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In the past nine months, the world has witnessed the outbreak of not one but two waves of pandemic influenza due to a new virus of swine origin. World public health authorities moved quickly to contain what appeared initially to be the severe pandemic that had been anticipated for so long.

Wave two has now subsided, giving public health authorities and health care providers alike the opportunity to assess the situation. The pandemic was not a hoax, as some have suggested, but it has been much lower in intensity than many expected. How did this happen? What have we learned about novel A(H1N1)? What is the likelihood that it will be back? This article considers these and other questions.

— The Editor

A(H1N1) 'Swine Flu' 2009 / 2010: Where We've Been, What We Now Know, Where We May Be Heading

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Prelude to a Pandemic

On April 21, 2009, the Centers for Disease Control and Prevention (CDC) released a report about two patients in Southern California who had become ill as a result of infection from a previously unknown influenza virus.[1] The new virus was a Type A, subtype H1N1, but distinct from the A(H1N1) human subtype that has circulated since the 1970s. It appeared that the origin of the virus was swine, so it became known initially as swine flu. Since then it has been referred to by a variety of names, including novel influenza H1N1 and influenza A(H1N1) 2009. The date of the first infection was March 28, and both patients recovered fully.

The first report was little noticed even in the medical world. A number of unusual sub-types of influenza originating in animals

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can occasionally be found in humans, usually as a result of close exposure to infected animals. There are human sub-types of flu and porcine sub-types of flu and avian sub-types of flu, and generally they stay within the species of origin, doing significant harm only to that species. The species barrier is only rarely breached, and usually not in a sustained fashion.

A recent major exception to that rule is high pathogenicity A(H5N1) avian influenza which burst on the world scene in Hong Kong in 1997 and subsequently spread throughout the entire Eastern Hemisphere.[2] It has never been found in the Western Hemisphere. H5N1 "bird flu" received much attention from both the scientific community and the general public over the past 12 years because it spread so rapidly over a huge geographic area and because it continues to exhibit a mortality rate of over 60%. By comparison, the mortality rate from the Spanish influenza of 1918 was only 2%, but because Spanish flu had a very high attack rate, it led to the most deadly pandemic of any kind in world history.

The 20th century saw three major pandemics: A(H1N1) "Spanish flu" in 1918, A(H2N2) "Asian flu" in 1957, and A(H3N2) "Hong Kong flu" in 1968. The historical record shows that the world experiences on average three to five influenza pandemics per century. As a result, there was a renewed focus in preparation for an influenza pandemic at the state, national, and international level. The Strategic National Stockpile, a mechanism to store essential medical materials that would be needed during a pandemic, was built up with many supplies, most notably the antiviral medications oseltamivir (Tamiflu) and zanamivir (Relenza).

Although H5N1 avian flu remains a highly lethal infection for humans who are unlucky enough to become infected by it, by 2008 it was clear that it would not be the cause of our next pandemic.[3] Three essential ingredients are needed for a pandemic to occur. First, a new virus must appear, one that has never been encountered by humans before. Second, there must be no pre-existing immunity to the new virus. Third, there must be sustained rapid spread from person to person. H5N1 met the first two criteria, but sustained rapid human-to-human spread has not been observed. Occasional person-to-person spread has been observed, particularly with intense exposure such as within a family unit, but casual contact does not appear to pose a risk for infection. As of 2010, fewer than 300 people worldwide are known to have died from H5N1 influenza, making it more of a pandemic for birds than for humans.[3] The concern continues to be that a mutated form of H5N1 might emerge that does have the ability to spread quickly from person to person through casual contact alone. If that happens, a major pandemic is likely, with potentially catastrophic results.

Given this background information, the announcement by the CDC on April 23, 2009 that swine flu had been detected in six additional patients in Southern California and Texas caused alarm among flu experts.[4] There was evidence in these cases for human-to-human spread. Initial genetic analysis by the CDC indicated that the virus had components from human, swine, and avian sources. On April 24, the Mexican government confirmed that at least seven cases had been detected in their country, with some deaths acknowledged. Swift genetic analysis by the CDC confirmed that the strains found in Mexico were identical to the strains found in the United States.[4] Soon after, the Mexican government announced a cluster of 854 cases of pneumonia and 59 deaths in Mexico City thought to be due to H1N1. If these data were accurate, the mortality rate for this virus could be close to 8%, four times deadlier than the 1918 Spanish flu and almost as high as the 10% mortality seen with the aborted 2002 SARS epidemic. It appeared that the world might be witnessing the earliest stages of a new, deadly flu pandemic.

Events unfolded rapidly over the next few days. On April 26, 20 cases were confirmed in five U.S. states, including a cluster of students in New York who had recently traveled to Mexico for spring break. In response, the Department of Health and Human Services (HHS) declared a public health emergency in the United States, and 25% of the antiviral supplies in the Strategic National Stockpile were released to the states for distribution.[5] By April 27, swine flu had been detected in at least five other countries, including New Zealand, Canada, UK, Spain, and Israel.[6] This caused the World Health Organization (WHO) to raise the pandemic alert level from III to IV on the six-level scale, indicating that a new virus with pandemic potential had been detected with "increased" person-to-person spread.[7]

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On April 29, just eight days after the world learned about swine flu, the WHO raised its pandemic alert level to V, indicating "significant" person-to-person spread of the virus.[8] Analysis of novel A(H1N1) strains showed that the virus was completely resistant to the adamantane class of influenza antiviral medications (Amantadine, Rimantadine), but no resistance to the neuraminidase inhibitors oseltamivir or zanamivir was detected.[9] A few days later, the CDC issued recommendations to prescribe oseltamivir or zanamivir to anyone with confirmed, probable, or suspected swine flu. The U.S. Food and Drug Administration (FDA) also issued an Emergency Use Authorization (EUA) for the use of oseltamivir in children younger than 1 year of age, an age group for which formal FDA approval had not yet been granted.[10] The objective behind these policies was to try to limit the spread of the virus through the use of antiviral medications.

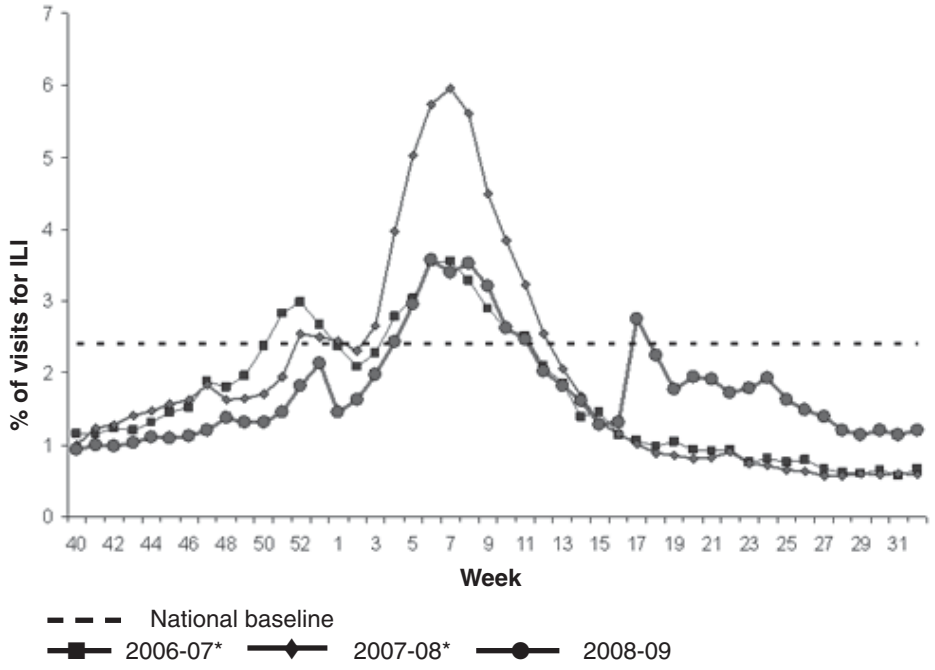
By the end of May 2009, there were 8,585 probable and confirmed cases of swine flu in the United States with 12 deaths, for a mortality rate of 0.14%.[11] The Mexicans government now reported 4,910 confirmed cases and 85 deaths, for a mortality rate of 1.7%, still very high but much lower than the 8% rate reported earlier.[12] There were also 2,673 cases of swine flu reported in 46 other countries around the world with only three deaths. It had become clear that except for the unusual number of deaths reported from Mexico, this virus was causing mortality in rates similar to or lower than mortality rates expected from seasonal flu. The data also indicated that the population most affected by the virus was the young; initial reports in the United States showed that the median age of those infected was 16 years, with very low attack rates for people older than age 50 years. This is highly unusual for influenza viruses, which infect 5%–10% of Americans each year and cause widespread morbidity and mortality in the elderly, who comprise over 90% of the 36,000 annual deaths due to flu.[13]

On June 11, the WHO raised the pandemic alert level to Phase VI, indicating sustained transmission of a novel influenza virus from person to person.[14] The world was now officially in an influenza pandemic.

While H1N1 continued to spread throughout the United States and the rest of the world during the month of May, the sharp upturn in influenza-like illness (ILI) that was detected during the week ending May 2 (week 17 for CDC tracking purposes) was not sustained. By week 18 the national level of ILI had begun to decline, and by week 19 it sharply declined to below the epidemic threshold of 2.4%. *(See Figure 1.)* The explosive spread of infection feared by many in the influenza world did not occur, and it appeared that, in fact, the outbreak might be receding even as the pandemic was being officially recognized. In part due to these data, the CDC in June issued new recommendations to focus the use of antiviral medications on those who were hospitalized with likely swine flu and outpatients at high risk for complications from influenza infection. Treatment was no longer recommended for otherwise healthy outpatients even if swine flu was likely.[15]

Figure 1. Percentage of Hospital Visits for Influenza-like Illnesses, as of August 15, 2009

Reported by the U.S. Outpatient Influenza-Like Illness Surveillance Network (ILINet), national summary 2008-09 and previous two seasons



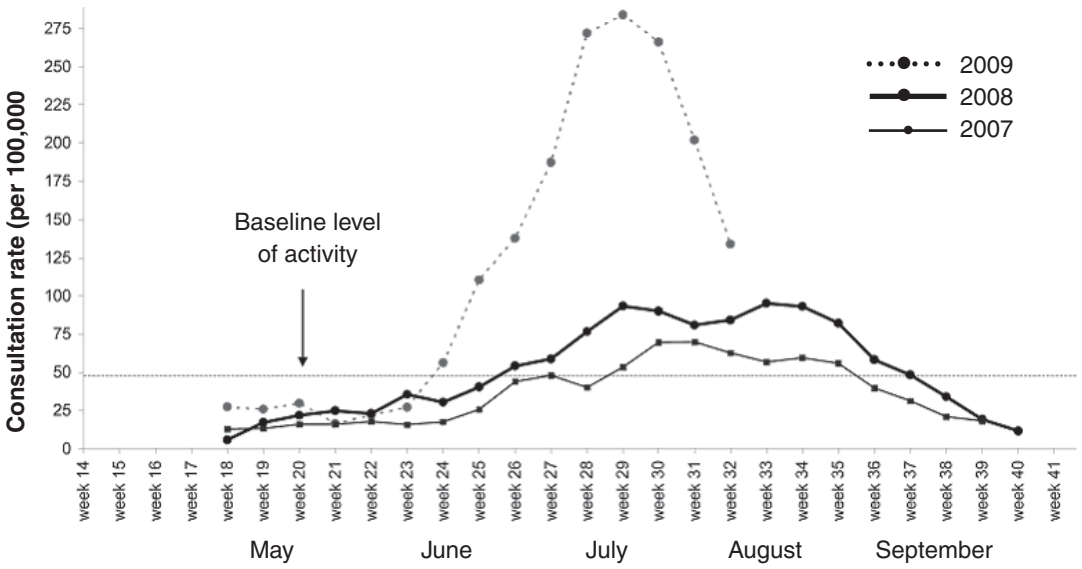
* There was no week 53 in the 2006-07 and 2007-08 seasons; therefore, the week 53 data point for those seasons is an average of weeks 52 and 1.

The Summer Lull

Summer in the Northern Hemisphere, corresponding roughly to CDC weeks 22–34, brought continued slow spread of the virus. The overall rate of patients reported with ILI remained elevated compared to the rate normally seen for the time of year, but it was still hovering below the threshold that helps to define the epidemic period during a "regular" flu season. Throughout the summer, only a handful of U.S. states reported widespread infections, meaning at least half of the counties in the state had confirmed the presence of novel H1N1 during the preceding week.

The summer lull was viewed by many as a chance to better prepare for the anticipated return of swine flu in a second major "wave." It was unknown when this second wave would occur. Would it arrive in the winter, during the time when influenza normally circulates at epidemic levels? Or would it arrive at an earlier time, perhaps after the end of the flu season in the Southern Hemisphere, which generally runs from mid-May to mid-July? The other unknown was what strain of the new virus would predominate in our second wave. Influenza viruses are character-

Figure 2. Influenza-like Illness in New Zealand, 2009



Adapted from Department of Health and Human Services. Assessment of the 2009 A(H1N1) pandemic on selected countries in the southern hemisphere: Argentina, Australia, Chile, New Zealand, and Uruguay. Aug. 26, 2009. Available online at http://www.flu.gov/professional/global/southhemisphere.html.

ized by a high rate of mutation. One of the biggest concerns was that novel H1N1 would cause high levels of infection in the Southern Hemisphere during the May–July flu season and provide a breeding ground for mutated versions of the virus. The fear was that while the virus we knew so far showed low degrees of lethality, the virus to come might be significantly worse.

There was a concerted effort during our summer months to produce a vaccine against swine flu. If we could produce the vaccine quickly, there might still be a chance to avert a potential disaster when the virus returned in the fall or winter. This effort was being made not just in the United States but all over the world. Candidate strains were identified by the CDC as early as June, after which the long, slow process of producing vaccine began. In general, at least six months are required to produce sufficient amounts of vaccine after a new strain of influenza virus appears. The irony was that although we had extra time to prepare a vaccine, we had no way to know if the vaccine would still be effective if a mutated form of the virus appeared.

Vaccine mismatches happen with some degree of regularity due to the unpredictable nature of influenza virus mutations. The Fujian flu of 2003/2004 is a case in point. That year, candidate strains were chosen in early 2003 as always, based on the viruses that were circulating at the end of the Northern Hemisphere flu season and based on new strains detected in the Southern Hemisphere early in their flu season. Unfortunately, Fujian flu wasn't detected until the fall, too late to be incorporated into that year's flu vaccine. Fujian was an A(H3N2) virus that, like many H3N2 subtypes, caused severe illness. As predicted, the 2003/2004 was a particularly severe season. When the CDC retrospectively analyzed the mismatched flu vaccine against the Fujian strain in the spring of 2004, it was found to have less than 50% effectiveness.[16]

Flu Season in the Southern Hemisphere

While the swine flu continued to circulate in significant but relatively low numbers in the Northern Hemisphere during the months of June and July, flu season arrived in the Southern Hemisphere. Recognizing that the experience in the Southern Hemisphere might provide some insight about what might be coming in the fall and winter north of the equator, HHS began to gather data on the Southern experience. The countries surveyed included Argentina, Australia, Chile, New Zealand, and Uruguay due to their similarities to the United States with regard to demographics and economic development. A full report was issued on August 26 and is available online.[17]

There were a number of notable findings in this report. First, the experience in all countries was that influenza arrived as usual in May/June, peaked in mid-July, and decreased afterward. This pattern is consistent with typical flu seasons in the Southern Hemisphere, and showed that H1N1 2009 did not circulate for an unusual period of time. **Figure 2** shows the data on visits to local physicians in New Zealand for ILI during the months of April to August, showing a typical eight- to 12-week period of heightened activity. The winter peak in activity during this time was three times that seen in the previous two seasons, indicating a more severe season than usual.[17]

The predominant strain was 2009 A(H1N1) influenza, which accounted for 89% of all influenza viruses in the Southern Hemisphere by August. Notably, sequencing data indicate that the virus was genetically and antigenically stable during this time; there was no significant degree of mutation.[17] Thus, the virus in late summer was still similar to the strains being used to produce vaccine throughout the world.

