

# Anchoring Bias as a Limiting Factor in High-Value Care: A Case of Fever of Unknown Origin in a Hospitalized Child

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A previously healthy 4-year-old girl with a 3-week history of subjective weight loss, diffuse abdominal pain, and fever presented to the emergency department for evaluation. Her examination was remarkable for right-sided abdominal tenderness. Initial laboratory results were significant for microcytic anemia with hemoglobin of 8.8 g/dL, thrombocytosis to 800 K/ $\mu$ L, leukocytosis to 19.8 K/ $\mu$ L (91% neutrophils), and elevated erythrocyte sedimentation rate to 128 mm/h. An ultrasound demonstrated a normal appendix with a small amount of nonspecific free fluid in the right lower quadrant, the latter of which prompted an abdominal MRI. Although there was no evidence of appendicitis, the MRI was incidentally notable for numerous punctate T2 hyperintensities of the liver, of unclear significance. The patient was admitted for further evaluation, at which time the gastroenterology and infectious disease services were consulted. Gastroenterology recommended a fecal calprotectin and perinuclear anti-neutrophil cytoplasmic antibodies to evaluate for the possibility of inflammatory bowel disease. On the basis of a recent kitten exposure, the infectious disease service recommended *Bartonella* serologies and polymerase chain reaction, echocardiogram, ophthalmologic examination, and empirical azithromycin for suspected diagnosis of visceral cat scratch disease (CSD).

All infectious studies were negative, casting doubt on the initial diagnosis. Nonetheless, in the absence of findings to support an alternative explanatory model, the primary team adhered to the leading hypothesis. Such anchoring was furthered by a selection of evidence that was most consistent with preexisting hypotheses, namely that antibiotics have not been found to significantly affect cure rate or time-to-cure in CSD, and that serologies may remain negative in infected patients.<sup>1</sup> After a lack of clinical response to azithromycin, rifampin was added.<sup>2</sup> A liver biopsy was obtained for clinical correlation of the hepatic lesions seen on MRI; however, these lesions were not visualized on ultrasound, and the tissue was without abnormalities. On hospital day 13, the initial quantitative fecal calprotectin resulted as elevated (393  $\mu$ g/g), suggestive of intestinal mucosal inflammation.<sup>3</sup> Esophagogastroduodenoscopy and colonoscopy were performed, and pathology was diagnostic of Crohn disease.

The decision to pursue an MRI as a result of nonspecific ultrasound findings became a pivotal juncture in the quality of care delivered. Risk-stratification algorithms have been developed to help support or exclude the clinical diagnosis of pediatric appendicitis. Although the best-studied clinical scoring systems, the Alvarado and pediatric appendicitis score, provide a useful clinical adjunct,<sup>4,5</sup> neither has adequate predictive value to be used as the sole diagnostic standard.<sup>6</sup> On the basis of these scores, the decision to proceed with ultrasound was

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justifiable.<sup>7</sup> However, absent additional ultrasonographic findings to support a diagnosis of appendicitis, there was no indication to proceed with MRI on the basis of the finding of free fluid alone.<sup>8</sup>

On MRI, incidental discovery of punctate T2 hyperintensities of the liver prompted a consultation of the infectious disease and gastroenterology services. For a liver incidentaloma of <0.5 cm in patients with low risk of malignancy, the American College of Radiology recommends no further follow-up.<sup>9</sup> As a result of premature closure on the diagnosis of visceral CSD, the consulting and primary services each developed explanatory models that attributed the MRI findings to microabscess formation. Because tissue diagnosis is considered the diagnostic gold standard for visceral CSD, an invasive diagnostic procedure was required to dispel a then-ingrained anchoring bias.<sup>10</sup> The ensuing decision to pursue liver biopsy serves as the hallmark of unnecessary care, and the culmination of a series of cumulative cognitive errors.

This case underscores the potential of incidental findings to mitigate high-value care by reinforcing anchoring bias in the setting of premature diagnostic closure. Given its potential to result in delayed or missed diagnoses, cognitive error has been identified as an important source of unnecessary costs to patients and the health care system.<sup>11</sup> Such errors motivate the provision of medical care that is both unnecessary and potentially harmful.<sup>12</sup> These costs are evidenced by studies demonstrating delayed or missed diagnosis in upward of 10% of hospital admissions.<sup>13</sup> In many such cases, cognitive error is identified as a principal a source of preventable death or permanent disability.<sup>12,14,15</sup>

The extent to which cognitive errors are directly attributable to the physician remains poorly characterized.<sup>16</sup> Nonetheless, attention to misleading or irrelevant aspects of the history and physical may prime physicians to prematurely adhere to an inaccurate explanatory model of illness.<sup>17,18</sup> The cognitive error in the described case

may have been mitigated by emerging strategies of behavioral modification, namely metacognition.<sup>19</sup> Metacognition is defined as the practice of analyzing and understanding one's own reasoning.<sup>14</sup> This practice requires that the physician evaluate his or her own approach to the diagnosis to neutralize cognitive bias.<sup>14</sup> Notably, metacognition requires a foundational understanding of the divisions and drivers of cognitive error, concepts that have been established as readily educable at the early stages of medical training.<sup>19,20</sup>

In summary, this case highlights the potential of nonindicated diagnostic studies to yield incidental findings, and thereby generate substrate for cognitive error. The initial decision to evaluate for appendicitis by way of abdominal ultrasound was indicated and supported by the evidence. Conversely, the ensuing decision to proceed with an MRI resulted in incidental findings that supported cognitive error in the absence of alternative information. Indeed, adherence to an erroneous explanatory model, motivated by confounding elements of the patient history and imaging studies, hastened premature closure on the misdiagnosis of CSD. This early misdiagnosis facilitated pronounced anchoring bias and precipitated a prolonged and costly hospitalization, characterized by the provision of unneeded care.

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