Munchausen Syndrome by Proxy
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DOI: 10.1542/pir.25-6-215

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Munchausen Syndrome by Proxy

The term "Munchausen Syndrome by Proxy" (MSBP) was coined to describe a form of child abuse in which a caretaker, usually the mother, feigns or induces symptoms in a child that result in unnecessary medical testing and procedures. Classically, the motivation for the behavior is to assume a "sick role" through the child, thereby engaging in an ongoing relationship with medical personnel. The syndrome was identified by Meadow, who reported two cases in 1977. One mother had poisoned her child repeatedly with salt from the age of 6 weeks to the child’s death at age 15 months; another woman had tampered with her child’s urine samples, provoking invasive investigations and both medical and surgical treatments for the child at several medical centers.

Since those first reports, MSBP has broadened in scope, also being applied to cases of “doctor-seeking” and of “imposed upper airway obstruction” (deliberate smothering/choking of an infant) when the parent’s purpose has been to assume the sick role by proxy. By contrast, parents harming their children from frustration or anger typify a more classic form of child abuse.

The incidence of MSBP is about 0.4/100,000 in children younger than 16 years of age and 2/100,000 in children younger than the age of 1 year. In one large study, the mean age at diagnosis was 20 months. Siblings of child victims of MSBP also frequently are abused; one series found a rate of 40% of abuse in siblings, and 18% had a history of sibling death.

The most common presenting signs for MSBP are bleeding (44%), seizures (42%), central nervous system depression (19%), apnea (15%), diarrhea (11%), vomiting (10%), fever (10%), and rash (9%). The most common methods of fabricating illness are lying, poisoning, suffocating, specimen tampering, and chart falsifying.

Morbidity and mortality from MSBP are substantial. In one series, 8% of children suffered long-term consequences, including the need for multiple esophageal surgeries, destructive joint changes, and mental retardation with cerebral palsy and cortical blindness. Overall mortality appears to be between 6% and 8%, but the rate is higher for MSBP involving poisoning or airway obstruction. Children who survive MSBP are at risk for a variety of psychiatric morbidities, including severe withdrawal, paranoia, preoccupation with bodily integrity and vulnerability, and chronic invalidism.

Perpetrators of MSBP are usually mothers; men are responsible for fewer than 5% of cases. Typically, the perpetrator has a distant or uninvolved partner but establishes an unusually close relationship with hospital staff. About one third have at least some training in a health profession. They usually are very pleasant, cooperative, and appreciative of medical staff. As described by Meadow, "...these two (mothers) flourished there as if they belonged, and thrived on the attention that staff gave to them."

The term MSBP has been used both as a descriptor of the abuse itself (pediatrics) and as a diagnostic label for the perpetrator (psychiatry). The latest DSM-IV manual has replaced the term MSBP with “Factitious Illness by Proxy.” The diagnosis applies to a perpetrator who intentionally produces or feigns symptoms in another person under her care and is motivated by a need to assume the sick role by proxy.

Although early reports did not suggest an overlap, recent research recognizes that Munchausen syndrome and MSBP frequently do intersect. As many as 72% of women who perpetrate MSBP display abnormal illness behaviors themselves, including somatoform disorder and factitious disorder. Conversely, adults diagnosed with factitious disorder are more likely to abuse their children through MSBP than are controls.

Despite the debates in terminology, it is most important for pediatricians to recognize that MSBP constitutes child abuse. Pediatricians dealing with these cases often become so involved in assessing the unusual and intriguing presentation that the proper management of child abuse is delayed. The most important means of identifying cases of factitious illness by proxy is to include


Rifampin, discovered in 1957, is a semi-synthetic derivative of rifamycin B, a fermentation product of Streptomyces mediterranei. Its mechanism of action involves inhibition of DNA-dependent RNA polymerase, thereby interfering with protein synthesis. Rifampin is effective against both coagulase-positive and -negative staphylococci as well as other gram-positive cocci, such as Streptococcus pyogenes, penicillin-sensitive S pneumoniae, enterococci, and Peptostreptococcus sp. Among gram-negative bacteria, Neisseria meningitides, N gonorrhoeae, and Haemophilus influenzae are the most sensitive. Rifampin also is active against Chlamydia trachomatis and C psittaci.

Most strains of Mycobacterium tuberculosis are as susceptible to rifampin as to isoniazid, but resistant strains have been isolated with greater frequency in recent years. Resistance is more likely among previously treated (about 9%) than new patients (about 4%). Rifampin kills both rapidly dividing bacilli and those that exhibit only occasional short bursts of metabolism. The susceptibility of other mycobacteria is variable: M leprae is killed faster by rifampin than by sulfones; M fortuitum and M chelonei are resistant.

