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Pediatric Condition Falsification Misdiagnosed by Misjudged Weight Growth from the Curve of Measured Weights

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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Patient: Male, 0–2
Final Diagnosis: Cow milk allergy
Symptoms: Obstipation • airway infections
Medication: —
Clinical Procedure: Elementary feeding
Specialty: Pediatrics

Objective: Rare disease

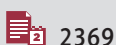
Background: Pediatric condition falsification (PCF) is a rare form of child abuse in which a caregiver fabricates or induces illness in the child. The diagnosis is difficult and controversial and can easily include false positives.

Case Report: A boy, 3.18 kg birthweight (P25 curve), lost weight between age 56 to 120 days. Cow milk allergy was suspected, feeding was changed to elementary formula, and he started catch-up weight growth while remaining significantly underweight. His pediatrician continuously interpreted his low weight as insufficient growth, despite prescribing 3 times the normal caloric intake, concluded that the mother purposely malnourished her son, diagnosed PCF, and the boy was separated from his family (days 502–755 of age). PCF was confirmed by 2 other pediatricians and 3 child protection physicians and was supported by 4 child protection agencies and 6 judges. However, proper analysis of the weight growth (kg/year) from the weight curve showed a normal weight gain. Beyond 120 days of age, weight gain at home was significantly above normal (during 347–489 days: 6.2 versus 3 kg/year of the P50). He reached P25 again at around 516 days.

Conclusions: The question “How could so many physicians misjudge weight gain?” has scientific and sociologic aspects. Scientifically, low weight was wrongly interpreted as insufficient weight growth, requiring that physicians learn how to assess weight gain from weight curves. Sociologically, physicians seem to follow a diagnosis made by a colleague without proper evaluation. Arguments provided by the parents against this diagnosis seemed to be neglected. Confirmation bias occurs when any information against PCF is disregarded.

MeSH Keywords: Body Weight • Case Reports • Diagnostic Errors • Weight Gain

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Background

Pediatric condition falsification (PCF) is a form of child abuse in which a caregiver, frequently the mother, fabricates or induces illness in the child. Other terminology used in the literature is: Munchausen Syndrome by Proxy, Fabricated or Induced Illness by Caregivers (FI), Factitious Disorder Imposed upon Another, Factitious Disorder by Proxy and, more generally, Medical Child Abuse. PCF is rare and epidemiological studies suggest that it affects at least 0.5–2.0 per 100 000 children aged under 16 years, and McClure et al. reported that the rate is at least 2.8 per 100 000 children under 1 year of age [1–3]. An editorial in *The Lancet* stated that “*The best epidemiological studies to date show that health professionals are likely to encounter at least one case of FI during their careers, with pediatricians seeing many more*” [4]. The pediatric author of the present paper encountered 3 cases during a period of 40 years. Perhaps contradictory to these statistics (which have been criticized based on double-counting certain case studies in separate articles [1,5]) is that the diagnosis of PCF, per exclusionem, has been stated as being difficult, controversial, and with a considerable likelihood that false positives occurring [1,5–8]. An example, Eichner’s seminal publication [1] (page 304) states that mitochondrial disease might be mistaken for PCF and is about 11 times more prevalent; Therefore, when a physician cannot find a diagnosis explaining the symptoms of a child, the diagnosis of PCF might be made. When a physician misses a correct diagnosis, PCF might wrongly be supposed. Symptoms may exist even when no diagnosis can be made.

The diagnosis of PCF is generally assumed to require proof of all of Rosenberg’s 5 criteria [9]:

1. All other diseases that could explain the symptoms are excluded.
2. Separation of child from the caregiver resolves the symptoms.
3. Standard treatments are ineffective.
4. There is objective evidence that the caregiver lies about the symptoms.
5. The caregiver seeks inappropriately for second opinions.

Unexplained failure to achieve a normal increase in weight, failure-to-thrive (FTT), is one of the conditions for which the diagnosis PCF is considered [1]. It requires both the exclusion of a vast list of known causes of FTT [1] and, importantly, an accurate evaluation of the weight curve, also referenced by Pankratz [5] on page 314. We present a case in which the pediatrician diagnosed PCF “*with 100% certainty*”, which in itself is very unlikely [10], a diagnosis that was incorrect due to a misjudged weight gain velocity from the curve of measured weights. To the best of our knowledge, this is the first well-documented report of this association. In this case report we aimed to identify why this false-positive diagnosis of PCF occurred.

