for the admission. On HD 6–7 she was placed on ECLS for lung protection and to facilitate fluid removal. On HD 7 she was initiated on CRRT. On HD 12, a blood culture returned positive and subsequently grew Pseudomonas aeruginosa with a minimum inhibitory concentration for meropenem of 0.25 mcg/mL. As a result of the positive blood culture, she was initiated on a regimen of vancomycin, meropenem, and amikacin. Meropenem was started with a 40 mg/kg bolus given over 15 minutes after which a continuous infusion of 240 mg/kg/hour (240 mg/kg/day) was initiated. On HD 15 (ECLS day 9) a meropenem of 21 mcg/mL was obtained, corresponding to a clearance of 7.9 mL/kg/minute, drastically higher compared with 4 mL/kg/min reported in the package insert. Repeat cultures from HD 13–15 (ECLS day 7–9) were sterile. Concomitantly, a meropenem regimen of a 40 mg/kg bolus followed by a continuous infusion of 240 mg/kg/day was successful in providing a target attainment of 100% for serum concentrations above the MIC for at least 40% of the dosing interval and was associated with a sterilization of blood in this complex patient on concurrent ECLS and CRRT circuits.

1156
USE OF ANTI-D IN PATIENTS WITH SEVERE DENGUE
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Learning Objectives: Severe dengue fever (SDF) is a potentially fatal disorder with bleeding being a common cause of mortality. Bleeding is generally secondary to thrombocytopenia, which is a common finding in patients with SDF. Intravenous anti-D is an approved therapeutic option in improving platelet counts in the management of idiopathic thrombocytopenic purpura (ITP). Even though it has also been tried in patients with SDF for improving thrombocytopenia, there is insufficient data regarding its efficacy and safety in patients with dengue. In the present case series, eight patients, admitted in intensive care unit (ICU), meeting World Health Organization (WHO) criteria for SDF, were treated with anti-D immune globulin (WinRho SDF), in a single dose of 50 mcg/kg (250 IU/kg) intravenously. Their mean age was 27 (+/- 12.05) years with 4 years being male. Most of the patients (5/8; 62.5%), were admitted to ICU with evidence of multi-organ failure but only two patients were having symptoms of minor bleeding (epistaxis and melena). The mean SOFA score was 9.5 (+/- 3.4). Mean platelet count was 29,000/mm3 before and 66,700, 93,600, and 1,16,000/mm3 after intravenous anti-D administration at 24, 48, and 72 hours, respectively.

This patient's serum amlodipine level was 300 ng/mL prior to pheresis, with a therapeutic range of 8-10 ng/mL; concentrations above 20 ng/mL are toxic. Amlodipine levels were drawn before and after treatment. Therapeutic concentrations of amlodipine were 8-10 ng/mL; concentrations above 20 ng/mL are toxic. This patient's serum amlodipine level was 300 ng/mL prior to pheresis, with a post-treatment level of 180 ng/mL. A second plasma exchange brought drug levels from 110 ng/mL to 56 ng/mL. A significant improvement in blood pressure was noted, allowing weaning of vasopressive support. The patient did later develop a malignant dysthymia requiring emergent ECMO cannulation, but was successfully decannulated after 2 additional plasma exchanges and was later discharged to home. These findings demonstrate that plasmapheresis effectively reduces serum amlodipine concentrations and should be considered as a therapeutic option for amlodipine intoxication.

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PLASMAFERESIS SIGNIFICANTLY REDUCES SERUM AMLODIPINE LEVELS FOLLOWING INTENTIONAL OVERDOSE
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Learning Objectives: Calcium channel blockers (CCBs) are widely used and overdose is associated with high mortality. Few therapies are well studied, although the use of vasopressor support, calcium infusions, and hyperinsulin-euglycemic therapy are described. Presented is a pediatric patient with distributive shock following an intentional overdose of the long-acting CCB amlodipine. We demonstrate a rapid, significant reduction of amlodipine concentrations using plasmapheresis. To our knowledge, this is the first report measuring the reduction of amlodipine levels using this modality. This 16 year old male patient with a history of depression and hypertension presented with abdominal pain and nausea. During evaluation, he endorsed the consumption of 30 tablets of amlodipine blood pressure tablet within 24 hours. Patient was transferred to a tertiary pediatric ICU, where blood pressures declined to approximately 60/30 mmHg despite fluid resuscitation, calcium infusion, and IV glucagon and lipid administration. Vasopressor support including epinephrine, norepinephrine, dopamine, and vasopressin failed to improve blood pressures. Hyperinsulin-euglycemic therapy was initiated, but little benefit was noted. Because amlodipine is highly protein bound, plasmapheresis was trialed within 10 hours of admission and serum amlodipine levels were drawn before and after treatment. Therapeutic concentrations of amlodipine are 8-10 ng/mL; concentrations above 20 ng/mL are toxic. This patient's serum amlodipine level was 300 ng/mL prior to pheresis, with a post-treatment level of 180 ng/mL. A second plasma exchange brought drug levels from 110 ng/mL to 56 ng/mL. A significant improvement in blood pressure was noted, allowing weaning of vasopressive support. The patient did later develop a malignant dysthymia requiring emergent ECMO cannulation, but was successfully decannulated after 2 additional plasma exchanges and was later discharged to home. These findings demonstrate that plasmapheresis effectively reduces serum amlodipine concentrations and should be considered as a therapeutic option for amlodipine intoxication.