Rifampin is well absorbed from the gastrointestinal system in the fasting state. An intravenous preparation is available when the oral route cannot be used. Dosing is the same for the oral and intravenous preparations. Rifampin is well distributed in body fluids and tissues, with therapeutic concentrations achieved in serum, urine, saliva, bone, pleura, pancreatic fluid, and cerebrospinal fluid. It is cleared by hepatic metabolism and biliary excretion, with a half-life of 2 to 5 hours. Hepatic disease can prolong clearance.

Resistance to rifampin develops rapidly when it is used alone; it never should be used as monotherapy for any serious infection. Rifampin plays an important role in the treatment of patients who have tuberculosis, both as part of a multidrug regimen for active disease and as prophylaxis for latent infection. As prophylaxis, rifampin is appropriate only when resistance to isoniazid is suspected or when isoniazid is contraindicated.

Another common use for rifampin in pediatrics is to eradicate the carrier state in close contacts of patients who have meningitis from N meningitides or H influenzae. Rifampin is the drug of choice for chemoprophylaxis against both organisms.

The use of rifampin in treating staphylococcal endocarditis and chronic osteomyelitis remains controversial. Adding rifampin to vancomycin and gentamicin may result in more rapid elimination of the organism from cardiac vegetations. One strategy is to reserve rifampin for cases that include metastatic abscesses or for patients who have persistently positive blood cultures after initial therapy. Rifampin also has been used for staphylococcal ventriculoperitoneal shunt infections because of its synergistic activity with nafcillin or vancomycin. Combined with other antistaphylococcal antibiotics, rifampin can eradicate nasal carriage of methicillin-resistant organisms, and it

Comment: Language is important.

Dr Sharif is doing her duty in reporting what the literature contains, but why in the world would we want to call smothering a child “imposed upper airway obstruction?” Please!!!

Henry M. Adam, MD
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has been used in combination with penicillin to eradicate chronic pharyngeal carriage of group A beta-hemolytic streptococci.

Rifampin usually is well tolerated, and children experience fewer adverse effects than do adults. Red-orange in the crystalline state, rifampin can turn urine, tears, and saliva reddish-orange (the most common adverse effect), and it may stain contact lenses. Immuno-logic reactions such as drug fever, rash, and eosinophilia can occur, as can an influenza-like syndrome, particularly in patients who take rifampin intermittently, usually less than twice weekly. Intermittent use of rifampin also has been associated with a more fulminant hemolytic reaction that can produce anemia, tubular necrosis, and shock. Both thrombocytopenia and leukopenia have been associated with the regular administration of rifampin.

The risk for hepatotoxicity with rifampin is associated with overdose or previous liver disease. Patients treated with both rifampin and isoniazid are more than twice as likely to develop hepatitis as are patients treated with rifampin and other antituberculosis drugs. However, rifampin induces hepatic microsomal cytochrome P450, thus decreasing the half-life of a wide range of other drugs, including oral contraceptives and methadone.

Rifampin crosses the placenta and is teratogenic in animals. It should not be used for chemoprophylaxis in pregnant contacts of patients who have meningitis. Despite the risk, rifampin remains indicated for the treatment of severe tuberculosis during pregnancy.

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Comment: Rifampin’s role in the management of tuberculosis (TB) is not always clear because the difference between latent infection and active disease can be confusing. For clinical purposes, a child who has a positive skin test result has been exposed to and infected with TB. If the child is asymptomatic and has a normal chest radiograph, the infection is considered latent, and the child needs prophylaxis to prevent progression to active disease. Isoniazid (INH) is the agent of choice for latent infection, except for TB that is resistant to INH or for patients who cannot tolerate INH; then, rifampin is the drug of second choice. However, if the child who has a positive skin test has symptoms of TB or evidence of TB on the chest radiograph (other than granulomas or calcification), active disease already has developed. The response to active disease, as opposed to latent infection, is treatment with a minimum of two drugs rather than prophylaxis with a single agent. Almost always, multidrug treatment regimens for tuberculous disease include rifampin.

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Editor, In Brief
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Fractures


Each year, 15 million children in the United States visit emergency departments for unintentional injuries, 50% of which involve musculoskeletal complaints. Fractures account for about 10% to 15% of all serious childhood injuries; not all are obvious, and the pediatrician must be able to diagnose those that are occult. Although complications from growth plate injuries are uncommon, failure to recognize and manage them properly may lead to growth arrest and deformity. Accordingly, the pediatrician must understand the unique features of the immature skeleton and how they affect the types of fractures that can occur.

Several distinctive features of the immature skeleton result in specific patterns of injury. Children’s bones have relatively more haversian canals than do adult bones, making them more porous and, thus, more likely to buckle with compression and to bow when bent. In younger children, torus ("buckle") and greenstick fractures are more common than the complete fracture through the cortex typical of denser mature bone. Further, the periosteum is thicker and stronger in children than in adults, thereby reducing the incidence of displaced fractures and often creating a more stable injury that does not require reduction. Pediatric bone also remodels more quickly than mature bone, promoting rapid healing, which may preclude the need for anatomic alignment. Remodeling alone, however, will not repair displaced intra-articular fractures.