We believe it is important for what follows to give the definition of (weight) growth. From elementary physics, any form of growth is *always* expressed proportional to reciprocal time (e.g., 1/year). Growth of weight, expressed as weight gain velocity, is defined as

$$\text{weight gain velocity [kg/year]} = \frac{\text{weight gained a certain time period [kg]}}{\text{duration of that time period [year]}}$$

Case Report

The youngest son of normal parents (sixth child, born at term, 3.18 kg birthweight, P25 or –0.6SD standard weight curve) grew along the –2SD weight curve until about 56 days of age, after which he developed a slightly negative weight growth (days 56–120), becoming seriously underweight (see Figure 1) and requiring hospitalization (99–114 days). Hirschsprung’s disease was excluded following a colon biopsy. Cow milk allergy was suspected because of frequent episodes of obstipation and undue crying and anxiety after food intake. Without further testing, feeding was subsequently changed to elementary formula. An increased calprotectin level in feces (values between 250 and 1200 versus 50 µg/g feces normal) was found, likely due to cow milk allergy. Tests identified an allele-22 deletion on chromosome 9, but the same mutation was found in the healthy father, so it was considered clinically insignificant. The cow milk allergy might explain other signs and symptoms of the infant: sleeping disorder, frequent periods of obstipation, abdominal cramps, airway infections, and colds. Despite extensive investigations, including immunology, endocrinology, and metabolic disorders, no other explanation for the low weight gain during days 56–120 was found. From day 140, elementary formula feeding was given through a nasogastric tube. Two more hospitalizations occurred (days 155–161 and 315–331). Remarkably, following a period of weight loss (from 8 to 7.35 kg) caused by gastroenteritis (days 337–346), the pediatrician reported: “*– he lost weight for 4 more weeks*”; but in reality, weight increase clearly recommenced immediately after the period of sickness (Figure 1). Nevertheless, the infant remained underweight (Figure 1). The pediatrician continuously interpreted low weight as inadequate growth and increased tube feeding to 3 times normal (2.8 liters/24 h daily). The pediatrician wrote the following conflicting remarks: “*it is worrying that he does not grow given the enormous food intake*”, and: “*there is no medical explanation of how the boy can handle so many calories*”. To “*prove*” that the boy did not receive these calories at home, he was hospitalized (days 489–502). Identical caloric intake caused severe vomiting, as the mother previously experienced at home and had reported to the pediatrician. The pediatrician denied the mother’s statements of having given the prescribed calories and that vomiting also occurred at home, and diagnosed the mother with PCF, but without documented consultations with experienced colleagues, violating

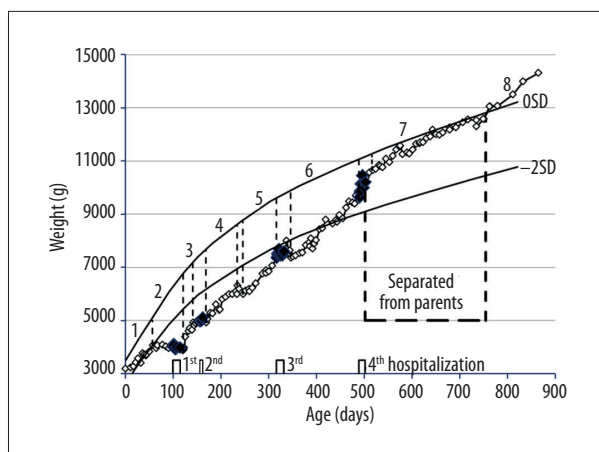


Figure 1. Weight versus age (until 865 days, white lozenges) with 4 hospitalizations (large black lozenges, the periods indicated just above the horizontal axis) and standard weight curves OSD (P50) and $-2SD$ for Dutch boys. Several short periods of weight loss were due to sickness (around 240, 340, 420 and 580 days). Table 1 summarizes the average weight gain velocities (in kg/year) during the 6 periods at home, during separation, and after returning home again, and the corresponding average weight gain velocities of the OSD curve.

the guidelines of the Royal Dutch Medical Association. The infant was subsequently separated from his family.