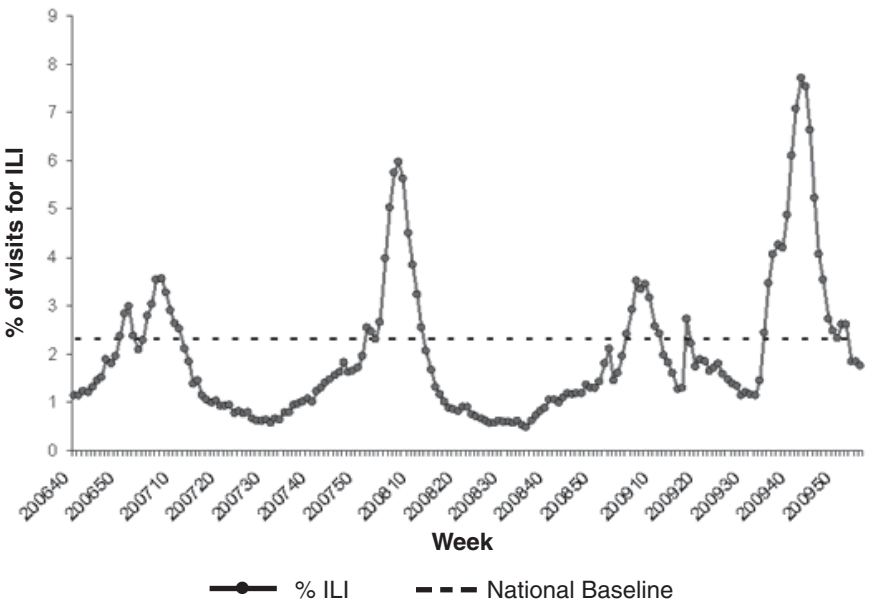
Clinical disease patterns resembled those from the United States and other countries to the north. 2009 A(H1N1) infections generally caused mild disease, with infections predominating in school-age children and adults younger than 65 years of age. Overall rates of hospitalization, severe illness, and death due to swine flu were similar to those observed thus far in the United States. Seventy-one percent of reported deaths occurred in adults 25–64 years old, with very few deaths reported in the over-65

category. The patients most at risk for complications included pregnant females and individuals with underlying medical conditions.

Antiviral medications were used in all five countries to treat various categories of patients, particularly those with medical conditions putting them at high risk for complications due to flu and those ill enough to be hospitalized. The virus remained sensitive to neuraminidase inhibitors throughout the season and no significant rate of resistance was found.

There were several implications of these findings for the expected second wave of infection in the Northern Hemisphere. It appeared that 2009 A(H1N1) had not mutated to a more deadly form during the time it circulated in the Southern Hemisphere. This meant that work could continue on vaccine development using strains selected early in the pandemic, with the growing likelihood that there would be a good match between the circulating strain and the vaccine strain. Furthermore, while certain subsets of the population were being affected more than would be expected during a normal seasonal flu outbreak, the overall lethality of the virus appeared to remain at or below that seen with seasonal flu.

Figure 3. Percentage of Visits for Influenza-like Illness (ILI), October 1, 2006 to January 23, 2010



Source: U.S. Outpatient Influenza-like Illness Surveillance Network (ILINet)

The Second Wave Arrives

By August 2009 A(H1N1) had been circulating in the United States for well over three months, and an estimated 1 million people had been infected.[18] CDC had recommended on July 24 that states discontinue reporting on individual cases of confirmed or probable swine flu and instead provide information on influenza-associated hospitalizations and deaths. During the period from April to the end of August, there were 9,079 laboratory confirmed hospitalizations and 593 deaths due to 2009 A(H1N1), a mortality rate of 6.5% in hospitalized patients but under 0.1% among all those estimated to be infected.[19] Rates of ILI continued to hover somewhat over the levels usually seen at that time of the year, but were well below epidemic thresholds.

A sudden increase in ILI was noted first in Georgia, then in neighboring states in the Southeast United States, beginning around August 15.[18] The Georgia State lab and CDC quickly confirmed that the increase was due to 2009 A(H1N1). Soon, rates of ILI increased to a level usually seen only during the peak periods of seasonal influenza in the winter, first in Georgia, then in other states. Over the next six weeks, more and more states reported increased levels of activity, peaking at 49 of 50 states in the continental United States reporting widespread infection as of the week ending October 16. Over the six weeks following October 16, swine flu decreased in activity at a rate similar to that observed in the Southern Hemisphere. *(See Figure 3.)*

Swine Flu: What We Now Know

During the first few months of the pandemic, most of the information on swine flu came from CDC and WHO briefings, *MMWR* dispatches, and online news reports regarding new information presented at national and international medical conferences. Beginning in the late summer, more and more information began to appear in medical journals throughout the world. The discussion below is a brief description of what we have learned about 2009 A(H1N1) up to the beginning of 2010.

The Virus. Genetic analysis of isolates from the original two infected patients in Southern California showed that the majority of the genes were similar to those of swine influenza viruses that have circulated in U.S. pig populations since 1999. However, two of the eight genes were from a Eurasian swine flu lineage. This combination of genes had never been found before in swine or human influenza viruses anywhere in the world.[1] More detailed studies on isolates from Mexico and the United States were published in July. One revealed that 2009 A(H1N1) influenza virus has components that can be traced to humans, birds, and swine, with swine the most recent host for all eight genes.[20] Very little genetic variability could be found in any of the isolates tested, suggesting that the introduction into humans may have been a single event.

Two publications in July pointed out that this virus and all viruses that have circulated in humans over the past 91 years can be traced to the 1918 A(H1N1) "Spanish flu" pandemic.[21,22] The 1918 virus, thought to be derived from avian sources, was transmitted to pigs sometime before 1930 and remained relatively stable in the swine population until the late 1990s. Meanwhile, A(H1N1) viruses circulated in humans from 1918 until the outbreak of a new pandemic caused by A(H2N2) in 1957, disap-

peared for a time, then re-emerged in 1977. From 1977 to 2009, human A(H1N1) continued to evolve, leading to substantial divergence between human A(H1N1) and swine A(H1N1) viruses by the end of the century. Around 1998, swine A(H1N1) viruses reassorted with contemporary human A(H3N2) viruses and an avian influenza virus, creating a "triple-reassortant" A(H3N2) virus in swine populations throughout North America. Shortly after, similar triple-reassortant A(H1N1) was also detected, which evolved rapidly over the next 10 years. These events all served to create in swine a reservoir of viruses so antigenically distinct from their related human counterparts that a human pandemic could result.

Transmissibility. The basic reproductive number (R0) represents the number of infections caused by spread from a currently infected person who is introduced into a completely susceptible population.[23] An R0 > 1 can support an epidemic while an R0 < 1 cannot. At an R0 of 2, spread of infection becomes logarithmic. The value for R0 is as high as 10 for varicella or measles, while values of 1.4 to 1.6 are typical for influenza. Soon after it was recognized that the 2009 A(H1N1) influenza virus was able to spread from person to person, work began on learning how transmissible it was. Early studies in Mexico and Peru showed that the R0 for this virus is probably between 1.2 and 1.7, making its transmissibility similar to seasonal flu.[24,25] The R0 for the 1918 Spanish flu was probably 2.0 or higher.

As of June 1, 2009, 938 case reports of persons with confirmed or probable 2009 H1N1 influenza had been collected by the CDC. Analysis of those cases revealed that acute respiratory illness developed in only 78 of 600 household contacts (13%), and that no secondary respiratory illness developed in 72% of the 216 households.[26] It was also found that household contacts younger than 18 years of age were twice as susceptible as those 19–50 years of age, and that those older than age 50 were the least susceptible. There was a mean of 2.6 days between onset of symptoms in an index patient and onset of symptoms in an infected household contact. A related study of an outbreak that occurred in a New York school from April 24 to May 8 showed a similar secondary attack rate in households of 17%.[27] That study also showed a significantly higher attack rate for students than for school employees. These data confirmed that this flu virus was attacking the young in far greater numbers than those older than age 50, a pattern greatly different from that expected from seasonal flu.

Epidemiology. Data on 1,591 confirmed 2009 A(H1N1) infections from Mexico and the United States during the period March 1 to May 5 revealed that two thirds of the infections were found in school-age children and adults younger than age 30, and another 10% of those infected were younger than 5 years of age.[28] Only 10% of infected patients were older than age 45, and 2% were older than age 60. In contrast to seasonal flu, the virus was preferentially infecting the young, and those older than 50 years were largely spared. *(See Table 1.)*

A similar distribution of infections was shown in a report from the Chicago area.[29] Investigators analyzed data from 1,557 patients with laboratory-confirmed 2009 A(H1N1) from April to July. The median age of confirmed cases was 12 years. The overall attack rate was highest among children age 5–14 years, at 147 per 100,000 population; next highest at 113 per 100,000 for children age 0–4; and lowest for those older than age 60, at 10/100,000. There was a consistent decrease in the attack rate for each successive age group older than the age of 14 years.

Table 1. Novel Influenza A (H1N1), United States and Mexico, by Age and Hospitalization (March 1 to May 5, 2009)

Age	Number of cases (% of Total)	Number hospitalized (% for age)
< 5	166 (11)	13 (8)
5-14	452 (29)	13 (3)
15-29	563 (36)	22 (4)
30-44	222 (14)	25 (11)
45-59	130 (8)	8 (6)
≥ 60	31 (2)	2 (6)
Total	**1,564**	**83**

Adapted from *MMWR* 2009;58(17):453-458.

A study published in the *New England Journal of Medicine* in September shed some light on a possible reason for this unusual age distribution of infection.[30] In this study from the CDC, investigators measured cross-reactive antibodies to 2009 A(H1N1) in stored serum samples from people who had been vaccinated with seasonal flu vaccine from 2005 to 2009, and from stored samples from patients who were vaccinated with the swine influenza vaccine of 1976. The analysis showed that people younger than 30 years of age had little to no evidence of pre-existing cross-reactive antibodies to 2009 A(H1N1), but at least 34% of those born before 1950 and 100% of those born between 1910 and 1929 had titres of 80 or more. Furthermore, 54% of patients vaccinated against the 1976 A(NJ/76) (H1N1) swine flu had evidence of cross-reactive antibodies against 2009 A(H1N1). Vaccination with contemporary seasonal flu vaccines did not lead to significant increases in cross-reactive antibodies against 2009 A(H1N1) in any age group. These data suggest that prior exposure to a 1918-like H1N1 virus had occurred in many older adults, and that this prior exposure resulted in cross-reactive antibodies against 2009 A(H1N1). This theory is also consistent with the information on the evolution of influenza viruses discussed in the previous section.

Symptoms. Two early reports on the swine flu showed a pattern of symptoms that was similar to seasonal flu, but with key differences. The first, released on April 30, was based on data col-

lected primarily from a New York City high school with 2,686 students and 228 staff members.[31] Between April 20 and April 24, at least 222 students and staff developed acute respiratory illnesses. There were 42 specimens collected on symptomatic patients in local physician offices and emergency departments, and 37 (88%) were later confirmed positive for swine flu by the CDC. Subjective fever was found in 96% of those with confirmed swine flu, and each of the following symptoms were found in at least 80%: cough (98%), fatigue (89%), headache (82%), sore throat (82%), runny nose (82%), chills (80%), or muscle aches (80%).[31] These symptoms are typical for influenza in this age group. However, 55% reported nausea, and 48% reported diarrhea, which are relatively rare in seasonal influenza infections.[32] Subsequent reports confirmed a relatively high rate of vomiting and diarrhea in patients with confirmed swine flu.[28,33,34]

Hospitalized Patients. In the May 8 *MMWR* report on the swine flu outbreak in a New York school, there was only one short hospitalization and no deaths among the 44 confirmed cases of 2009 A(H1N1), indicating that, at least in this low-risk population, the virus did not appear to be any more severe than seasonal flu. Subsequent data confirmed that hospitalization rates for school-age children were no higher overall than for seasonal flu.

Characteristics of patients who did require hospitalization have been detailed in several reports and have a consistent pattern: unusually high proportions of the young required inpatient management, two thirds had underlying medical conditions, about one quarter required management in an intensive care unit, and 7%–11% died.[33,34] These data are in sharp contrast to data from several decades of surveillance for seasonal flu, which show that the overwhelming majority of hospitalizations due to influenza are in patients older than 65 years of age, with a secondary peak in hospitalizations for children younger than 5 years of age.[35]

In the first report, data were extracted from the charts of 272 patients hospitalized with confirmed swine flu in 24 states from May 1 to June 9.[33] This represented about 25% of the 1,082 hospitalizations reported to the CDC during this period. Seventy-three percent had at least one underlying medical condition, including 60% of the children and 83% of the adults. The median time from onset of symptoms to hospital admission was three days. The median age of the patients was 21 years. Forty-five percent of the patients were children younger than 18 years; over one third were between 18 and 49 years of age; and only 5% were age 65 or older. Forty percent of those who had a chest radiography performed on admission had evidence for pneumonia. Sixty-five of the 272 patients (25%) were admitted to an intensive care unit, and 42 required mechanical ventilation (14%). Overall, 7% of admitted patients were pregnant females, a group that comprises only 1% of the general population.

A subsequent report on 1,088 patients hospitalized with confirmed swine flu in California from April to August showed remarkably similar results.[34] In addition, while this study demonstrated a very high rate of admission for the young, particularly children younger than 1 year of age, hospitalized persons older than age 50 had the highest mortality. This study also found a strong association between morbid obesity and hospitalization.

A study from Argentina focused on hospitalizations for children infected by swine flu between May and July.[36] Rates of hospitalization were double the rate expected from seasonal flu. Care in an intensive care unit was required for 19%, and 17% required mechanical ventilation. Overall, 5% died. The hospitalization rate was twice the rate observed during the 2008 seasonal flu. Hospitalization rates were particularly high for children younger than 1 year of age, after which rates dropped off sharply. The most striking finding in this study was that the mortality rate was 10 times higher in children with 2009 A(H1N1) than it was for the seasonal flu circulating in 2007.

Two studies examined patients admitted to intensive care units for confirmed 2009 A(H1N1) infection.[37,38] Both reported mortality rates of about 17%. Both found an unusual concentration of patients in the pediatric or young adult age groups, with few patients older than age 65. Both reported a proportion of obese patients and pregnant patients that was significantly higher than in the general population.

Mortality. The early reports suggesting up to 8% mortality in Mexico proved to be overestimates. By May 8, the mortality rate in Mexico for confirmed cases had been revised down to 4.4%,[39] and further data gathered to June 1 showed an overall mortality rate of about 2%.[40] As of May 8, there were no deaths reported for the 309 persons with laboratory-confirmed infection outside of the United States and Mexico, and only two deaths out of the first 1,487 confirmed cases in the United States.

