis does not benefit the fetus in the case of acute salicylate overdose and that emergent delivery is the most beneficial for the fetus at risk for in utero salicylate toxicity at or near term [4]. Maternal hemodialysis was not initiated in our case, with no adverse effect on fetal outcome.

As shown in Figure 1, neonatal salicylate levels, although higher than maternal levels, also declined spontaneously within 24 h of life.

As the patient’s condition stabilized without hemodialysis, she spontaneously began to labor and mode of delivery became a concern. The fetal heart tracing continued to show signs of fetal distress, and despite progressive cervical change, it was thought that the fetus would not tolerate a vaginal delivery. As salicylate metabolites accumulate in the fetal brain [5], potentially fatal intracerebral hemorrhage could result, as has been reported in other cases.
[2, 8]. Given these concerns for fetal well-being, a Cesarean section was determined to be the optimal mode of delivery.

Our experience with this case and further research on similar situations illustrates that maternal hemodialysis is of little benefit to the fetus in cases of salicylate ingestion and that expectant management is reasonable until maternal condition stabilizes, at which time, the patient should be counseled for a Cesarean section to avoid fatal fetal intracerebral hemorrhage.

Conflict of interest statement. None declared.

References


Received for publication: 8.6.11; Accepted in revised form: 26.7.11