In the Dutch privacy of the juvenile court system, assigning PCF to the mother was defended by the first pediatrician and 3 child protection agencies, stating that she had malnourished her boy on purpose and that the boy's safety required that he had to remain separated from his parents. The judge, however, disagreed with them and ordered that the boy was to be returned home. The pediatrician was "flabbergasted", appealed this conviction, and found a colleague (second) pediatrician who supported the PCF diagnosis. During the second court session, now with 3 judges (also including the first judge), some written quotes are: "it's difficult to explain how he only grew during hospitalizations" (second pediatrician) and "separation from his parents reversed his growth towards normal" (National Child Protection Counsel). Both pediatricians and the Counsel declared that all Rosenberg criteria applied in this case. The 3 judges of the second law court confirmed PCF and prolonged the separation of the infant from his parents. The parents appealed this conviction but the third court of appeal reconfirmed PCF. The parents then decided not to appeal further to the Dutch Supreme Court. Eventually, the infant returned home after 8 months, albeit under legal supervision. Prior to this last court session, even a third pediatrician (from the same hospital as the second pediatrician) confirmed PCF, commenting on the boy's weight growth during days 346–489: "The weight gain velocity remains continuously negative

compared to OSD; it should have been strongly positive (catch-up growth following malnutrition)", supported by a fourth child protection agency. Earlier psychiatric and police investigations cleared the parents unconditionally from being instrumental in the boy's FTT. Except for the first judge during the first court session, all judges subsequently discarded our analysis of the boy's weight curve in which we showed beyond doubt that weight gain velocities were even above normal at home and that none of Rosenberg's criteria could have applied.

Analysis (Figure 1, Table 1) shows unmistakably that weight gain velocities at home always exceeded those of the OSD curve from 120 days onward by factors varying between 1.3 and 2.3. During separation, the infant grew 2 times slower (not faster, as was stated by the National Child Protection Counsel) than previously at home (3.1 versus 6.2 kg/year, Figure 1, Table 1), although still stronger than the OSD (2.4 kg/year). Interestingly, the first 16 weeks of being back home again showed an increased weight growth compared to the separation period, from 3.1 to 5 kg/year. Subsequently, the boy developed completely normally, albeit with susceptibility to nasal colds.

Discussion

Why did 3 pediatricians and 3 child protection physicians supported by 4 child protection organizations wrongly judge that this infant had FTT beyond day 120 and that it was caused by PCF of the mother?

The answer to this question has scientific as well as sociologic components.

Scientifically, during days 56–400, the infant's weight was below $-2SD$. However, discussions about the criteria for defining FTT [11, 12] show the following. If the child is doing well, this is contradictory to FTT. Also, the current most important FTT criterion is lack of adequate growth. Olsen concluded in her review [13] that "Weight gain is the predominant choice of indicator". And "For the time being, FTT predominantly seems to be used to describe children with slow or falling weight gain". Thus, FTT is defined as insufficient weight gain velocity, not as low weight [11–13]. Not all infants grow above $-2SD$. Thus, a normal weight gain (see [14]), also for weights below $-2SD$, is not a sign of abnormality [14]. Consequently (Figure 1, Table 1), the period of FTT in this case lasted only 9 weeks, from 56 to 120 days. This was explained by a cow milk allergy and because he gained weight at a normal rate when on elementary feeding. He reached his birth weight curve (P25, $-0.6SD$) around day 516, 2 weeks after the separation period started, obviously showing that he achieved a significantly above-normal average weight gain velocity at home (after day 120, between 17.1 and 6.2 kg/year versus 7.8 and 3 of OSD, during a

Table 1. Summary of the boy's average clinical weight velocities in 8 periods, calculated by linear trend lines in Excel, those of the OSD curve of Figure 1 calculated from last minus first weight divided by the period, and their ratios. Period 7 denotes separation.