Annual mean influenza-associated mortality rates for underlying pneumonia and influenza deaths in the United States were estimated for various age groups in a study from 2003. This study showed a rate of 0.3 deaths due to flu per 100,000 in children younger than 1 year of age, 0.2 in children 1–4 years, 0.2 in patients age 5–49 years, 1.3 in those 50–64 years, and 22.1 for patients older than age 65.[41] This study also showed that 90% of the influenza-associated deaths occur in persons older than age 65.

Data on mortality due to influenza in children have been collected nationally by the CDC only since the severe 2003–2004 Fujian flu epidemic, when a total of 154 deaths were recorded nationally for a mortality rate of 0.2 deaths per 100,000 children.[42] In all subsequent years until 2009, the total number of deaths due to influenza in children have not exceeded that number. By comparison, the mortality rate for children with confirmed 2009 A(H1N1) influenza reported in a recent report from Buenos Aires from May to July was 1.1 per 100,000 children, five times the rate from Fujian flu and 10 times the rate for the less severe 2007 seasonal flu.[36]

In August, an analysis of the first 36 deaths in U.S. children from laboratory-confirmed 2009 A(H1N1) showed that two thirds of the children had high-risk medical conditions. Furthermore, 92% of the children with high-risk medical conditions had neurodevelopmental conditions.[43]

Testing. When 2009 A(H1N1) first appeared, and for a short

time after, there was an urgent need to track progression of the pandemic through testing of subjects with flu-like illness. Some of this was done with point-of-care rapid influenza tests that were already available, and some was done with RT-PCR confirmatory assays that were available only through the CDC. Within only a few weeks of the appearance of the new virus, it was clear that spread throughout the United States had already occurred, and that the rise in ILI cases was due to swine flu. At that point, the CDC changed several of its recommendations about testing. Since it was no longer necessary or helpful to confirm every case, the CDC adopted a strategy of confirming only those cases of flu-like illness in hospitalized patients, which would permit public health authorities to monitor the severity of the pandemic as measured by hospitalization and death. Many state labs, which had obtained reagents for rRT-PCR testing from the CDC by July, adopted a similar strategy.

Early studies indicated that the available rapid tests lacked sensitivity in the detection of 2009 A(H1N1). Sensitivities of 60%–90% are reported for the detection of influenza by rapid tests during normal seasonal flu outbreaks.[44] In contrast, rapid tests approved for the detection of seasonal influenza showed sensitivities of only 10%–70% in the detection of swine flu depending on the viral titer.[45] Given this poor performance, the CDC issued recommendations to limit most influenza testing to nucleic acid amplification assays (rRT-PCR) in inpatients, and to manage outpatients based on their risk category and symptoms.[46] The decision to use antiviral medications depended on symptoms and risk category, not on the result of a rapid test, so there was little benefit in the use of rapid tests in the management of outpatients.

Treatment. A number of studies have been published on the use of neuraminidase inhibitors in the treatment of both children and adults with acute influenza infection. In general, the literature shows a modest benefit in both groups if the medication is started within 48 hours of symptom onset.[47,48] There is no published evidence that the use of neuraminidase inhibitors decreases significant morbidity or mortality in children, including children at high risk for complications due to flu. There are some studies showing reductions of morbidity and mortality in adults at high risk for complications, but those studies were in hospitalized patients or high-risk older adults.[49] There have been no published studies showing a reduction in mortality in young, healthy adults who were treated with neuraminidase inhibitors. In both children and adults, the primary benefit from the use of neuraminidase inhibitors for the treatment of acute influenza is a 12–36 hour reduction in symptom duration.

Initial recommendations from CDC regarding the use of neuraminidase inhibitors for the control of 2009 A(H1N1) focused on treating anyone with confirmed, probable, or likely swine flu. Once spread throughout the nation was apparent, CDC issued new recommendations to treat two primary groups of patients: those who were ill enough to be hospitalized with flu-like illness, regardless of test results, and all outpatients with underlying medical conditions that put them at high risk for complications due to influenza infection.[50] Treatment of ILI with antiviral medications was not recommended for the management of otherwise healthy outpatients who were not in any of the high-risk groups. These recommendations were revised several times, including a revision on September 22 that removed children between 2 years and 5 years of age from the list of groups at high risk.

Data reported early in the outbreak indicated that treatment with antiviral medications was beneficial to patients hospitalized with confirmed 2009 A(H1N1) infections, even if they had been symptomatic for more than 48 hours.[33] In October, the FDA also issued an EUA for Peramivir, a new neuraminidase inhibitor available only for intravenous use but not yet FDA-approved.[51]

Vaccine. Trials of 2009 A(H1N1) influenza vaccine began in mid-summer and continued into the fall. Early reports showed the vaccine to be immunogenic in a single dose for adults, with low side-effect and adverse-event profiles.[52,53] The vaccine was also found to be highly immunogenic and safe in a vaccine trial in infants and children.[54,55] It is interesting to note that although that vaccine trial in children showed excellent vaccine response after only one dose of the vaccine, the U.S. Advisory Committee on Immunization Practices continues to recommend two doses of vaccine for children younger than 9 years of age.[55] On Sept. 15, the FDA approved the manufacture of four monovalent vaccines against 2009 A(H1N1), and vaccines became widely available in the United States by mid October.[56]

There was considerable controversy around the vaccine from the start, in part because it arrived at a time when the second wave appeared to be already peaking, in part because the vast majority of those infected suffered only a self-limited illness, and in part because of concerns about vaccine safety. A national survey of parents conducted by the University of Michigan in August found that only 40% of parents intended to have their children vaccinated against 2009 A(H1N1).[57] A survey conducted by the Associated Press in October found that 38% of parents were unlikely to give permission for their child to receive the vaccine.[58]

An article published in *Lancet Infectious Disease* in December 2009 added another wrinkle to the topic.[59] The authors of this article point out that there is evidence that infection with influenza A viruses can induce at least partial protective immunity to unrelated influenza viruses. This "heterosubtypic immunity" may explain why people older than age 50 have such a low attack rate for A(H1N1) 2009. The authors of this article express concern that regular yearly influenza vaccination of immunologically naïve populations such as children may actually leave them more susceptible to future pandemic strains of influenza because they never have a chance to develop this heterosubtypic immunity through natural infection.

The Advisory Committee on Immunization Practices (ACIP) issued a statement in mid-summer identifying priority groups for vaccine.[60] The intent was to provide vaccine first to those most at risk for infection and severe complications. When the vaccine did arrive in mid-October, it was available only in limited supplies, so the high-risk groups, including medical workers, were the first to

receive the vaccine. Of the 24 million doses of vaccine administered in the United States by mid-November, 21 million doses (85%) went to members of the initial target groups.[61]

Vaccine manufacturers shipped 85 million doses of vaccine by December and 105 million doses by Jan. 4, 2010.[62] Despite the plentiful supply of vaccine, however, vaccine coverage has been poor. As of Jan. 2, 2010, only 20.3% of the U.S. population (61 million persons) had received the vaccine. Vaccine coverage has been estimated to be 29.4% for children younger than age 18, 38% for pregnant females, 22.3% for healthcare personnel, and 11.6% for adults between 25 and 64 years of age with underlying high-risk medical conditions.[61] So far, the 2009 H1N1 vaccine has shown a similar safety profile as seasonal flu vaccines, with no evidence of unexpected adverse events. The most common side effects have been mild, primarily limited to soreness, redness, tenderness, or swelling at the injection site.

Table 2. CDC Estimates for 2009 H1N1 Cases, Hospitalizations, and Deaths (April to December 12, 2009)

2009 H1N1	MID-LEVEL ESTIMATED* CASES	MID-LEVEL ESTIMATED HOSPITALIZATIONS	MID-LEVEL ESTIMATED DEATHS
0-17 years	18 million (33%)	78,000 (32%)	1,180 (9.4%)
18-64 years	32 million (58%)	145,000 (59%)	8,620 (77.2%)
65 years and older	5 million (9%)	23,000 (9%)	1,360 (12%)
TOTALS	**55 MILLION**	**246,000**	**11,160**

* Mid-point of estimated range; data adapted from CDC, available at http://www.cdc.gov/h1n1flu/estimates_2009_h1n1.htm#Table.

Season Summary: Where We Are Today

The peak of the second wave of A(H1N1) 2009 occurred during the week ending Oct. 24, 2009, when widespread activity was reported in 49 states. The majority of U.S. states still reported widespread activity by November 21, but by Christmas week there were only seven states with that level of activity.

The weekly percentage of outpatient visits for ILI also peaked during the week of October 24. The peak rate of 7.7% ILI was higher than the peak rate during the 2006–2007 influenza season (3.5%) or the 2007–2008 influenza season (6%).[63]

A 2010 CDC report summarized flu activity from August 30 to January 9.[64] Over 99% of specimens that were positive for influenza and subtyped during that period were confirmed 2009(H1N1). Influenza B accounted for 0.3% of identified isolates, and seasonal influenza A accounted for less than 0.1%. As of late January 2010 there was negligible seasonal influenza in circulation in the United States. Hospitalization rates since the second wave arrived in late August have been highest for children age 0–4 years, and generally declined for each successive age group. From the recent historical perspective, those rates are much higher than normal for children in the 0–4 year age group, and much lower than normal for adults older than age 65. Oseltamivir resistance has remained negligible. Only 52 oseltamivir-resistant viruses have been detected since April, and at least 77% of those patients had documented exposure to oseltamivir. In only three cases was there no known exposure to oseltamivir.

The impact of A(H1N1) 2009 was initially tracked by CDC and other public health authorities using only confirmed cases. By the middle of the summer, it became too cumbersome to continue this procedure, particularly since by then many health professionals were no longer performing confirmatory tests for outpatients with suspected infection. In July, experts from the CDC and the Harvard School of Public Health developed a model designed to estimate illness, hospitalization, and death due to the virus.[65] Their model indicated that every reported case of H1N1 may represent 79 actual cases, and every reported hospitalization may represent 2.7 hospitalized patients. The model yielded estimates for total infected, total hospitalized, and total deaths.[66] The mid-level estimated totals from April to December included 55 million infected Americans (18% infection rate), 246,000 hospitalizations, and 11,160 deaths. *(See Table 2.)*

While the overall rate of hospitalizations due to 2009 (H1N1) is in the range estimated for yearly seasonal flu, the breakdown by age is highly unusual. In a routine flu season, at least 60% of hospitalizations are in adults older than age 65, and fewer than 10% are in children younger than 5 years of age.[67] In contrast, adults older than age 65 accounted for fewer than 10% of hospitalizations due to 2009 (H1N1), while children accounted for more than 30%.

Deaths due to 2009 (H1N1) have been well below the 36,000 estimated to occur during a typical influenza season.[41] In a normal influenza season, adults older than 65 years of age account for more than 90% of all deaths, and children account for fewer than 1%. Deaths due to 2009 (H1N1) were primarily concentrated in younger adults age 18–64 years (77%), while 9% of deaths occurred in children and another 12% in adults older than age 65.

Conclusion

The year 2009 will be remembered as the year of the influenza pandemic due to novel swine-origin A(H1N1) virus. After the first wave appeared in late spring and summer, some experts predicted that the subsequent wave would infect 30%–50% of the population, lead to 1.8 million hospitalizations, and cause 90,000 deaths.[68] While this virus did prove to be unusually lethal for children and young adults compared with seasonal influenza, it was quite mild in its effect on older adults, and the overall attack rate was substantially lower than predicted. As a result, the overall number of infections, hospitalizations, and deaths was far below early estimates, and the strain on our health care system was far less than was initially feared.

As of February 2010, activity of 2009 H1N1 influenza has dropped below the epidemic threshold, and seasonal flu activity

remains negligible. It is possible that there will be no winter outbreak of influenza in the Northern Hemisphere this year. Despite concerns about successive waves of pandemic influenza due to this new virus, the historical record suggests that subsequent waves are not inevitable.[21] This virus has demonstrated only modest transmissibility compared to other influenza viruses. In addition, a large segment of the population appears to have at least some pre-existing protection against infection with 2009 (H1N1). Finally, a large proportion of the susceptible population has already been infected, leaving little "fuel" for further flare-ups. At this point, we should stay vigilant for the possible return of 2009 A(H1N1) swine flu, the influenza virus of the young.

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CME Questions

21. Which of the following is true about the beginning of the swine flu pandemic?
    A. It started in Southeast Asia and arrived in North America in April.
    B. Planners had about 6 week to prepare for the arrival of the virus in the United States.
    C. The period between initial identification and spread to other

CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge.

To clarify confusion surrounding any questions answered incorrectly, please consult the source material. After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a credit letter. When your evaluation is received, a credit letter will be mailed to you.

CME Objectives

Upon completion of this educational activity, participants should be able to:

- Recognize specific conditions in pediatric patients presenting to the emergency department;
- Describe the epidemiology, etiology, pathophysiology, historical and examination findings associated with conditions in pediatric patients presenting to the emergency department;
- Formulate a differential diagnosis and perform necessary diagnostic tests;
- Apply up-to-date therapeutic techniques to address conditions discussed in the publication;
- Discuss any discharge or follow-up instructions with patients.

continents was two weeks.

D. Spread to Europe did not occur until the time of the second wave in the United States.

22. Which of the following statements is true about H5N1 "bird flu"?
    A. It is easily transmissible from person to person.
    B. The mortality rate for those infected with H5N1 is well over 50%.
    C. This virus has already caused a minor pandemic.
    D. It has caused more than 150 deaths in North and South America.

23. Which is true about the response to swine flu by the Centers for Disease Control and Prevention (CDC) and other federal authorities?
    A. There were no recommendations regarding testing or treatment until the appearance of the second wave in September.
    B. The Department of Health and Human Services declared a public health emergency in the United States less than a week after the first case was identified.
    C. The pandemic was not considered severe enough to use any of the antiviral medications in the Strategic National Stockpile.
    D. Oseltamivir cannot be used in children younger than 1 year of age because the FDA has not yet approved it.

24. Which of the following is true about the overall mortality rate from swine flu?
    A. It is similar to the mortality rate from the 1918 "Spanish flu."
    B. It is higher than the mortality rate from H5N1 "bird flu."
    C. It is higher than for normal seasonal flu.
    D. It has been substantially higher in Mexico than in any other country.

25. Which of the following medications is useful against A(H1N1) 2009?
    A. Amantadine
    B. Rimantadine
    C. Acyclovir
    D. Peramivir

26. Which statement is true about A(H1N1) 2009 vaccine?
    A. Its introduction in October clearly caused the second wave to wane.
    B. Low supplies have led to few people being vaccinated.
    C. It has been linked conclusively to Guillain-Barré Syndrome.
    D. Most doses administered in October were given to high-risk groups and medical personnel.

27. What age group has had the most deaths from A(H1N1) 2009 in the United States?
    A. Age 0–4 years
    B. Age 18–64 years
    C. Age 65–85 years
    D. Older than age 85

28. What did we learn from the 2009 survey of the winter influenza season in the Southern Hemisphere?
    A. A(H1N1) 2009 did not show significant change in its genetic makeup.
    B. The flu season in the Southern Hemisphere lasted much longer than normal.
    C. The attack rate was typically high for the elderly.
    D. Rates of flu-like illness were well below the levels of 2007 or 2008.