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A CASE OF JIMSONWEED TOXICITY
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Learning Objectives: Multiple patients are managed for drug overdose with an unclear etiology. An 18 year old male was found by police in a state of delirium and agitation, seeing ants and faces on walls, picking, and giggling. Significant objective findings were HR of 120 b/min, bilateral mydriasis, dry skin, repetitive inappropriate speech, a staggering gait, scribbles as clock drawing, CPK of 338 with increase to 537. 4 days after admission, urine toxicology positive for cannabinoids, and an EKG with sinus tachycardia. Combination of mydriasis and tachycardia can be suggestive of both an anticholinergic toxicity and/or a sympathomimetic toxiodynamic. But the latter includes diaphoresis and the former includes anhidrosis. Serotonin syndrome in addition to mydriasis, tachycardia, and diaphoresis also includes neuromuscular hyperactivity. Considering the above, etiologies of infection, endocrine disturbance, seizure, atypical alcohols, electrolyte abnormality, obvious poison, or organ dysfunction were ruled out. Cannabinoids did not explain agitation or mydriasis and since no diaphoresis or hyperthermia, diagnosis was an anticholinergic toxicodynamic. He responded initially to IV phystostigmine 2 mg and lorazepam, became cooperative and lucid. Clock drawing improved after phystostigmine. An hour later, he became agitated, hyperactive, and had visual and tactile hallucinations. After multiple doses of lorazepam and phystostigmine transferred to the ICU. After 2 hours, another episode of agitation and combativeness with auditory, visual, and tactile hallucinations was occurred. He was intubated and sedated with propofol. Time period occurred day 3 of hospitalization after phystostigmine 1–2 mg every 30 minutes. The patient was reported to have ingested an unknown amount of Jimsonweed (Datura stramonium) night before admission, to become altered. Jimsonweed, a plant in the Solanaceae family, used for asthma symptoms and as an anti-spasmylic, has anticholinergic properties due to the presence of hyoscyamine and scopodamine. Overdose may also lead to motor function loss, coma, and respiratory failure.

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CHYLOPTYSIS WITH MEDIASTINAL LYMPHANGIOMA IN 46-YEAR-OLD FEMALE
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Learning Objectives: Chyloptysis is rare but known to be associated with congenital or traumatic thoracic lymphatic tissue malformation such as lymphangiomatosis, lymphangioma, lymphangiectasis or lymphatic dysplasia syndrome. We have a unique case of a 46-year-old female who has chyloptysis with mediastinal lymphangioma. It is rare to have late onset disease without history of trauma to thoracic duct as most thoracic lymphangiomas are congenital and found before age of two. Case Description: A 46-year-old previously healthy female was admitted for recurrent left hemi-chylothorax with mediastinal mass. Patient initially presented to outside hospital with chest pain, palpitation and night time cough and was found to have thickened pericardium with small pericardial effusion and 4 by 2 centimeter sub-carinal soft tissue mass by Computed Tomography of chest and Transthoracic echocardiogram. All infectious and rheumatological work ups were negative at the time. However, over next few months she developed worsening dyspnea, orthopnea, and tachycardia can be suggestive of both an anticholinergic toxicity and/or a sympathomimetic toxiodynamic. But the latter includes diaphoresis and the former includes anhidrosis. Serotonin syndrome in addition to mydriasis, tachycardia, and diaphoresis also includes neuromuscular hyperactivity. Considering the above, etiologies of infection, endocrine disturbance, seizure, atypical alcohols, electrolyte abnormality, obvious poison, or organ dysfunction were ruled out. Cannabinoids did not explain agitation or mydriasis and since no diaphoresis or hyperthermia, diagnosis was an anticholinergic toxicodynamic. He responded initially to IV phystostigmine 2 mg and lorazepam, became cooperative and lucid. Clock drawing improved after phystostigmine. An hour later, he became agitated, hyperactive, and had visual and tactile hallucinations. After multiple doses of lorazepam and phystostigmine transferred to the ICU. After 2 hours, another episode of agitation and combativeness with auditory, visual, and tactile hallucinations was occurred. He was intubated and sedated with propofol. Time period occurred day 3 of hospitalization after phystostigmine 1–2 mg every 30 minutes. The patient was reported to have ingested an unknown amount of Jimsonweed (Datura stramonium) night before admission, to become altered. Jimsonweed, a plant in the Solanaceae family, used for asthma symptoms and as an anti-spasmylic, has anticholinergic properties due to the presence of hyoscyamine and scopodamine. Overdose may also lead to motor function loss, coma, and respiratory failure.