Clinically, the most important feature of children’s bones is the presence of the growth plate (physeis), the area of rapidly proliferating cells located between the metaphysis and epiphysis of all long bones. Although growth plate injuries may occur at any age, accounting for 15% to 30% of all pediatric fractures, they are most common during periods of rapid growth, peaking between the ages of 10 and 16 years, with boys more likely than girls to sustain the injury.

The growth plate consists of four distinct areas or zones of cell growth: resting cells, proliferating cells, maturing/hypertrophic cells, and provisional calcification. The area most susceptible to injury is the hypertrophic zone, sparing the proliferating zone and, thus, leaving the growth potential of the plate intact. Because the epiphysis provides the blood supply for the growth plate, any injury to the growth plate that also involves the epiphysis is likely to have impaired healing and require more intensive treatment and follow-up.

Among the several classifications of growth plate fractures, the Salter-Harris system is used most widely and categorizes five fracture types:

Type I: A fracture along the growth plate, rather than across it, that separates the epiphysis and the metaphysis. Without any cortical break, radiographs often appear normal, and the diagnosis is made on clinical grounds.

Type II: A fracture along the growth plate, with an oblique extension through a piece of the metaphysis. This is the most common growth plate fracture and, as is the case with type I fractures, generally does not require operative reduction for a good prognosis.

Type III: A fracture through the growth plate that extends into the epiphysis and joint space.

Type IV: A fracture through the growth plate that extends into both the metaphysis and the epiphysis and into the joint space. Both types III and IV fractures threaten growth potential and articular integrity, usually requiring open anatomic reduction and fixation.

Type V: A compression of the growth plate, usually recognized only after the fact, when failure of growth is noted. This crush injury is the rarest type of fracture.

Most injuries occur while children are at play, and each age group has a typical pattern of trauma leading to common types of fracture. Young children most commonly fall forward onto outstretched arms. Upon maturity, more injuries are associated with motor vehicle accidents, bicycle falls, and sports-related trauma. Children’s ligaments are stronger and withstand more mechanical force than those of adults, but by not giving way, they pass on traumatic forces to the bone. An injury to an adult that might result in a sprain or joint dislocation often results in a fracture to a child. Therefore, it is important to be wary of the diagnosis...
of sprain or strain in the younger patient and consider instead an occult growth plate injury.

Although an older child can describe an injury and localize the area of pain, a young child or infant may present only with irritability, pseudoparalysis of the injured area, or refusal to walk or bear weight. Physical examination must include observation for deformity, spontaneous movement of the limb, and position of the child at rest as well as assessment of the skin and of the neurologic and vascular competence of the injured area. Careful palpation of the entire limb is indicated to locate the point of maximum tenderness. Once the injury has been localized, appropriate radiographs should be obtained. Films should include the joints above and below the suspected fracture site in both anteroposterior and lateral views. Oblique views may be needed to assess growth plate injuries. Salter I and V fractures often are not visible on radiographs, but comparative views and correlation with physical findings can help make the appropriate diagnosis. The presence of swelling and point tenderness directly over a growth plate suggests a Salter I fracture, even in the absence of initial radiographic findings. Follow-up films may show periostial changes that confirm the diagnosis.

Although breaks of the upper extremity happen more frequently, one of the most common fractures in children younger than 4 years of age is the “toddler’s fracture,” a nondisplaced spiral fracture of the tibia induced by a rotational injury. Often the injury is not witnessed and occurs during any of the usual mishaps of toddlerhood that can result in a twisting force on the leg. The child may present with limp, widened gait, or complete refusal to bear weight. Tenderness to palpation, with minimal if any swelling, may be the only physical finding. The spiral toddler’s fracture may not even be visible on routine radiography, leaving the presence of point tenderness with an abnormality of gait as the only criterion for diagnosis. Consultation with an orthopedist can be helpful for both diagnosis and appropriate management.

Orthopedic injuries are common in children of all ages, and the presence of growth plates in immature bones mandates vigilance from the pediatrician. Injuries that threaten growth may present only with subtle physical findings and negative radiographs. Suspecting an occult fracture or injury to the growth plate can save a child from a long-term deformity and protect his or her potential for growth.

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Comment: Better even than properly treating an occult bone fracture and a damaged growth plate is preventing the injury. Most fractures occur in children while they are at play, and many communities either have not set appropriate standards of safety for playgrounds and schoolyards or fail to enforce them. In the larger context, injuries remain by far the most common cause of morbidity and mortality to children in the United States, and our public health efforts have fallen far short of protecting our children as well as we should.

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