29. What is the likely reason why people over 50 have not been infected in same proportion as they are in a seasonal influenza outbreak?
    A. Many people over 50 have pre-existing cross-reactive antibodies against A(H1N1) 2009.
    B. People older than age 50 have a better response to the A(H1N1) 2009 vaccine.
    C. Social distancing
    D. Effective use of oseltamivir prophylaxis

30. Which statement is true about the A(H1N1) 2009 pandemic?
    A. The virus is still causing widespread disease in most of the United States as of February 2010.
    B. This pandemic caused the deaths of many more children than is normally seen during a seasonal flu outbreak.
    C. The total number of deaths is higher than expected during a normal influenza season.
    D. The pandemic is over as of February 2010.

Answers: 21. C, 22. B, 23. B, 24. D, 25. D, 26. D, 27. B, 28. A, 29. A, 30. B

In Future Issues:

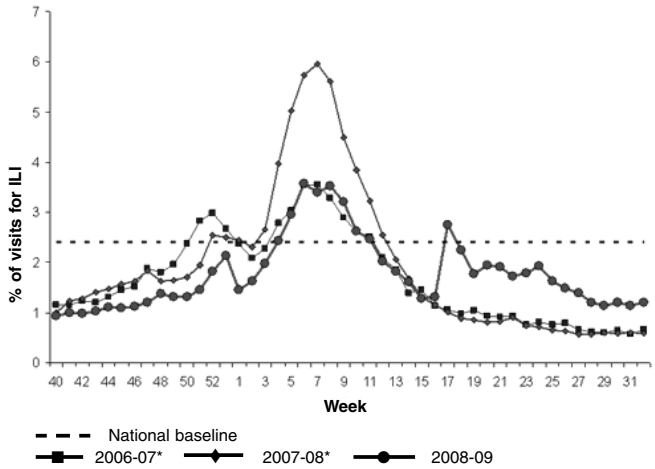
Evaluating Pediatric Trauma

PEDIATRIC Emergency Medicine Reports
The Practical Journal of Pediatric Emergency Medicine

H1N1: 2010 Update

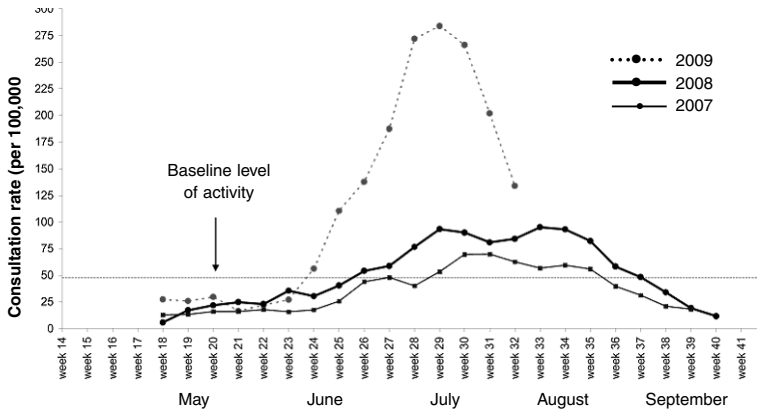
Percentage of Hospital Visits for Influenza-like Illnesses, as of August 15, 2009

national summary 2008-09 and previous two seasons



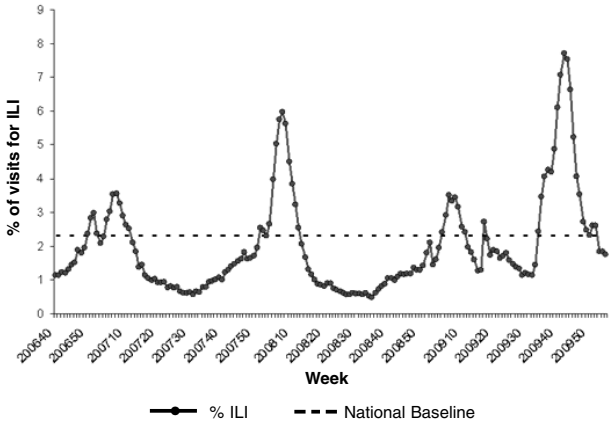
* There was no week 53 in the 2006-07 and 2007-08 seasons; therefore, the week 53 data point for those seasons is an average of weeks 52 and 1.

Influenza-like Illness in New Zealand, 2009



Adapted from Department of Health and Human Services. Assessment of the 2009 A(H1N1) pandemic on selected countries in the southern hemisphere: Argentina, Australia, Chile, New Zealand, and Uruguay. Aug. 26, 2009. Available online at http://www.flu.gov/professional/global/southhemisphere.html.

Percentage of Visits for Influenza-like Illness (ILI), Oct. 1, 2006 – Jan. 23, 2010



Source: U.S. Outpatient Influenza-like Illness Surveillance Network (ILINet)

Novel Influenza A (H1N1), United States and Mexico, by Age and Hospitalization (March 1 – May 5, 2009)

Age	Number of cases (% of Total)	Number hospitalized (% for age)
< 5	166 (11)	13 (8)
5-14	452 (29)	13 (3)
15-29	563 (36)	22 (4)
30-44	222 (14)	25 (11)
45-59	130 (8)	8 (6)
≥ 60	31 (2)	2 (6)
Total	**1,564**	**83**

Adapted from *MMWR* 2009;58(17):453-458.

CDC Estimates for 2009 H1N1 Cases, Hospitalizations, and Deaths (April – Dec. 12, 2009)

2009 H1N1	MID-LEVEL ESTIMATED* CASES	MID-LEVEL ESTIMATED HOSPITALIZATIONS	MID-LEVEL ESTIMATED DEATHS
0-17 years	18 million (33%)	78,000 (32%)	1,180 (9.4%)
18-64 years	32 million (58%)	145,000 (59%)	8,620 (77.2%)
65 years and older	5 million (9%)	23,000 (9%)	1,360 (12%)
TOTALS	**55 MILLION**	**246,000**	**11,160**

* Mid-point of estimated range; data adapted from CDC, available at http://www.cdc.gov/h1n1flu/estimates_2009_h1n1.htm#Table.

Supplement to *Pediatric Emergency Medicine Reports*, March 2010: "A(H1N1) 'Swine Flu' 2009 / 2010: Where We've Been, What We Now Know, Where We May Be Heading." *Author*: **James A. Wilde MD, FAAP,** Associate Professor of Pediatrics and Emergency Medicine, Medical College of Georgia, Augusta. *Peer Reviewer:* **Ghazala Q. Sharieff, MD, FACEP, FAAEM, FAAP**, Division Director/Clinical Professor, Rady Children's Hospital Emergency Care Center and the University of California, San Diego; Director of Pediatric Emergency Medicine, California Emergency Physicians.

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Trauma Reports

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March / April 2010

Injuries to the genitourinary system occur in 10% to 20% of patients sustaining major trauma.[1-3] The majority of these injuries are not immediately life-threatening and may not be as dramatically obvious as are other injuries. Unfortunately, the failure to identify them can lead to significant morbidity and, occasionally mortality.

Since many injuries of the urologic system are subtle, it is important that the examining physician not only recognize the signs of these injuries, but also develop an organized approach to their diagnosis and management.

This article reviews the mechanisms of injury, patient presentation, diagnostic approach, and management strategies for injuries to the urethra, urinary bladder, ureter and kidney.

— The Editor

Genitourinary Trauma: Etiology, Imaging, and Emergency Management

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Peer Reviewer: **John P. Santamaria MD**, Affiliate Professor of Pediatrics, USF School of Medicine, Tampa, FL

Urethral Injuries

Definition of the Problem and Epidemiology. Urethral injuries are rare, constituting about 10% of all injuries to the genitourinary system. However, they have the potential to be among the most debilitating type of urologic injuries because of the incidence of complications including impotence, incontinence and urethral strictures.[1] Urethral injuries are almost always the result of blunt trauma such as motor vehicle or bicycle accidents, falls or pedestrian injuries and are often seen in relation to pelvic fractures.[1-3] Uncommonly, urethral injuries are the result of penetrating trauma, such as a gunshot wound, stabbing, or a bite from an animal or human. Occasionally these injuries can be caused by foreign body insertion (which is often self-inflicted) or strangulation.[1,4] Urethral injuries are seen almost exclusively in the male population, with a higher incidence in

Statement of Financial Disclosure

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males 15-25 years of age.[1] Within the female population, urethral injuries almost always occur in relation to pelvic fractures or vaginal lacerations.[5,6]

Mechanism and Pathophysiology. Injuries to the urethra are classified as penetrating (gunshot wound or stabbing), blunt (straddle injury or penile fracture), or iatrogenic (caused by insertion of a urinary catheter or other foreign body).[7] Often these injuries are further classified by anatomical region. The male urethra is divided by the urogenital diaphragm into the anterior and posterior divisions. *(See Figure 1.)* The posterior division of the urethra is further divided into the prostatic and membranous sections, while the anterior division is divided into the pendulous and bulbous urethra regions.

Injuries to the posterior urethra usually occur as a result of a shearing force across the prostatomembranous junction. Since the prostatic urethra is maintained in a fixed position by the attachments of the puboprostatic ligaments, fracture of the bony pelvis can lead to stretching or tearing of the membranous portion of the urethra. Dreitlein and colleagues report "it is estimated that disruption of the posterior urethra accompanies 10% to 25% of pelvic ring fractures and 80% to 90% of posterior urethral injuries occur in combination with pelvic fractures."[8] While the majority of posterior urethral injuries occur in the setting of pelvic ring disruption, injury has taken place in the complete absence of pelvic fracture and in the setting of simple symphyseal diastasis.[9,10] The degree of pubic symphysis diastasis and amount of displacement of inferomedial pubic bone fracture fragments were recently identified as independent predictors of urethral injury.[11] Posterior urethral injuries are also associated with vaginal lacerations in women and up to 35% of bladder injuries.[8]

Figure 1. Anatomic Depiction of the Urethra

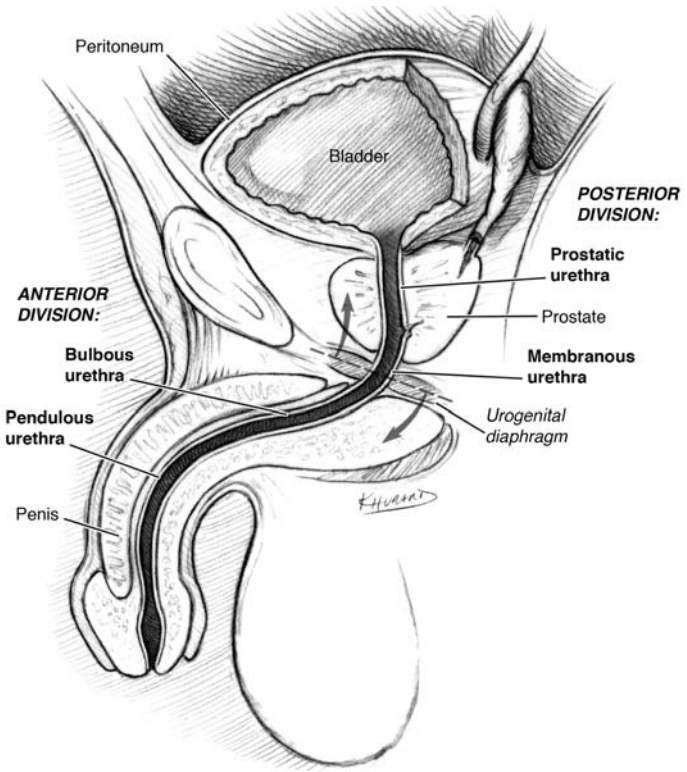

Illustration courtesy of Knox Hubbard

Unlike posterior urethral injuries, anterior injuries usually occur in isolation and most often result from blunt force trauma to the perineum that causes a crushing effect on the tissues of the urethra against the symphysis pubis.[8] Anterior urethral injury should be suspected in any person who presents to the emergency department (ED) with a history of straddle injury or direct trauma to the perineum. It has been suggested that most cases of bulbar strictures are the result of anterior urethral injury, in which case men may present to a physician years after the injury.[7]

Varying degrees of urethral injuries can also occur in 10% to 38% of patients who present with penile fracture, a rare condition that occurs when the corpora cavernosa ruptures as a result of blunt trauma, most commonly during sexual intercourse or masturbation.[7,12-13] Patients present to a physician after hearing a cracking or popping sound associated with a sudden, sharp penile pain. These symptoms are followed by detumescence, swelling, voiding difficulties, discoloration, and deviation of the penis.[14-15] Approximately one third of patients in these cases have complete urethral transection, and these injuries are often confirmed and repaired during the repair of the tunica and corpora cavernosa.[16]

Clinical Features. When the urethra is ruptured, blood is present at the urethral meatus in the majority of patients.[4,9] Other signs of urethral injury may include a patient's inability or difficulty with voiding; a palpable, distended bladder; or the inability to pass a urinary catheter into the bladder.[4,17] The presence of a hematoma in

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a sleeve distribution along the shaft of the penis indicates a distal urethral injury in which Buck's fascia remains intact, while the presence of a "butterfly" hematoma with extravasation into the scrotum also indicates distal urethral injury but occurs when Buck's fascia is ruptured.[7] Injury must also be suspected in the presence of a palpable rectal mass in association with urine or blood extravasation in the perineal region or in the presence of a cephalad or "high riding" prostate during digital rectal examination.

The diagnosis of urethral injury is much more difficult in females due to the length of the female urethra and its resistance to injury. Most females with an injury to the urethra present with incontinence. Additionally, the diagnosis of urethral injury must be entertained when the vaginal exam demonstrates a hematoma on the urethra or urine leak into the vagina.[18] There should also be a high suspicion of urethral injury in the presence of a pelvic fracture, regardless of whether or not there is blood at the introitus or one is able to pass a Foley catheter.[19] Many times the diagnosis can only be made in an operative setting during the repair of a pelvic fracture.[8]

Diagnosis and Imaging. In the past, the diagnosis of urethral injury was often based solely on the presentation of blood at the meatus accompanied by a difficulty or inability to void and a full, palpable bladder. Furthermore, the inability to pass a catheter into the bladder was also used as a diagnostic tool for urethral injury. Currently, many institutions consider diagnostic urethral catheterization inappropriate because it may introduce infection, lead to an increased incidence of stricture formation or convert a partial urethral rupture to a complete rupture.[2] Some sources in the urological literature currently suggest a single attempt to gently pass a urinary catheter into the bladder in the presence of blood at the meatus, or if there is any suspicion of urethral injury. If attempted, this should only be tried by an experienced clinician.[4]

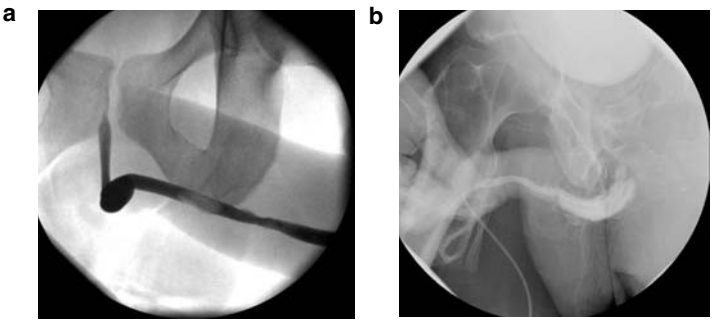
The retrograde urethrogram is the standard imaging study for the evaluation of urethral injury. *(See Figure 2a.)* It is performed by injecting 30 mL of contrast into the urethral orifice, ideally under fluoroscopic evaluation. Traditionally, the injury is evaluated by obtaining an oblique radiograph of the penis and abdominal region. In the setting of pelvic fracture, some authors recommend conducting the entire urethrogram in the supine position in order to maintain the integrity of a stable retropubic hematoma that may have formed.[19] The extravasation of contrast anywhere along the course of the urethra confirms the presence of disruption. *(See Figure 2b.)* If bladder filling is present, the disruption is classified as partial, whereas if no contrast reaches the bladder, the disruption is complete. In female urethral injuries, urethrography may be able to detect extravasation of contrast from areas of disruption, but often urethral evaluation is best accomplished by uretheroscopy.[20] Unfortunately, this makes the diagnosis of acute urethral injury in women difficult, and is the reason this diagnosis is often made only at the time of surgical repair of a pelvic fracture.