Period	Days	Average weight velocity (kg/year)		
		Boy	OSD	Boy/OSD
1	0–56	5.7	10.4	0.55
2	56–120	–1.14	8.4	–0.14
3	120–141	17.1	7.8	2.2
4	168–234	6.5	5.1	1.3
5	246–316	7.1	4.4	1.6
6	346–489	6.2	3.0	2.1
7	516–755	3.1	2.4	1.3
8	755–865	5.0	2.2	2.3

period of more than 1 year, Figure 1, Table 1). This is in contrast with the third pediatrician's estimate by a factor of at least 2.1. Stronger catch-up weight velocity was seen after the introduction of solid foods. The judges ordered weighing the infant weekly. It is, however, impossible to evaluate weight gain velocity from weekly intervals since a full or empty bladder or colon can cause the same weight difference as a weekly weight gain. Most likely, the pediatricians were impressed by the weight below –2SD and confused by the weekly changes in weight. Low weight was wrongly interpreted as low weight gain velocity. The many physicians making this mistake suggests that we may have identified a hitherto unknown false-positive PCF diagnostic mechanism.

The sociological components basically cover the expected hurdles that all pediatricians, child protection physicians, and agencies kicked over to come to a (false) conclusion. First, the conclusion of the first pediatrician could have been supported by the other physicians and child protection agencies just because of disinterest in challenging a colleague or of being reluctant to consider alternative hypotheses, a situation that may frequently occur [5]. Second, a "confirmatory bias" may have developed, in which any information is interpreted negatively, even if contradictory to a PCF diagnosis. An example is the bizarre 3-times normal food intake (first pediatrician) before and during the fourth hospitalization. This implies an intake of 2.64 liters/day plus drinking of another 0.9 liters/day, giving a total food intake of about 0.4 liters/kg/day (for comparison, an adult would then have to drink 30 liters/day). Such a feeding includes a dangerous amount of proteins of about 10 gr/kg/day (1.5 is normal), which could increase blood urea concentrations to intoxication levels and also a dangerous dose of vitamin A according to Dutch Nutrition Center guidelines. Another example is the mother's nightly stay with her son (third hospitalization) where intentional observations acknowledged her as loving and

worrying, nevertheless making the first pediatrician state in court that: "but this does not prove that she is innocent". Third, separation of the boy from his family, called "separation test", is criticized by Pankratz [5] and Wrennall [15], stating that "In case after case, the separation test is manipulated ----" [5], and "---- the separation test is likely to produce massive numbers of false-positive diagnoses of child abuse" [15], as indeed occurred in this case by falsely reporting that weight growth velocity prior to separation was smaller than during separation (Table 1). Fourth, the importance of listening to the parents was violated by all physicians and agencies, particularly by the first pediatrician, whose denial that the mother had reported the boy's heavy vomiting at home, and thus "she had lied", became the ultimate proof of PCF. This additional example of confirmation bias calls for awareness and the need for training of pediatricians and child protection physicians on the importance of listening to parents. Fifth, the argument that a parent's denial of guilt is further evidence of PCF actually was used by all 4 child protection agencies.

Our analysis produces totally different answers to Rosenberg's criteria than in the written statement of the Dutch National Child Protection Counsel. We showed that cow milk allergy caused the 2 months of weight loss and thus FTT; that weight gain velocities at home beyond day 120 were even much stronger than OSD (Table 1); and that the mother never lied about her boy's symptoms and never inappropriately sought second opinions. This proves beyond any doubt that PCF of a caregiver has no relationship with this case.

Conclusions

Our first, science-based, conclusion is that this case report confirms that PCF can easily be misdiagnosed, which emphasizes

that pediatricians and child protection physicians must be more careful than demonstrated here to consider temporary (in this case 9 weeks) FTT as a sign of PCF. Also, this is the first well-documented case demonstrating that 6 physicians were likely unable to correctly assess weight growth from a weight curve, which resulted in a false-positive PCF diagnosis. Correct analysis, requiring very simple and elementary differential calculus, such as determining the (average) weight gain over a certain age period and dividing it by that period, equation (1), can prevent this perplexing and likely novel cause of misdiagnosis from occurring again.

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Conflict of interests

None.