Antenatal corticosteroids to reduce preterm deaths in low-income settings

The Comment1 by Kishwar Azad and Anthony Costello opposing scale-up of antenatal corticosteroids misdirects the discussion of this topic towards speculation about differences in low-income settings. Our experience in Malawi provides a concrete example of the rapid scaling up of antenatal corticosteroid treatment with dexamethasone.

Malawi has the highest estimated preterm birth rate worldwide.2 In Bwaila Maternity Hospital, Lilongwe, that has more than 15 000 deliveries annually with more than 2900 preterm, we increased targeted coverage of antenatal corticosteroids from 8% to 80% in 16 weeks in women at risk of preterm delivery from 24 to 34 weeks’ gestation. After this pilot study, we began programmes in three other hospitals, reaching 59–83% coverage from a baseline of 1–6% within 6 weeks. This intervention has thus far been associated with a drop in preterm neonatal mortality contribution from 60% to 24% at 0–6 days of age. Although this intervention was not done as part of a trial, and focuses only on quality improvement, we noted no increase in the rate of maternal or neonatal infections.

Antenatal corticosteroids induce fetal lung maturation through the same biological mechanism in low-income settings as in high-income settings and reduce the need for neonatal mechanical ventilation.3 Although antenatal corticosteroids might not be a so-called magic bullet as a standalone vertical intervention, no biological basis exists to presume that babies born preterm in resource-poor settings will succumb to respiratory distress syndrome any more than do those in resource-rich countries. I support the existing recommendation of a single-course of antenatal corticosteroids to mothers at high risk of preterm birth between 24 weeks and 33 weeks’ gestation, but question Azad and Costello’s unrealistic prerequisite for round-the-clock access to level 2 care in a low-income setting.

Low-income settings, which have the highest burden of preterm neonatal deaths, urgently need proven beneficial interventions, not the assessment of therapeutic efficacy on the basis of resource profiling that could delay treatment. Contrary to Azad and Costello’s speculation,1 antenatal corticosteroids are likely to have a greater effect in the absence of level 2 care, not a lesser effect.4 The difference between low-income and high-income settings is not biology, but an increased burden of disease and reduced access to even basic health care. Our experience in Malawi offers a powerful example for generalising this standard of care to the regions where it will save the most lives and also reduce neonatal disability. I declare no competing interests.

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