Table 1. Classification, Location, and Description of Urethral Injuries

CLASSIFICATION	LOCATION	DESCRIPTION
Type I	Posterior urethra	Stretched without disruption
Type II	Posterior urethra	Laceration above urogenital diaphragm
Type III	Anterior and posterior urethra	Partial or complete laceration with disruption of urogenital diaphragm
Type IV	Posterior urethra and bladder neck	Bladder neck laceration with extension into the prostatic urethra
Type IVa	Bladder base	Injury to bladder base simulating type IV injury due to periurethral extravasation
Type V	Anterior urethra	Partial or complete laceration of anterior urethra

Adapted from Goldman S, Sandler C, Corriere J, et al. Blunt urethral trauma: A unified anatomical mechanical classification. *J Urol* 1997;157:85-89.

Figure 2a-b. Retrograde Urethrograms



a. Normal retrograde urethrogram with air bubble in urethra
b. Retrograde urethrogram demonstrating contrast extravasation

Management. The initial management of urethral trauma should be made in the context of other injuries in coordination with other specialists including urologists and orthopedic surgeons. The primary goal of treatment should be achieving urinary continence while minimizing stricture formation and sexual impotence.

For partial tears that fail primary catheterization, or complete type II, III, and IV injuries, multiple surgical options exist. *(See Table 1.)* These include initial placement of a suprapubic catheter with delayed repair, endoscopic primary alignment or initial surgical exploration with bladder drainage and urethral realignment occurring at a later time.[8,21] Some practitioners consider immediate suprapubic catheterization with later repair the "gold standard" of treatment. Recent studies have suggested that complications to posterior urethral injury, such as sexual and voiding dysfunction, both related and unrelated to pelvic fracture, are more likely to be a result of the injury itself and not the method of management.[2,9,22] Furthermore, many of the latest studies show that early endoscopic or fluoroscopic realignment of posterior urethral injuries offers an effective option for repair, with similar or even improved functional outcomes (mainly decreasing the severity of stricture), when

Figure 3. Anatomic depiction of the bladder

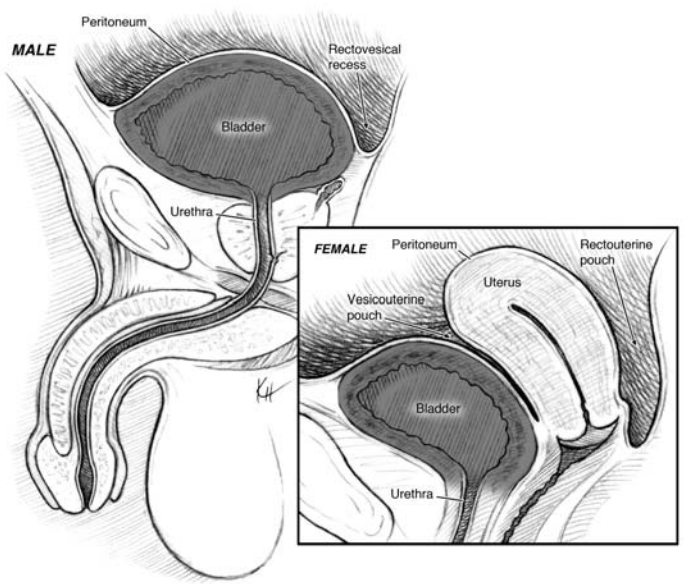


Illustration courtesy of Knox Hubbard

compared to suprapubic cystostomy and delayed repair.[22-26] In general, these sources suggest that, when it is practical, early realignment of urethral disruption should be performed, especially in hemodynamically stable patients.[25]

The treatment of injuries confined purely to the anterior urethra is similar to posterior treatment, with urethral catheterization and follow-up voiding cystourethrogram for minor contusions. Initial suprapubic cystostomy is the treatment of choice for most blunt crushing injuries to the anterior urethra, although bulbous urethral disruption has been successfully managed by immediate primary urethral realignment.[4,7,27] For penetrating anterior urethral wounds, early urethral realignment is the treatment of choice, with complete disruptions undergoing primary repair unless other major associated injuries or hemodynamic instability of the patient are present.[7]

Bladder Injuries

Definition of the Problem and Epidemiology. Bladder injuries represent one of the most common injuries involving the urinary tract and are frequently associated with severe multi-system trauma.[28] Blunt abdominal trauma is the most common cause of bladder injury, with most injuries occurring in males between the mean ages of 30 and 40 years.[29-31] Historically, injury to the bladder has been linked to a high rate of mortality, although most deaths are caused by accompanying non-urological injuries.[31-32] The recognition and treatment of bladder injuries is an important aspect of care for a victim of multiple trauma because of the relationship between bladder injury and mortality.

Mechanism and Pathophysiology. The empty adult bladder lies almost entirely in the minor pelvis and is therefore almost completely protected by the pelvic bones. As the bladder fills with urine, the fundus distends, the dome of the bladder rises out of the true pelvis into the abdominal cavity reaching the level of the umbilicus, and the bladder becomes more susceptible to injury.[33-34] For this reason, most injuries to the bladder occur as the result of either a direct blow to the lower abdomen when the bladder is distended or an associated pelvic fracture.[34]

Additionally, it is important to note the anatomy of the bladder in relation to the peritoneum. The peritoneum covers the superior surface of the bladder, which anteriorly continues to the abdominal wall. In males it reflects posteriorly to the abdominal wall creating the rectovesical recess, while in females, it travels over the uterus forming both the vesicouterine and rectouterine pouches before meeting the posterior wall of the abdomen.[35] *(See Figure 3.)* A bladder rupture occurring above the peritoneal reflection permits extravasation of urine into the intraperitoneal space, while injuries below the peritoneal reflection result in extraperitoneal extravasation.[36]

Common mechanisms of injury to the bladder include motor vehicle accidents, pedestrians struck by automobiles, falls, industrial accidents, gunshots, stabbings or direct trauma to the suprapubic region. Most bladder injuries caused by motor vehicle collisions can be classified as deceleration injuries and are seen in passengers wearing seatbelts or when occupants are thrown against an uncompromising object.[4] Fatal isolated bladder ruptures resulting from minor blunt trauma are documented in the medical literature, although a high percentage of these injuries are extraperitoneal and related to pelvic fractures.[37]

The vast majority of bladder ruptures are associated with pelvic disruption (83% to 90%), while bladder rupture is only seen in approximately 6% to 10% of patients with pelvic fracture.[8,21,29,38-40] In the setting of abdominal trauma, the normal protection of the pelvic ring may be lost due to the distention of the bladder above the pelvic ring or due to fracture of the ring itself, leading to laceration of the bladder. Widening of the sacroiliac joint and pubic symphysis diastasis, as well as fractures of the sacrum, iliac, and superior ramus, are fractures commonly associated with injury to the bladder. Multiple studies suggest that a widened symphysis pubis is the strongest predictor of bladder injury.[37,40-41]

Clinical Features. Gross hematuria is the hallmark finding in the patient with bladder injury, with an incidence at or approaching 100% in many recent studies.[30,34,42] Although gross hematuria is common, bladder injury can also present with minimal urinalysis findings, as is the case with spontaneous bladder rupture.[32] The majority of patients complain of lower abdominal or suprapubic pain and/or an inability to urinate.[32] The presence of gross hematuria or the presence of both pelvic fracture and microscopic hematuria together are the usual indications for either plain film or computed tomography (CT) cystography.[43]

Occasionally, bladder rupture is isolated and occurs without significant external trauma. Some penetrating bladder trauma, for instance, may have minimal findings on urinalysis due to the smaller size of the bladder laceration. In cases of isolated bladder rupture, presentation tends to be delayed and is often associated

with an increase in serum blood urea nitrogen and creatinine, abdominal distention, fever, or signs of an acute abdomen.[30] Most often, these patient present days after the associated trauma with complaints of ill-defined abdominal discomfort. They often have a history of head injury or alcohol intoxication contributing to a delay in diagnosis.[44]

Imaging and Diagnosis. Traditionally, plain film cystography is accepted as the most accurate radiologic study to diagnose bladder rupture. With the increased availability and use of CT imaging for trauma patients, many centers use either plain film cystography or CT imaging to evaluate injury to the bladder.[45] CT cystography is especially useful in stable trauma patients who are already undergoing CT evaluation for other trauma related injuries. Recent studies demonstrate that CT cystography is an accurate method for evaluation of bladder injury when performed in conjunction with routine CTs of the abdomen and pelvis.[40,43,46]

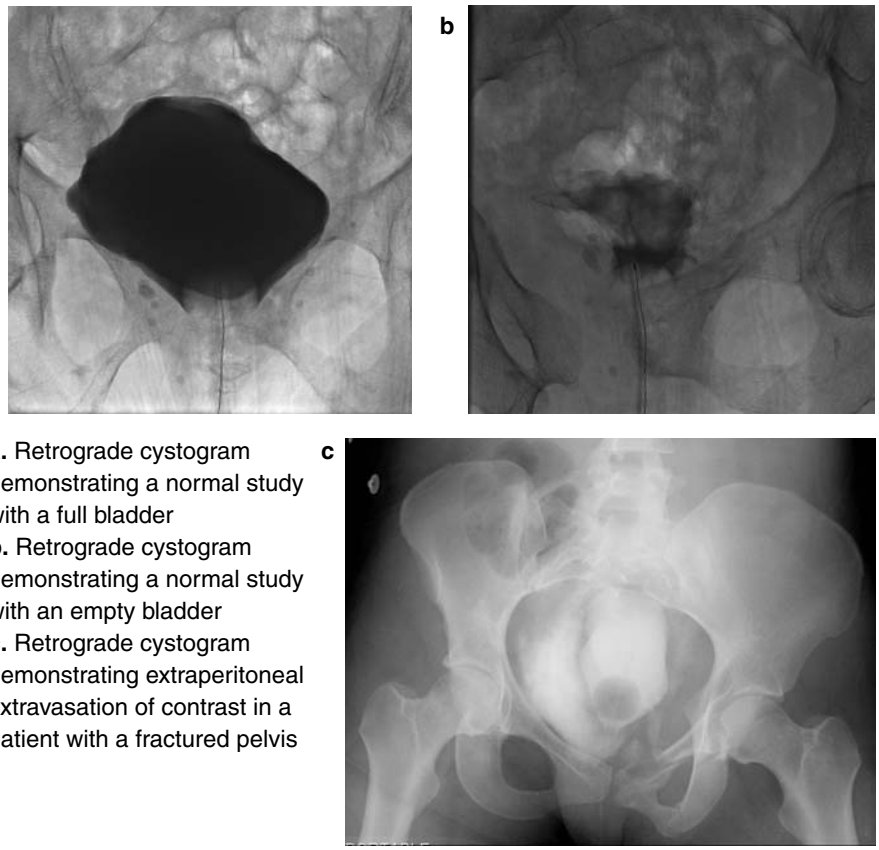
The procedure for doing a plain film cystography starts after an initial pelvic radiograph is obtained. If there is clinical suspicion of urethral injury, a retrograde urethrogram must be performed prior to the placement of the urinary catheter. After successful catheter placement, approximately 100 mL of contrast material is placed into the bladder via the urinary catheter and a plain film is taken to check for gross bladder extravasation. If this is negative, an additional 200–250 mL of contrast material (the total amount of contrast needed is approximately 5 mL/kg) is placed until the bladder is filled completely, and a radiograph of the entire abdomen is obtained. *(See Figure 4a.)* After obtaining the radiograph, the bladder is drained and a post-drainage radiograph is obtained to check for contrast extravasation behind a formally distended bladder.[34] *(See Figures 4b and 4c.)* Plain cystography is nearly 100% sensitive in detecting bladder rupture if films are obtained after both fully distending and emptying the bladder.

Like plain film cystography, CT cystography is done by performing retrograde filling of the bladder with a minimum of 350 mL of contrast material. Multiple studies demonstrate that 10 mm axial images obtained from the dome of the diaphragm to the perineum are adequate and sensitive for detecting bladder injuries.[47] *(See Figure 5a.)* Post-drainage images through the decompressed bladder are not necessary, as contrast extravasation behind the bladder will be seen on the axial sections.[34,40,47]

Plain film cystography with extravasation completed prior to routine CT scan of the abdomen and pelvis may hamper identification of pelvic arterial hemorrhage in some patients.[48] This is important to note because a delay in diagnosis may lead to delay in definitive treatment of ongoing bleeding. Practitioners should be wary when performing conventional cystography and consider waiting to perform the test until after routine abdomen and pelvis CT scans have been completed on stable patients. For similar reasons, retrograde filling of the bladder with contrast for CT cystogram prior to completion of a CT scan of the pelvis should be avoided.[48]

The bladder injury classification system based on cystography was revised in 2004.[49,50] *(See Table 2.)* The type of injury is deter-

Figure 4a-c. Retrograde Cystogram



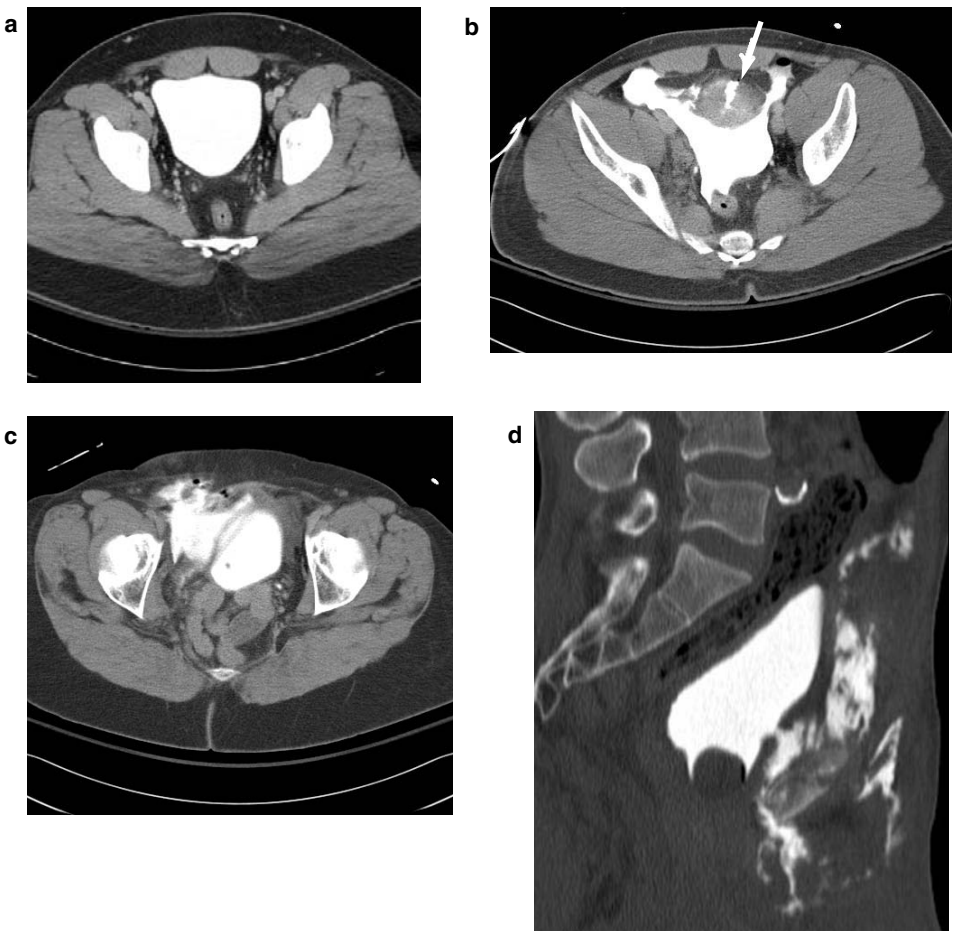
a. Retrograde cystogram demonstrating a normal study with a full bladder
b. Retrograde cystogram demonstrating a normal study with an empty bladder
c. Retrograde cystogram demonstrating extraperitoneal extravasation of contrast in a patient with a fractured pelvis

Table 2. Classification and Description of Bladder Injuries

CLASSIFICATION	DESCRIPTION
Type I	Simple contusion or incomplete tear of mucosa
Type II	Intraperitoneal injury with urinary extravasation into the abdomen
Type III	Extraperitoneal rupture with urine extravasation limited to the perivesicular space
Type IV	Both intraperitoneal and extraperitoneal rupture present

Adapted from Sandler C, Hall J, Rodriquez M, et al. Bladder injury in blunt pelvic trauma. *Radiology* 1986;158:633-638.

