Hackers Spy Scientists

Two weeks ago, I received an e-mail from the editor of a reputed journal requesting me to download an agreement report by using a link in that email. After clicking on the link, I was directed to a web page which requested me to logon with my e-mail credentials. As I am an information technology scientist, I detected that I am facing to a phishing attack. In information security literature, phishing attack is an attempt for stealing users' sensitive information by using a fake website similar to the authentic one [1]. Hackers steal e-mail credentials of journals' editor, and then send many spam mails to steal sensitive information of some researchers who know the editor. In a phishing attack, cyber-criminals design a website similar to the target website. After designing fake website, cyber-criminals direct users to their fake page; when researchers open fake website and enter their information, cybercriminals gather this information.

In recent years, phishing attacks are expanding to scholarly publishing and academic world. Journal phishing, or hijacked journals, are journals that mimic reputable journals with similar names and ISSNs [3,4]. Researchers are receiving e-mails in names of editors, popular universities or eminent researchers. In some e-mails, sender request the receiver to open attachment or login to a website by using his/her e-mail credentials. When the user open the attachment or login to in the mentioned website, his e-mail credentials are stolen by cyber-criminals. A question that may arise is how cyber-criminal could send e-mails by using official emails of researchers or institutes? They use an "e-mail spoofing technique." This technique uses the vulnerability present in the TCP/IP protocol (TCP/IP is the computer networking model and the set of communication protocols used to connect computers over a network) that allows them to send e-mail from any address. However, they cannot receive answers from sent e-mails, and they allows include their phishing website's URLs in spoofed emails to cheat researchers and direct them to their phishing websites so that they can steal their information. It is important for scientists to be aware of their vulnerability to these attacks.

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Concomitant Infections Should not Deter Clinicians from Diagnosing Kawasaki Disease

We recently came across a case of a previously healthy 11-month-old boy admitted with acute gastroenteritis. The child had a 5-day history of passing watery stools, vomiting and fever. Physical examination at admission revealed irritability and signs of some dehydration. There were no signs of meningeal irritation. Investigations revealed hemoglobin 9.2 g/dL, leukocyte count 12×10^9/L (neutrophils 72%), and C-reactive protein (CRP) 54 mg/dl. Stool analysis done as part of national surveillance program using enzyme immunoassay (IDEIA Rotavirus kit, Dako Diagnostics) showed rotavirus antigen. With standard management, diarrhea subsided on day-8 of illness but fever persisted. The child had faint maculopapular rash involving face, congestion of oral mucosa and bilateral cervical lymphadenopathy. There was no conjunctival congestion. Total leukocytes, ESR and CRP at this stage were 18×109/L, 80 and 155 mg/dL, respectively. Bacterial cultures of blood and urine were

negative. Liver function tests were normal. Persisting fever 48 hours after parenteral antibiotics (ceftriaxone, 75 mg/kg/day), leukocytosis and elevated CRP coupled with thrombocytosis (platelet count 750×109/L) led to diagnosis of 'incomplete' Kawasaki disease. Administration of intravenous immunoglobulin (2 g/kg over 12 hours) and aspirin (80 mg/kg/day) subsequently lead to normalization of body temperature in 24h and normalization of inflammatory markers (ESR 12 and CRP 10 mg/dL) in the next 48h. Echocardiography done on day–10 and during later follow-up revealed no coronary artery abnormalities.

Infectious etiology of Kawasaki disease (KD) has been long debated. Putatively, at least 1 in 3 cases of KD have concomitant infection which could be systemic or focal [1,2]. In tropical countries where uncommon presentation of common infections is very common, one needs to be vigilant not to miss KD which may follow recovery from infections such as dengue [3]. In fact, apart from rotavirus and dengue, KD is reportedly associated with more than 20 bacterial and viral infections, and also observed post-vaccination [4,5]. Also, seasonal clustering of cases suggests existence of an environmental trigger. However, such temporal associations have not so far been proven to be causal.

Failure to identify a single etiological agent despite 40 years of research implies that KD might represent an aberrant yet predictable immunological phenomenon triggered by exposure to a variety of environmental factors in a genetically predisposed host. The learning point in this case is the clinician's prudence in the diagnosis and treatment of incomplete KD as a syndrome based on clinical criteria irrespective of other underlying specific and non-specific infectious conditions.

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Pediatric Multiple Sclerosis

An 11-year-old girl presented to us with history of sudden diminution of vision in the right eye. There was no associated history of altered sensorium, fever, headache, vomiting, rashes or head injury. She also had a history of feeling of decreased sensation in left arm 4 weeks ago, which recovered completely and spontaneously within 2 weeks. There was decreased visual acuity in the right eye, with evidence of optic atrophy on fundoscopy. The Visual Evoked Potential (VEP) test revealed increased latency and decreased amplitude in the right eye.

Magnetic resonance imaging (MRI) of the brain showed multiple lesions involving white matter in bilateral periventricular, bilateral fronto-parietal subcortical and right temporal subcortical regions. On contrast enhanced cerebral MRI, few enhancing lesions were located in the corpus callosum, periventricular and bilateral frontal regions (***Fig.* 1**). MRI spinal cord showed T2 hyperintense lesions in cord at C2, C4 and D12 vertebral levels. The cerebrospinal fluid analysis revealed normal cytology and biochemistry, with no oligoclonal band. Anti NMO antibodies were negative. According to Polman (2010 revised Mcdonald criteria) [1], diagnosis of multiple sclerosis (MS) was made and pulse corticosteroid therapy with methylprednisolone was started with strict monitoring of vital and laboratory parameters. The vision improved within 24 hours of initiation of therapy and the girl was discharged on oral steroids, after 3 days of intravenous therapy.

Multiple sclerosis (MS) is a chronic demyelinating disorder of brain, spinal cord and optic nerves characterized by a relapsing-remitting course of neurologic events, separated in time and space, without encephalopathy, thus distinguishing it from acute disseminated encephalomyelitis [2]. Oligoclonal band in cerebrospinal fluid is considered to be a useful aid in diagnosis, but may be absent in up to 60% of confirmed