Figure 5 a-d. CT Cystogram of Bladder



a. Normal CT cystogram
b. CT cystogram demonstrating intraperitoneal extravasation of contrast material and bladder rupture. The hole in the bladder is seen in this view and highlighted by the arrow.
c. CT cystogram demonstrating extraperitoneal extravasation of contrast material and bladder rupture
d. Sagittal reconstruction of a CT cystogram with extraperitoneal extravasation of contrast material

mined by both the location of the injury and the degree of bladder wall involvement. The diagnosis of type 1 injuries, or bladder contusions, is typically a diagnosis of exclusion in patients with hematuria following blunt pelvic trauma for which no other cause can be found. Plain film and CT cystography will be normal. Type 2 injuries, or intraperitoneal bladder ruptures, show contrast material extravasation into the paracolic gutters, between mesenteric folds and around bowel loops. *(See Figure 5b.)* Approximately 25% of these injuries occur without associated pelvic fracture. Type 3 injuries, show extravasation either limited to the perivesical space (simple extraperitoneal rupture) or with extravasation beyond the perivesical space and via a variety of fascial planes into the anterior abdominal wall, penis, scrotum, or perineum (complex extraperitoneal bladder ruptures). *(See Figures 5c and 5d.)* Radiographic imaging of type 4 injuries, or combined bladder ruptures usually demonstrates patterns of extravasation consistent with both intraperitoneal and extraperitoneal injuries.[28,50]

Management. The first priority in the treatment of patients with suspected bladder injury is stabilization of the patient and treatment of associated life-threatening injuries.[8,50] The operative management of these patients usually involves multi-specialty cooperation between trauma surgeons, orthopedic surgeons and urologists. Further classification of these injuries is not needed in the case of penetrating trauma, as all penetrating abdominal injuries with suspected bladder involvement require operative exploration. However, with blunt bladder injury, further subdivision consisting of the type of injury (contusion vs. rupture) and the location of injury (intraperitoneal vs. extraperitoneal) is necessary when dictating treatment.

Bladder contusions (type 1 injuries) are deemed minor injuries and usually do not require therapy unless there is significant hemorrhage. Treatment involves placement of a large-bore urinary catheter (22-24F) which remains in place until the urine clears (usually in 1–4 days).[34,50] All intraperitoneal bladder ruptures (type 2) require operative management for multilayer closure. Traditionally, this has been done by laparotomy, but there are multiple reports of successful laparoscopic repair of intraperitoneal rupture.[42,50-52] Urine should be diverted using either suprapubic drainage or a large-bore urinary catheter, which can be removed once the urine is clear, usually after 10–14 days.[20] A recent study by Parry and colleagues suggests that similar outcomes and complication rates can be obtained via transurethral catheterization alone instead of suprapubic and transurethral catheters together.[53] Type 3 injuries (extraperitoneal bladder ruptures) often can be successfully managed by urinary catheter drainage alone. However, if the patient with a suspected type 3 injury is to be surgically explored because of associated injuries, formal bladder repair should be performed.[34,42] Combined bladder injuries (type 4) require surgical repair with a combination of the surgical procedures mentioned above.

Ureteral Injuries

Definition of the Problem and Epidemiology. The ureters are the least often injured component of the urological system, with injury occurring in less than 4% of penetrating and 1% of blunt trauma.[4] Overall, ureter injuries account for less than 1% of all external traumatic injuries to the genitourinary system.[54-55] Although rare, if the injury is unrecognized at the time of patient presentation, it can be devastating. Delays in diagnosis are associ-

Figure 6. Anatomy of the Ureters

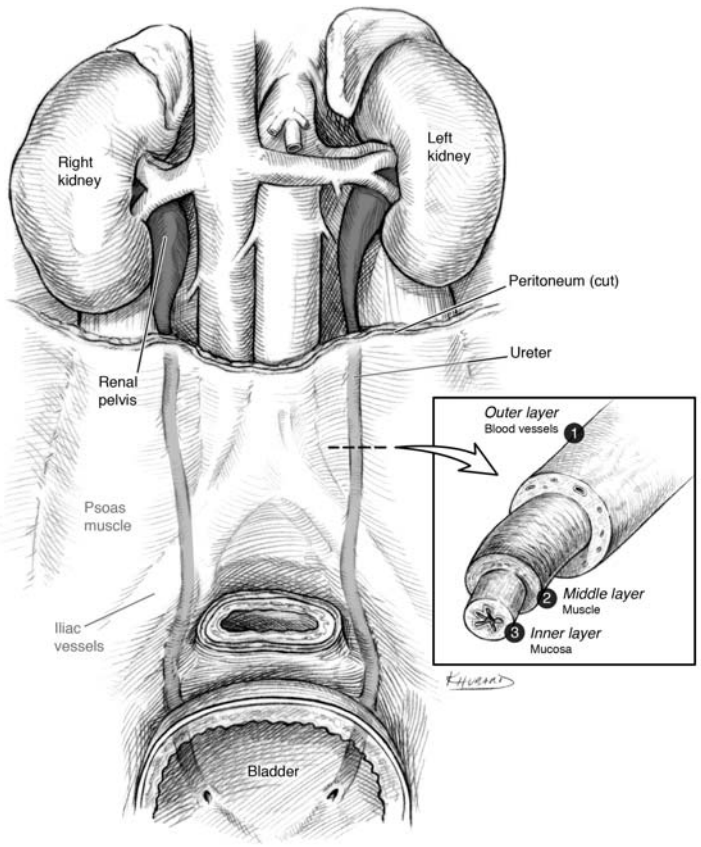


Illustration courtesy of Knox Hubbard

ated with morbidity from urinomas, fistulas, strictures, sepsis, the loss of renal function and renal destruction.[56-57] In general, adult patients with ureteral injuries tend to be young men injured by penetrating trauma.[56,58-59] Ureteral injuries seldom occur in isolation. Anywhere from 50% to 100% of patients have associated co-existing abdominal injuries to the small and large bowel, liver, kidney, bladder and iliac vessels.[57,60-61] The co-existing injuries and the critical condition of these patients make diagnosis of ureteral injuries difficult. Often these injuries are only discovered intra-operatively or when complications, such as fever, sepsis, leukocytosis, abdominal pain, or urinoma occur, which increases the morbidity of the condition.

Mechanism and Pathophysiology. The anatomy and mobility of the ureters help to protect them from trauma.[60] The ureters are bilateral, peristaltic, expandable muscular tubes that run between the renal pelvis and the posteriosuperior angle of the bladder. A ureter is composed of three layers: an outer layer, which harbors blood vessels; a medial layer, which consists of both longitudinal and circular smooth muscle; and an inner mucosal layer.[60] *(See Figure 6.)* The abdominal portion of both ureters lies in the retroperitoneum, adhering closely to the parietal peritoneum and anterior to the psoas major muscle. As the ureters pass into the pelvis minor, they cross the rim of the pelvis, traveling anterior to the origins of the external iliac arteries and then posterorinferiorly on the lateral wall of the pelvis. In males, the ureters then run lateral to the ductus deferens and enter the bladder superior to the seminal vesicle. In females, the ureters run close to the fornix of the vagina before entering the posteriosuperior bladder.[62]

The majority of ureter injuries (80% to 95%) are caused by penetrating rather than blunt trauma, with the most common cause being gunshot wounds (accounting for 88% to 95% of penetrating wounds), followed by stabbings.[8,56,59-60,63] In gunshot wounds to the back, abdomen, or flank, the occurrence of ureteral injury is estimated to be between 2% and 5%.[8,56-57] Penetrating ureteral injury is thought to be the result of either ureteral transection or by disruption of the intramural blood supply which over time leads to necrosis of the ureter wall. These injuries occur both by direct trauma causing tissue injury or from the temporary cavitation or "blast effect" created by most fast moving missiles, such as bullets and shotgun pellets.[60] Cavitation injury causes microscopic vascular injuries that may not be apparent on initial visualization of the ureter and are thus difficult to identify, often resulting in a delayed diagnosis of injury and increased morbidity.[63-64] Additionally, shotgun pellets can migrate, causing acute ureteral obstruction days after the initial injury.[65]

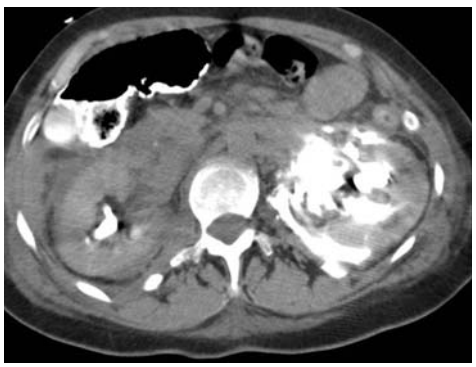
Blunt ureteral injuries are rare and usually occur in the setting of acceleration/deceleration trauma such as those caused by automobile collisions. In these cases, severe hyperextension of the trunk or a direct blow to the L2–L3 region may result in a shearing of the ureter away from the renal pelvis at the ureteropelvic junction.[57] Ureteral injury should be highly suspected in any patient with thoaracolumbar spinal dislocations or fractured lumbar processes.[4] Although a majority of blunt ureteral injuries occur at the ureteropelvic junction, cases of ureteral injuries from blunt trauma occurring below the ureteropelvic junction are documented in the literature, and this must be considered in all cases of acceleration/deceleration trauma.[66]

Clinical Features. The clinical presentation of ureteral injury is difficult to recognize, and the key to successful recognition is maintaining a high index of suspicion. Injuries to the iliac vessels, bladder, sigmoid colon, renal pelvis and the transverse processes of the lumbar spine should raise suspicion of ureteral trauma, as well as any patient who presents with hemodynamic instability due to abdominal, flank or back trauma.[57] Furthermore, patients who develop leukocytosis, fever, abdominal pain, or an unexplained intra-abdominal fluid collection should be further evaluated for the possibility of ureteral trauma.[8] Since hematuria is often absent, it is not a reliable sign of ureteral injury.[19,57,60,67] Frequently, patients with ureteral injury are critically ill and have multiple associated injuries which may delay recognition of the injury. Since clinical features of ureteral injury are nonspecific and inconsistent, ureter injuries are often only found intraoperatively.[58-59]

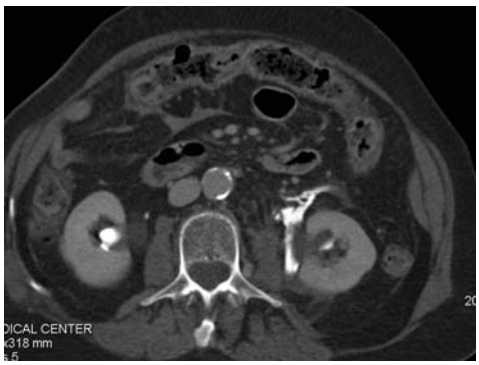
Diagnosis and Imaging. The diagnosis of ureteral injury remains elusive and is complicated by the absence of clinical and laboratory findings specific for ureteral injury.[57] Urinalysis may show gross or microscopic hematuria in 53% to 75% of patients, but hematuria is hardly specific for ureteral injury and may be absent in 30% to 60% of patients.[56-58,60,67] Ureteral injuries are also difficult to diagnose radiographically. The key to successful diag-

Figure 7 a-b. Urinary Extravasation

a

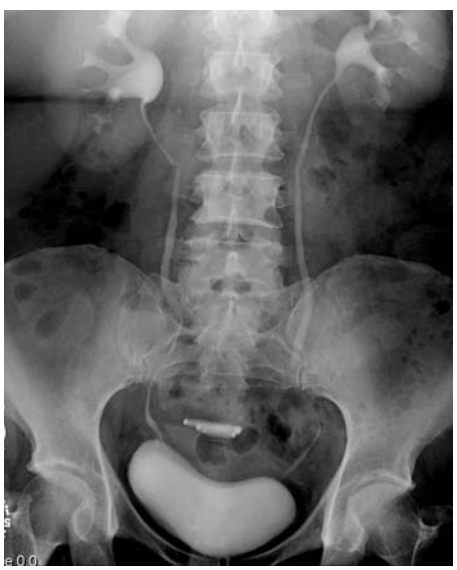


b



a. CT of the abdomen and pelvis with delayed images demonstrating urinary extravasation from the kidney/proximal ureter
b. CT scan of the abdomen with delayed images demonstrating extravasation of urine

Figure 8. Normal Intravenous Pyelogram



nosis is maintaining a high index of suspicion based on mechanism of injury, location of injury, and associated injuries.

CT, specifically delayed image CT and pyelography, is becoming the primary diagnostic tool used in the evaluation of ureteral injury. CT is a valuable diagnostic tool because it helps in the assessment of other abdominal injuries and evaluates the presence of intra-abdominal fluid collections related to trauma.[57] The most common finding is the extravasation of contrast into the medial perirenal space, which may or may not be associated with urinoma.[57,60] *(See Figure 7.)* While the absence of contrast material in the distal ureter can be diagnostic for complete ureteral transection, care must be taken to capture delayed (5–20 minutes after contrast injection) images with quality tracings of the entire course of both ureters.[57,68-70] Although CT is helpful in diagnosing ureter injury in the setting of blunt trauma, its use in penetrating trauma has not yet proven to be better than intravenous pyelogram or IVP, as penetrating abdominal trauma is usually brought directly to surgery for exploratory laparotomy. *(See Figure 8.)* Intraoperative one-shot pyelography is still recommended in these cases.[71]

Initial urinary tract imaging can also be accomplished by obtaining an intravenous urogram. Contrast extravasation is diagnostic. Unfortunately, in many cases, an intravenous urogram may simply show dilatation or deviation of the affected ureter and is therefore only diagnostic in approximately 14% to 54% of studies.[58-60,72] Other imaging modalities, such as retrograde pyelogram, nuclear renal scans, and magnetic resonance imaging (MRI), can be used to evaluate ureteral injury, but these studies are not always logistically possible in the acute setting of a patient with multi-organ trauma common with ureteral injury. *(See Figure 9.)*

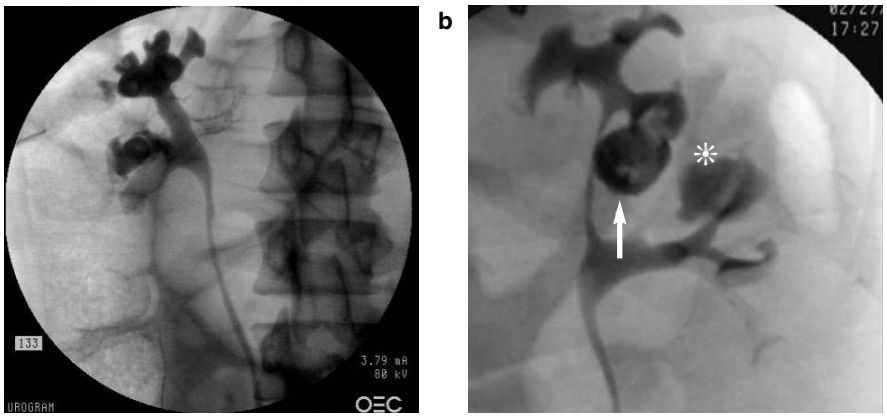
Many ureter injuries, including most caused by penetrating trauma, are diagnosed during laparotomy performed for the treatment of associated abdominal injuries.[60] In the operative setting, direct visualization of the ureter and the surrounding tissue is possible. In fact, according to Azimuddin and colleagues, direct visualization and "exploration of the retroperitoneum remains the only definitive method of excluding ureteric injuries."[58] Intraoperative recognition can be aided by intravenous or intraureteral injection of indigo carmine or methylene blue, which may demonstrate leakage from a transected ureter, although the efficacy of this test can be limited by renal hypoperfusion or hypotension.[59-60] Curiously, many clinicians are now abandoning preoperative radiographic studies altogether, instead favoring intraoperative exploration in patients who warrant laparotomy. Studies by both Digiacomo and Medina demonstrate that preoperative radiographic staging of ureteral injuries is unwarranted in patients who will undergo exploratory surgery. They suggest that direct ureteral visualization and retroperitoneal exploration during laparotomy are sufficiently accurate to avert preoperative radiographic examination.[56,73]

Management. The appropriate management of ureteral trauma depends not only upon the grade and location of injury but also the overall condition of the patient, his or her past medical history and co-morbidities, as well as the time of diagnosis. The severity of ureteral injury can be classified using the grading system developed by Moore and colleagues for the American Association for the Surgery of Trauma.[74] *(See Table 3.)* Additionally, the complexity of ureteral repair secondary to trauma was recently found to correlate with the number of associated injuries and increasing American Association for the Surgery of Trauma-Organ Injury Scale (AAST-OIS) injury grade.[54] The ideal management option for penetrating ureteral trauma is primary repair; however, this is not

always possible because of the co-existing injuries of the patient.[75] In these cases, repair should be deferred until the associated injuries and inflammation have resolved.[60] Ureteral injuries with a significant delay in definitive treatment should initially be managed by either percutaneous nephrostomy or endoscopic ureteral stenting. At the appropriate time, debridement of devitalized tissue and the performance of the appropriate repair can occur.

Minor ureteral contusions without devascularization (grade I injuries), should be treated with stent placement. Often, diversion of the urine stream via stent or nephrostomy may be the only treatment needed.[57] Care must be taken to fully evaluate these injuries, as microvascular injury can lead to stricture or ureteral necrosis resulting in urine leakage and patient morbidity. Severe or large areas of contusion should be treated with excision and ureteroureterostomy.[4] Most grade II–IV lacerations are managed surgically.[57,60,76] Postoperative stenting is usually recommended for up to two weeks after ureteral surgery or until radiographic evidence of an open anastomosis can be obtained.[60]

Figure 9a-b. Kidney Pyelogram



a. Normal retrograde pyelogram.
b. Abnormal retrograde pyelogram demonstrating urinoma *(arrow)* and urine extravasation from calyx *(✳)*.

Table 3. Classification and Description of Ureteral Injuries

CLASSIFICATION	DESCRIPTION
Grade I*	Simple contusion or hematoma
Grade II*	Laceration with less than 50% transection
Grade III	Laceration with greater than 50% transection
Grade IV	Complete ureter transection with 2 cm of devascularization
Grade V	Ureteral avulsion with greater than 2 cm of devascularization

* If bilateral injuries present, advance one grade until reaching Grade III classification.

Adapted from Moore E, Cogbill T, Jurkovich G, et al. Organ injury scaling III: Chest wall, abdominal vascular, ureter, bladder and urethra. *J Trauma* 1992;33:337-339.

Renal Injuries

Definition of Problem and Epidemiology. The kidneys are the most commonly injured genitourinary organs. Up to 10% of trauma patients will sustain injuries to the urologic system and one-half to two-thirds of these injuries will involve the kidneys.[71,77-79] Approximately 80% of these are the result of blunt trauma, most commonly occurring during motor vehicle collisions, falls, assaults, or sports events.[71,77,80] Fortunately, most renal injuries from blunt mechanisms are classified as minor (> 90%).[71,79] Penetrating mechanisms account for about 5% of injuries and are associated with varying degrees of renal lacerations which often require operative interventions. Renal pedicle injuries account for about 2% of kidney injuries. In blunt trauma, the most common pedicle injury is thrombosis of the renal artery. Arterial or venous lacerations and venous thrombosis are also seen.

The majority of renal injuries are not immediately life-threatening and may not be obvious on presentation. However, multi-organ involvement is the rule when renal injuries are found (occurring in 80%–95% of penetrating and 75% of blunt mechanisms) and these associated injuries may be significant and severe.[71,79,81] These more obvious and life-threatening injuries can result in the delayed diagnosis of kidney injuries. To further complicate the diagnosis of kidney injuries, the presence or degree of hematuria does not correlate with the presence or severity of renal injury. This realization, along with the development of better imaging modalities, has resulted in a change in our diagnostic approach to the patient with presumed renal injuries in the last two decades.

Mechanism and Pathophysiology. The kidneys are paired retroperitoneal organs surrounded by adipose and loose areolar connective tissue. The kidney is composed of an outer cortex and an inner medulla. The inner medulla contains renal papillae which drain the renal calyces. Lying against the diaphragm superiorly and the psoas muscles posteriorly, the kidneys are adjacent to the lower two thoracic and first four lumbar vertebrae.[78] The upper portions of the kidneys are protected by the ribs, but the lower poles are often inferior to them and consequently more vulnerable to injury.[82] *(See Figure 10.)* The right kidney is injured more commonly than the left because of its inferior displacement by the liver.

The kidneys are not fixed in the retroperitoneum and move with the diaphragm and with movement of the patient. They are supported by the renal vasculature, the perinephric fat, and a fibrous layer of fascia (Gerota's fascia). The renal artery, renal vein, and the ureter make up the renal pedicle at the indented medial border (hilum) of the kidney. Rapid decelerations, like those seen in motor vehicle collisions and falls from heights, can result in shearing injuries to the renal pedicle and vasculature because the hilum does not move as freely as the kidney.[81,83]

Figure 10. Anatomy of the Kidney

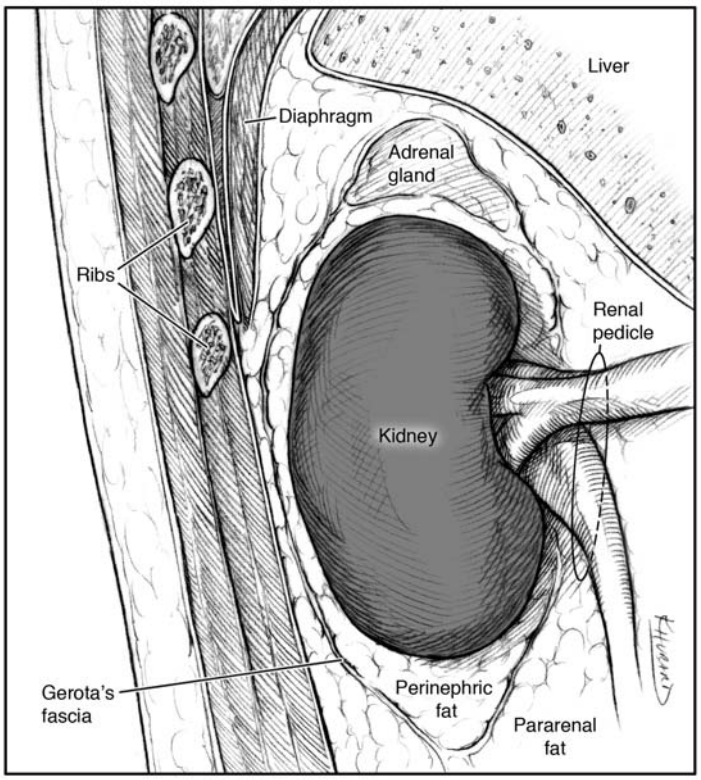
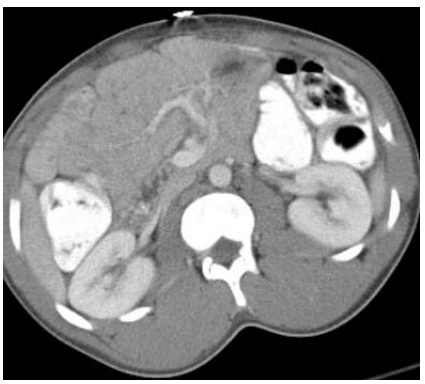
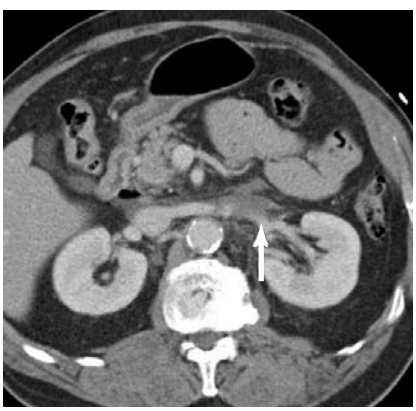


Illustration courtesy of Knox Hubbard

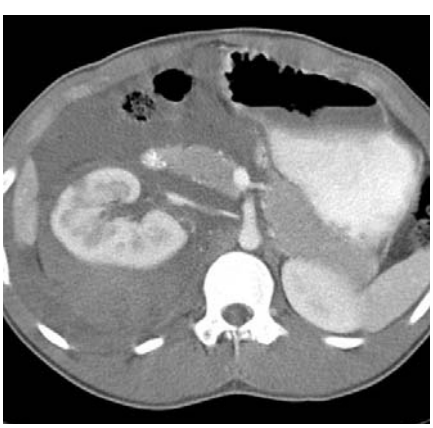
Figure 11a-d. CT Scans of the Kidney



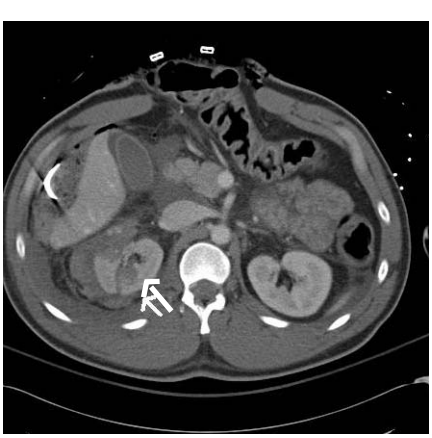
a. Normal CT scan of the kidneys



b. CT scan of the abdomen demonstrating a left renal vein injury noted by stranding marked by the arrow



c. CT scan of the abdomen demonstrating right renal laceration with a large surrounding hematoma



d. CT scan of the abdomen demonstrating a right renal laceration with significant renal parenchymal damage

When viewed in context with the mechanism of injury, the anatomical relationship of the kidneys with surrounding structures and the mobility of the kidney compared to the hilum can offer important clues regarding the possibility of renal injury. Significant deceleration mechanisms should always lead to suspicion of a shearing injury, while penetrating mechanisms to the abdomen, back or flank should always raise the suspicion of direct renal trauma.[81]

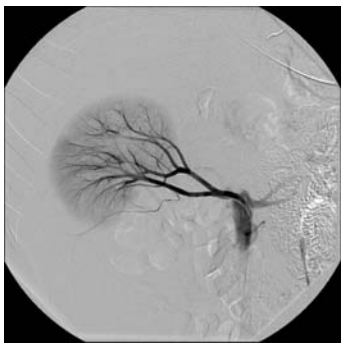
Clinical Features. Renal injuries are seldom immediately life-threatening and may not be obvious in the setting of multiple trauma. Deceleration mechanisms of injury or penetrating trauma in close proximity to the kidneys should certainly alert the clinician to the possibility of renal trauma. Similarly, tenderness or ecchymosis of the abdomen or flank should arouse suspicion. Radiographic evidence of lower rib or thoracolumbar fractures should also trigger further investigation.

Hematuria is the best indicator of urinary tract injury. Over 95% of patients sustaining renal trauma will have some degree of hematuria (> 5 RBC/HPF) but the degree of hematuria is not related to the severity or extent of injury. Renal artery lacerations, complete renal avulsions and other pedicle injuries may not generate any hematuria at all.[71,77,78,82] Particular attention should be paid to any signs of hemodynamic instability. Hypotension, even if transient, in the setting of hematuria mandates imaging of the kidneys.[83,84,85]

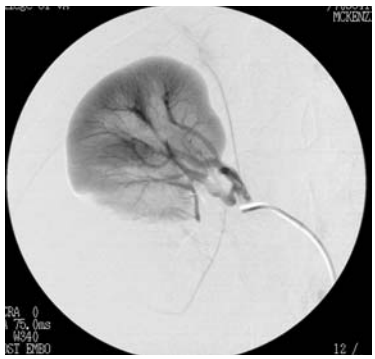
Diagnosis and Imaging. A prospective study based at San

Figure 12a-b. Angiograms of the Kidney

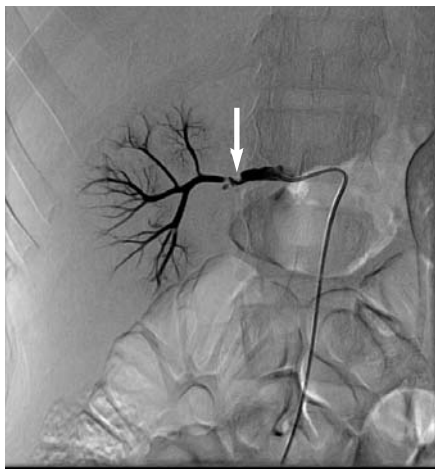
a



b



c



a. Normal angiogram of the right kidney
b. Angiogram of the right kidney depicting a lower pole filling defect due to vascular injury.
c. Angiogram that shows right renal vascular injury where there is a filling defect between the catheter tip and the kidney body *(arrow)*

Francisco General Hospital established indications for radiographic imaging of the kidneys in the setting of presumed renal trauma. This study demonstrated that patients requiring imaging for presumed renal trauma present with either gross hematuria or microscopic hematuria with shock.[83-85] Shock was defined as a systolic blood pressure of less than 90 mmHg at any time, even if this hypotension was only transient in nature. Conversely, microscopic hematuria is unlikely to represent significant blunt renal injury in the absence of hemodynamic instability. After reviewing the existing literature, Ahn and colleagues concluded that patients with microscopic hematuria without evidence of hemodynamic compromise had a significant renal injury in only one out of 500 patients.[86] Therefore, patients with microscopic hematuria in the absence of hypotension do not require emergent imaging of their kidneys.

The presence or absence of hematuria is not predictive of renal injury in the setting of penetrating trauma. The location of the penetrating wound in relationship to the kidney is the most important factor in determining the need for radiographic studies. Significant injuries to the kidney can occur in penetrating trauma without hematuria.[19]

The San Francisco studies demonstrate that contrast enhanced CT scanning is the imaging study of choice in the evaluation of presumed renal trauma. Subsequent studies have shown this study to have diagnostic accuracy of up to 98% for injuries to the kidneys.[87] CT scans provide precise delineation of renal lacerations, determine the presence and location of renal hematomas with or without extravasation, and also indicate the presence of urinary extravasation or devascularized parenchymal segments.[87] *(See Figure 11.)* The advent of helical CT and improved multi-detector array scanners have decreased the time needed to perform the studies and improved resolution. However, CT scans do not define renal venous injuries well. If suspicion for this injury exists, angiography or MRI may be more accurate.[71]

Injuries of the ureteropelvic junction (UPJ) are best diagnosed with delayed imaging techniques that allow contrast to be excreted into the collecting system and ureter. Therefore, delayed scans performed 5–10 minutes after the initiation of intravenous contrast is the preferred method of evaluating the renal collecting system.[88-90] A UPJ injury is indicated by an intact renal parenchyma with extravasation in the medial perirenal space with or without contrast in the ureter distal to the point of injury.[89,91]

Before CT scans were readily available, the imaging procedure

Table 4. Classification and Description of Renal Injuries

CLASSIFICATION	DESCRIPTION
Grade I	Hematuria +/- normal imaging study or subcapsular non-expanding hematoma
Grade II	Non-expanding perirenal hematoma confined to retroperitoneum OR <1 cm laceration of renal cortex without urinary extravasation
Grade III	> 1 cm renal cortex laceration that does not involve collecting system or urinary extravasation
Grade IV	Lacerations involving cortex, medulla, or collecting system OR Renal artery or vein injuries with contained hemorrhage
Grade V	Completely shattered kidneys OR Injuries involving renal hilum that devascularize the kidney

Adapted from American Association for the Surgery of Trauma. Abstracts, 49th annual session. American Association for the Surgery of Trauma. Chicago, Illinois, October 5-7, 1989. *J Trauma* 1989;29:1024-1040.

of choice for presumed renal trauma was intravenous pyelography.[78,90] *(See Figure 8.)* IVPs allow for an assessment of both the anatomy and function of the kidneys, however, they are frequently time consuming and are less sensitive than CT scans for defining the nature and extent of the injury. For these reasons, CT has replaced IVP as the imaging modality of choice for renal trauma. However, the IVP may still have a role in evaluating the unstable patient in the operating room where a limited or "one-shot" study can be performed to assess the gross function and anatomy of a presumed uninjured kidney prior to the removal of a significantly injured one.[78]

The use of angiography in the evaluation of renal injuries has diminished in the current era of high-resolution CT scanners. *(See Figure 12.)* Selective renal angiography still provides more detailed information regarding the exact location of a vascular injury than a CT scan, and it remains the gold standard for evaluation of suspected renal vein injury, particularly in the setting of penetrating trauma. Increasingly, angiography with embolization or stent placement is being utilized in the therapy of renal artery injuries.[92] Angiography may also be indicated for additional clarification when there is no evidence of renal function on either a CT or IVP.[78,87,92]

Ultrasound has little role in the evaluation of renal trauma other than to identify the presence of two kidneys and to demonstrate free fluid in the hepatorenal or splenorenal recesses. Contrast-enhanced ultrasonography has been assessed in small studies and may show future promise in the evaluation of kidney injuries.[93]

MRI is rarely used as a first-line imaging modality due to its greatly increased time of study when compared to CT. However, it can be used in patients with a known contrast allergy or when a CT is not available. MRI has demonstrated a level of sensitivity similar to the CT for renal trauma. In fact, in some instances, it is superior to CT. MRI can differentiate intrarenal from perirenal hematoma more accurately than CT. It can also reveal focal lacerations and non-viable renal segments more accurately than CT in the setting of a perirenal hematoma. MRI may also be the follow-up imaging study of choice for victims of multi-organ trauma in order to decrease the cumulative radiation dose of CT scans.[94]

Management. A variety of grading systems have been developed to classify renal injuries through the years. The most commonly utilized system at present was developed by the American Association for the Surgery of Trauma (AAST) and is based on the depth of injury and the involvement of vessels or the collecting system.[95] *(See Table 4.)* Multiple studies have confirmed the value of this grading system. The higher the grade of injury, the more likely a complication will arise that requires surgical intervention, and the need for follow-up imaging studies will be greater.[81,96,97] However, just as our diagnostic approach to renal trauma has changed, so has our therapeutic approach.[78]

A more conservative approach to the treatment of renal injuries has evolved over the last two decades, and there has been a decline in the rate of both immediate repair of renal injuries and nephrectomy.[78] Non-operative management is advocated for most blunt renal injuries, many renal stab wounds and selected gunshsot wounds.[79] Grade I and II injuries have been handled non-operatively for some time. This management includes observation, fluid hydration, serial hemoglobin checks, and serial urinalysis monitoring for the resolution of hematuria. Nearly all of these injuries will heal spontaneously without sequelae. For example, an adult patient with microscopic hematuria in the absence of hypotension and without evidence of coexisting major organ or lower urologic injury requiring imaging or observation, may be safely discharged from the ED without any imaging studies. However, follow-up is mandatory to determine that the microscopic hematuria has cleared. If it does not, then contrast-enhanced CT or MRI is required.[19,83-86]

In the past, surgical intervention was often the rule for major injuries (Grade III–V). Today, the only absolute indications for surgical intervention are persistent renal bleeding with hemodynamic instability, active extravasation of intravenous contrast, or an expanding or pulsatile perirenal hematoma, which suggests a Grade V vascular injury.[71] Injuries to the ureter or renal pelvis are also indications for repair. Relative indications include urinary extravasation, nonviable tissue, delayed diagnosis of arterial injury, segmental arterial injury and incomplete staging.[71,79]

It is now routine to manage Grade III injuries non-operatively. In a meta-analysis of 16 published reports, 90% of 324 Grade IV blunt renal injuries were effectively managed non-operatively with only 4.6% ultimately requiring nephrectomy. Even Grade V injuries have been managed non-operatively. Bozeman and colleagues reviewed the management of blunt trauma patients with either Grade IV or V renal injuries that were managed either conservatively or surgically. This small study demonstrated no statistically significant difference in morbidity between the two groups.

The only significant predictor of the failure of conservative management was the presence of another solid organ injury.[98] Altman et al reported fewer ICU days, transfusions and complications in a small number of Grade V patients managed conservatively.[99]

Conservative management of selected victims of penetrating injuries has also been advocated. Multiple studies have demonstrated the ability to expectantly manage hemodynamically stable victims of stab or gunshot wounds.[79] Patients selected for nonoperative management undergo serial hemoglobin determinations and CT scans. Embolization via angiography can prevent surgical management in cases of continued bleeding.[100]

The drawback of expectant management in major renal injuries is an increased complication rate.[100] Renal injuries with a significant amount of devitalized parenchyma are more likely to develop short term complications such as continued hemorrhage, continued urinary extravasation and abscess formation and long term complications such as hypertension. Husmann and colleagues reported that major renal lacerations extending into the collecting system with devitalized fragments comprising a quarter of the kidney have an 80% complication rate including perinephric abscess, infected urinoma or continued hemorrhage.[101] Interestingly, in the setting of renal artery thrombosis or avulsion, the kidney can be safely left in place to atrophy over time with a low risk of complications.[77]

With modern management techniques, renal salvage rates approach 85% to 90%.[82] Although the trend for conservative management has extended into Grade IV injuries, nephrectomy for Grade V injuries occurs in 90% of cases and is often needed to control hemorrhage and to improve hemodynamic stability. Even under ideal circumstances and prompt surgical repair, the kidney salvage rate for pedicle injuries is less than 20%.[79,100,102,103]

Conclusion

Serious genitourinary injuries are often well hidden among other more life-threatening injuries, making them difficult to diagnose and treat. The mechanism of trauma should raise or lower a practioner's index of suspicion and guide imaging and treatment. Management goals are focused primarily on the prevention of long-term morbidity, and initial interventions are often temporizing due to the urgency of repairing a patient's more immediate life-threatening injuries.

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CNE/CME Objectives

Upon completing this program, the participants will be able to:

a.) discuss conditions that should increase suspicion for traumatic injuries;
b.) describe the various modalities used to identify different traumatic conditions;
c.) cite methods of quickly stabilizing and managing patients; and
d.) identify possible complications that may occur with traumatic injuries.

CME / CNE Instructions

Physicians and nurses participate in this CME/CNE program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. **After completing this activity, you must complete the evaluation form provided and return it in the reply envelope provided in order to receive a letter of credit.** When your evaluation is received, a letter of credit will be mailed to you.

CME/CNE Questions

1. The next step in management of a trauma patient with an identified partial urethral tear should be:
   A. Urologic consult if the urine does not clear within 24 hours
   B. One attempt to carefully pass a catheter
   C. Placement of a suprapubic catheter
   D. Abdominal CT to look for associated injuries
   E. Proceed to operating room for repair

2. A 25-year-old male presents with a gunshot wound to the left flank. Vital signs are stable. A urine dipstick is negative for blood. Which of the following statements is correct regarding analysis of the urine in this case?
   A. More than 5 RBC/hpf is considered significant if the patient was hypotensive at any time.
   B. Only gross hematuria is of significance.
   C. The presence or absence of hematuria is not a helpful screen for injury.
   D. The presence or absence of hematuria is more important than anatomic location of the injury in guiding further evaluation.

3. A 35-year-old male arrives at the emergency department after having been ejected from his automobile. He has an obvious pelvic fracture, gross hematuria, and an expanding retroperitoneal hematoma. He also has a large abdominal wound with small bowel protruding. Vital signs are blood pressure 90/40 mmHg, pulse 130, and respirations 30/min. Which of the following is correct regarding evaluation of the urinary system in this patient?
   A. A single-shot IVP done in the trauma bay is adequate to demonstrate the vascular integrity of the kidneys.
   B. CT using IV contrast should be performed to assess the full extent of abdominal injury before operative management.
   C. A retrograde urethrogram should be performed before taking the patient to the operating room.
   D. A bladder rupture should be assumed, and a suprapubic catheter should be placed to monitor urine output.
   E. Intraoperative "one shot" IVP should be performed to evaluate the integrity of the kidneys if the patient is stable enough to tolerate the procedure.

4. Which of the following statements is most accurate regarding bladder rupture?
   A. Most extraperitoneal bladder ruptures are associated with femur fractures.
   B. Plain film cystogram is the most accurate study to evaluate for bladder rupture.
   C. The classification of bladder rupture as intraperitoneal or extraperitoneal is academic because the management options are the same.
   D. In patients with a pelvic fracture, the absence of gross hematuria virtually eliminates the possibility of bladder rupture.

5. An 18-year-old male presents with a straddle injury that occurred while riding a bike. He has blood at the meatus but is able to urinate. You suspect an anterior urethral contusion. Considering the long-term outcome and morbidity any injury like this might have on him, what is your disposition?
   A. Discharge home with follow-up if any problems develop.
   B. Discharge home with follow-up with a urologist in two weeks.
   C. CT scan with contrast
   D. Discharge home after placement of a Foley catheter by urology with follow-up in two weeks.

6. A 35-year-old male prisoner arrives in the ED with a complaint of urinary retention. While obtaining a history, he notes he placed a pen's ink cartridge in his urethra two days ago and has not been able to urinate since. His bladder is palpable and enlarged. Bladder scan demonstrates approximately 500 mL of urine. The ink cartridge is palpable on the ventral aspect of the penis and easily removed. Two hours later, the patient is still unable to void and attempts at passing a Foley fail. What is the next step?
   A. Psychiatric consult
   B. Discharge for care at the prison
   C. Retrograde urethrogram
   D. Both A and C

7. A 39-year-old female presents with an unstable pelvic fracture after being thrown from and stepped on by a horse. She has blood in the vagina on bimanual exam but denies recent menses. Vital signs are: blood pressure 90/60 mmHg, heart rate 125, respirations 14/min, pulse ox 99% RA. What is the next appropriate course of action?
   A. Attempt to insert a Foley catheter
   B. Emergent urologic consult
   C. Insert a Foley catheter and obtain a retrograde urethrogram in the ED before ordering an abdomen and pelvis CT
   D. Send the patient to interventional radiology (IR) and perform cystogram and retrograde urethrogram in IR

8. A 25-year-old female involved in a high speed motor vehicle crash presents with the following vital signs: blood pressure 110/75 mmHg, heart rate 90, respirations 15, pulse ox 99%. The pelvis is stable on exam but a plain film of the pelvis shows widening of the pubic symphysis. A bedside FAST exam demonstrates free fluid in both the RUQ and LUQ. Secondary to pain and the patient's pannus you are unable to adequately view the bladder with US. There is gross hematuria in the Foley, which was passed on first attempt. What injury do you suspect and what do you expect to see on CT cystogram?
   A. Intraperitoneal rupture of bladder with extravasation of contrast into the abdomen
   B. Intraperitoneal bladder rupture with extraperitoneal extravasation of contrast
   C. Extraperitoneal rupture of bladder with extravasation of contrast into the abdomen
   D. Extraperiotoneal rupture of bladder with no contrast extravasation

9. A 56-year-old male presents with multiple stab wounds to the lower back. Vital signs are stable. The patient is stable and the decision is made to obtain a CT scan of the abdomen and pelvis to assess the extent of retroperitoneal injuries. What additional imaging, if given the opportunity for an additional study of your choice, would you request?
   A. CT cystogram
   B. None. Abdomen and pelvis CT should adequately evaluate for renal laceration and extravasation.
   C. None. The patient needs to go directly to the OR regardless and renal injury is best evaluated for under direct exam intraoperatively.
   D. IV pyelogram

10. A 25-year-old male is struck in the flank with a baseball bat. His systolic blood pressure is always above 100 mmHg in the ED and his exam is only remarkable for a flank hematoma without abdominal tenderness. In which scenario is imaging required?
   A. He requires imaging because of the mechanism of injury and the location of the hematoma.
   B. Clear urine with dipstick positive for blood.
   C. Clear urine dipstick positive for blood and medic report of transient systolic blood pressure of 88 mmHg.
   D. Clear urine with dipstick positive for blood that has resolved by primary care physician follow up in one week.

Answers: 1. B; 2. C; 3. E; 4. B; 5. D; 6. D; 7. A.; 8. A; 9. B; 10. C.

In Future Issues:

CT in Pediatric Abdominal Trauma